

## Dimensionality vs Taxonicity of Schizotypy: Some New Data and Challenges Ahead

Kirsty V. Everett<sup>1,2</sup> and Richard J. Linscott<sup>\*,1,3</sup>

<sup>1</sup>Department of Psychology, University of Otago, Dunedin, New Zealand; <sup>2</sup>Porirua CMHT, Capital and Coast District Health Board, Wellington, New Zealand; <sup>3</sup>Department of Psychiatry and Psychology, Maastricht University, Maastricht, The Netherlands

\*To whom correspondence should be addressed; Department of Psychology, University of Otago, William James Building, 275 Leith Walk, Dunedin 9016, New Zealand; tel: +64 3 479 5689, fax: +64 3 479 8335, e-mail: [linscott@psy.otago.ac.nz](mailto:linscott@psy.otago.ac.nz)

**Heterogeneity in the expression of schizotypy may arise from underlying dimensional processes or a taxonic population structure. In a 2-phase study, we tested the taxonicity of self-reported schizotypy within a general psychiatric sample ( $n = 109$ ) and examined taxon validity by testing its association with clinical schizotaxia in follow-up subsamples. Taxometric analyses indicated a taxonic structure (schizotypy prevalence = 38.8%) provided the best description of the underlying population distribution. After a year, schizotypal ( $n = 14$ ) and nonschizotypal ( $n = 14$ ) subsamples returned for diagnosis of clinical schizotaxia by assessment of executive functioning, attention, memory, and negative symptoms. Seven patients met diagnostic criteria, all members of the schizotypy class. Schizotypy was associated with impaired attention and memory, more negative symptoms, poorer global functioning, and more extensive psychiatric histories. We reconcile inconsistencies in the literature by discussing threats to the validity of this and similar research on Meehl's taxonomic model of schizotypy, including conceptual limitations of the lexical hypothesis and conventions of factor analysis. Scrutiny of Meehl's model should involve disambiguation and better measurement of the schizotaxia-schizotypy phenotype.**

**Key words:** schizophrenia/schizotypal personality/schizotaxia/taxometrics/latent variable modeling/negative symptoms/neuropsychological impairment

### Introduction

The schizotypy phenotype is heterogeneous. There are 2 main ways this heterogeneity is explained or reduced. The most common involves attributing the heterogeneity to an underlying *dimensional* process or processes present in all individuals and yielding different quantities of their associated phenotypes in different individuals. The second involves attributing heterogeneity to an underlying *taxonic* population structure. In this case, the population comprises different

classes of people defined by the presence or absence of phenotype-generating processes. There is currently no agreement on which of these alternatives has greater veracity. Here we have 2 objectives. First, we contrast these dimensional vs taxonomic bases for heterogeneity in schizotypy. Second, we show why this and similar evidence falls well short of what is required to address the dimension vs taxon question.

Meehl<sup>1-3</sup> proposed that schizophrenia is a decompenated end-state of the interaction of *schizotaxia*, an inherited neural integrative defect, with the environment. Under all circumstances, this interaction leads to schizotypy—an enduring, intrinsic dispositional *schizophrenia phenotype* that constitutes liability for schizophrenia. Among schizotypes, the probability of clinical disorder increases as a function of environmental risk exposures and the effects of a dozen or more polygenic potentiators (eg, anxiety, introversion, hypohedonia).<sup>3</sup>

As the inheritance of schizotaxia is a binary outcome, Meehl<sup>3</sup> predicted that schizotaxia indicators should have a taxonic distribution and, given the epidemiology of schizophrenia, that the taxon prevalence within the general population would be 10%. Findings from taxometric studies are mostly consistent with this prediction, suggesting a taxon of 8.5%–10.5% in general population and undergraduate samples.<sup>4-6</sup> However, much of this evidence derives from studies of single attributes, self-report measures, and convenience samples of undergraduates.<sup>7-9</sup> Typically, classes are not validated. Nevertheless, several studies show the findings are generalizable to biological risk groups and assessment using schizophrenia endophenotypes.<sup>10-12</sup>

The seminal taxometric examination of schizotypy was of self-report data from nonpsychotic psychiatric patients. Golden and Meehl<sup>13</sup> identified a schizotypy class comprising 37% of the cohort using 7 criterion-keyed indicators. However, there was no independent validation of the class and, since their report, there have been no other examinations of the schizotypy taxon, or validation of it, in general psychiatric samples.

In a 2-phase study, psychiatric patients completed a self-report positive schizotypy questionnaire. After a year, we examined the validity of class membership using Tsuang et al's<sup>14</sup> clinical schizotaxia model, a model of schizophrenia risk based on negative symptoms and cognitive impairment. This risk model was preferred over those based on psychosis or psychosis-like experiences because the initial screening was based on positive features. Thus, clinical schizotaxia is a more rigorous validity criterion. (Tsuang et al's<sup>14,15</sup> schizotaxia bears no resemblance to the meaning Meehl<sup>13</sup> applied when he coined the term: the concepts apply at different levels of description, have different determinants, have different course parameters and clinical implications, and different statistical properties. Here, we apply the adjective *clinical* when referring to Tsuang et al's schizotaxia.) Tsuang et al's<sup>14,15</sup> criteria for clinical schizotaxia are: 6 or more moderate, marked, or severe negative symptom ratings on the Scale for the Assessment of Negative Symptoms (SANS)<sup>16</sup>; a clinically significant impairment (ie,  $\geq 2$  SD below average) in one neuropsychological domain (attention, verbal memory, or executive function); and a mild impairment (ie,  $\geq 1$  SD below average) in a second neuropsychological domain. The hypotheses were that schizotypy would be taxonic with a base rate over 10% and class membership would be associated with negative symptoms, neuropsychological impairment, and clinical schizotaxia.

## Phase 1 Method

### *Participants and Procedure*

Psychiatric patients ( $n = 109$ ; age  $M = 39.5$  years,  $SD = 10.4$  years; 64% female) of public tertiary services volunteered as participants. Inclusion criteria were: English as first language, age  $\geq 18$  years, competent to provide informed consent, diagnosis with a psychiatric disorder. Exclusion criteria were: history of head injury, neurological disorder, substance abuse diagnosis in the past 6 months, or intellectual disability. Volunteers received a questionnaire pack containing study information, consent forms covering participation and release of psychiatric records, instructions, a self-report schizotypy questionnaire, and researchers' contact details. Participants who posted the completed questionnaire to the research team received a \$10 gratuity for participating. Upon receiving a questionnaire, data from a participant's medical records were collated.

Phases 1 and 2 were separately reviewed and approved by the Otago Ethics Committee, a committee accredited by the New Zealand Health Research Council. All participants gave written informed consent to participate. The study was conducted in a manner consistent with New Zealand and international codes of ethics.

### *Measure*

Schizotypy was assessed with the Thinking and Perceptual Style Questionnaire (TPSQ).<sup>17,18</sup> The TPSQ is a self-report measure with 99 items rated on 5-point scales. The TPSQ has 10 factor-based subscales: disorganized thought, social anhedonia, social fear, magical ideation, hallucinations, self-reference ideas, body illusions, solitary pursuits, dyscontrol illusions, and thought disruption. Alpha coefficients for the subscales range from .63 (dyscontrol illusions) to .88 (disorganized thought), with  $M = 0.80$ .<sup>17</sup> Four-week test-retest reliabilities range from .56 (dyscontrol illusions) to .80 (disorganized thought, social anhedonia, magical ideation), with  $M = 0.74$ . All subscales correlated significantly with Golden and Meehl's<sup>13</sup> 7-item scale ( $r = .10-.45$ ), with the exception of the social anhedonia subscale ( $r = .07$ ). Evidence obtained using alternative subscale scoring also indicates the TPSQ has good test-retest reliability and concurrent validity.<sup>17,19</sup>

### *Analyses*

The taxonicity hypothesis was tested using maximum covariance (MAXCOV) analysis, a method for distinguishing dimensional from taxonic population structures through the identification of artifacts within (vis-à-vis modeling of) multivariate distributions.<sup>20</sup> The consistency of the MAXCOV result was tested using a second taxometric procedure (mean above minus below a cut [MAMBAC])<sup>21</sup> and latent profile analysis (LPA), a method for modeling a population structure using 2 or more homogeneous groups. Linscott et al<sup>20</sup> provided a nontechnical description of MAXCOV and LPA and compared the strengths and weaknesses of these in an earlier issue of *Schizophrenia Bulletin*.

Multivariate outliers were identified using leverage. TPSQ subscales were screened in order to eliminate parataxonic correlations among indicators<sup>22</sup> (ie, negative correlations that indicate multivariate data are nonmonotonic and, therefore, that the associated distribution cannot be reduced to a 2-class structure). MAXCOV analysis was undertaken iteratively using Grove's R code,<sup>23</sup> with removal of indicators based on indicator validities. MAXCOV results were corroborated using base rate variance, Jöreskog and Sörbom goodness-of-fit, MAMBAC analysis of the observed data, and comparison of observed MAMBAC curves with those from simulated dimensional and taxonic data (simulation settings: 10 replications, 50 cuts,  $n = 25$  end margins,  $100 \times n = 107$  comparison samples, and the MAXCOV-derived base rate). MAMBAC analyses were completed using Ruscio's<sup>24</sup> R code. As the sample size was relatively small for taxometric analysis, albeit sufficient given the expected base rate, consistency was also examined with LPA using MPlus 6.<sup>25</sup> Log-likelihood, the Akaike and Bayesian information criteria (AIC and BIC), the sample size-adjusted BIC (BSS), and the Lo-Mendell-Rubin

adjusted likelihood-ratio test (LMR-LRT) indexed model fit. Higher (less negative) log-likelihood and lower information criteria indicate better fit, as does  $P \geq .05$  for the LMR-LRT with  $k + 1$  classes.

### Phase 1 Results

Data from 2 multivariate outliers were removed from further analyses. There was no evidence that TPSQ scores (table 1) differed by sex or correlated with age. Two subscales were excluded from taxometric analysis because of parataxonic correlations (social anhedonia, solitary pursuits). MAXCOV analyses were conducted using the remaining 8 indicators (slab width = 0.25 SD). Low-validity indicators were removed in 4 iterations: body illusions ( $K = 0.64$ ), self-reference ideas ( $K = 0.56$ ), magical ideation ( $K = 0.73$ ), and dyscontrol illusions ( $K = 1.08$ ). Figure 1 shows the covariance curve obtained for the 4 remaining indicators. These had a mean validity of  $K = 1.63$  ( $SD = 0.35$ ) and mean base rate of 40.1% ( $SD = 14.7\%$ ), which was significantly greater than 10%,  $t(11) = 7.09$ ,  $P < .001$ .

The MAXCOV goodness-of-fit was 0.870. Mean within-class correlations for the schizotypy and complement classes were .15 and .29, respectively. MAXCOV analyses using 0.33 SD slab widths yielded similar results. The schizotypy taxon contained  $n = 40$  patients. Taxonicity was corroborated by the MAMBAC results: The MAMBAC curve appeared peaked (figure 2), the observed base rate was 49.4% ( $SD = 6.4\%$ ),  $d = 1.88$ , and the comparison curve fit index (0.65) favored a taxonic interpretation.

LPA of the same 4 indicators identified the same 2-class structure obtained with MAXCOV (schizotypy class  $n = 46$ ). Bayesian posterior probabilities of class membership (BPPCM) from LPA and MAXCOV were strongly

correlated,  $r = .89$ . A better-fitting 3-class LPA model (LMR-LRT  $P = .42$  for 4 classes) was obtained with the 4 TPSQ subscales (figure 1). However, LPA of the set of 8 positive schizotypy indicators and LPA of all 10 TPSQ subscales, both of which were better powered than the 4 indicator analysis, showed the 3-class model was not significantly better than the 2-class solution (LMR-LRT  $P = .42$  and .29, respectively), was not replicable across different starts (ie, may reflect the influence of a local minima), or both. The MAXCOV BPPCM correlated  $r = .80$  and  $r = .79$  with the 8-indicator (schizotypy class  $n = 41$ ) and 10-indicator (schizotypy class  $n = 39$ ) LPA BPPCM, respectively.

### Phase 2 Method

#### Participants

BPPCM from a preliminary MAXCOV analysis of Phase 1 data were used to identify and recruit participants for Phase 2. The recruitment protocol prioritized those with extreme BPPCM ( $P = \sim 1$  and  $P = \sim 0$ ) and we obtained a schizotypy sample ( $n = 14$ ) after identifying 26 high-BPPCM patients and a control sample ( $n = 15$ ) after identifying 21 low-BPPCM patients. Of the 47 identified, 5 declined to participate, 8 could not be contacted, 2 had neurological or substance use disorders, 1 had low IQ (ie,  $< 70$ ), 1 was deceased, and 1 had moved from the region. After Phase 2 data collection, errors in the initial MAXCOV analyses were identified. These were corrected and the 29 patients were re-classified on the basis of 3 BPPCMs: from the corrected MAXCOV and the 4- and 8-indicator LPAs. Specifically, patients were classified as schizotypal if 2 or 3 BPPCMs were greater than .5, and to the control group if no BPPCM was greater than .5. Original classifications changed for 2 participants (one in

**Table 1.** Phase 1 TPSQ Scores by Sex, Correlations With Age, and Skewness

TPSQ Subscale	Items	Males		Females		Whole Sample	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	$r_{age}$	Skew
Disorganized thought <sup>a</sup>	11	18.63	8.72	18.39	8.94	.09	0.12
Social anhedonia <sup>b</sup>	11	19.84	8.45	19.58	9.58	.02	0.17
Social fear <sup>a</sup>	7	14.95	6.47	17.00	7.05	-.16	-0.05
Magical ideation <sup>c</sup>	9	9.13	7.69	8.72	6.03	-.10	0.88**
Hallucinations <sup>a</sup>	7	8.66	6.64	7.96	6.57	.15	0.65**
Self-reference ideas <sup>c</sup>	6	7.37	5.32	5.58	5.02	.00	1.01**
Body illusions <sup>c</sup>	3	2.21	2.91	1.57	2.42	.05	1.76**
Solitary pursuits <sup>b</sup>	4	6.32	3.42	5.43	3.65	-.16	0.41
Dyscontrol illusions <sup>c</sup>	4	2.34	2.93	2.59	2.63	-.03	1.32**
Disrupted thought <sup>a</sup>	4	8.08	3.49	8.23	3.00	.01	-0.14

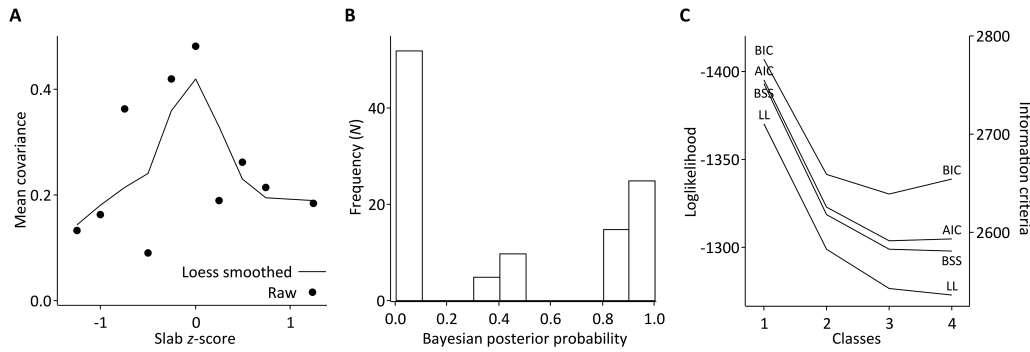
Note: TPSQ, Thinking and Perceptual Style Questionnaire.

<sup>a</sup>Retained in final maximum covariance (MAXCOV) analysis.

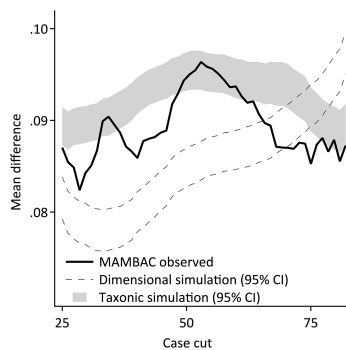
<sup>b</sup>Excluded from MAXCOV analysis because of parataxonic correlations.

<sup>c</sup>Removed during MAXCOV iterations because of low validities.

\*\* $P < .01$ .



**Fig. 1.** Phase 1 taxometric and latent profile results ( $n = 107$ ). (A) The maximum covariance (MAXCOV) analysis plot obtained on the final iteration with 4 indicators (disorganized thought, social fear, hallucinations, and disrupted thought). Dots indicate mean covariance and the line, the loess smoothed mean covariance. (B) Frequencies of Bayesian posterior probabilities of schizotypy class membership obtained from the MAXCOV analysis. (C) Latent profile analysis fit statistics for 1–4 classes obtained on 4 indicators. LL, log-likelihood; AIC, Akaike information criterion; BIC, Bayesian information criterion; BSS, sample size-adjusted BIC.



**Fig. 2.** Mean-above-minus-below-a-cut (MAMBAC) difference plot ( $n = 107$ ) showing the observed MAMBAC curve (solid line) and how this compares to the 95% CIs for the mean MAMBAC curves estimated from 100 dimensional (dashed lines) and 100 taxonic (shaded area) simulated distributions of the same sample size.

each direction); and classification of one participant was ambiguous (only one BPPCM  $> .5$ ) and was excluded from further analysis.

The schizotypy ( $n = 14$ , 6 males) and control ( $n = 14$ , 5 males) groups did not differ in age or follow-up latency, but did differ in years of education (table 2). In the schizotypy group, current DSM-IV diagnoses were major depressive disorder ( $n = 9$ ), posttraumatic stress disorder (3), schizophrenia (2), bipolar I disorder (2), anorexia nervosa (2), schizoaffective disorder (1), panic disorder (1), and social phobia (1). These patients had current prescriptions of antipsychotics (7), anxiolytics/hypnotics (7), antidepressants (9), anticonvulsants (2), and lithium (1). In the control group, diagnoses were major depressive disorder (9), bipolar I disorder (4), schizoaffective disorder (1), panic disorder (1), social phobia (1), specific phobia (1), and posttraumatic stress disorder (1). Control patients had current prescriptions of antipsychotics (5), anxiolytics/hypnotics (5), antidepressants (10), anticonvulsants (3), and lithium (1).

There was no association between schizotypy and loss to follow-up ( $\chi^2 = 0.60$ ,  $P = .44$ ). Within group  $t$  tests of

TPSQ subscale scores by loss to follow-up, and a binomial probability test of the direction of differences on subscales, provided no evidence that participants completing Phase 2 differed from those lost to follow-up.

### Measures

A 2-subtest IQ estimate was obtained using the Vocabulary and Matrix Reasoning subtests from the Wechsler Abbreviated Scale of Intelligence (WASI)<sup>26</sup> or the Vocabulary and Block Design subtests from the Wechsler Adult Intelligence Scale (WAIS-III)<sup>27</sup> (the latter was used for 2 participants). Sustained attention was measured using the continuous performance test, identical pairs version (CPT)<sup>17</sup> and the Paced Auditory Serial Addition Task (PASAT);<sup>28</sup> verbal memory with the Wechsler Memory Scale (WMS-III) Logical Memory subtest<sup>29</sup> and the Selective Reminding Test (SRT);<sup>30,31</sup> executive functioning with the Wisconsin Card Sorting Test (WCST);<sup>32,33</sup> DSM-IV diagnoses with the Structured Clinical Interview for DSM-IV-TR (SCID-I),<sup>34</sup> which also included the Global Assessment of Functioning (GAF); and negative symptoms with the SANS.<sup>16</sup>

### Procedure

R.J.L. identified participants for Phase 2; K.V.E. conducted assessments and was blind to group membership until all assessments were completed. Most completed Phase 2 across 2 appointments and were reimbursed \$25 for each. In the first, participants completed the WMS-III and WASI (or WAIS-III) subtests, the CPT, the PASAT, mental status test of the SANS, the SRT, and the WCST. In the second, the SCID-I interview was administered. SANS ratings were made after the participant had completed all assessment tasks.

### Statistical Analyses

The power to detect large effects (Cohen's  $d = 0.8$ ) in neuropsychological and symptom measures was modest

**Table 2.** Phase 2 Sample Characteristics

Variable	Schizotypy ( <i>n</i> = 14)		Control ( <i>n</i> = 14)		<i>d</i>	<i>P</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
Age	43.6	8.2	44.2	9.1	0.07	.851
Follow-up latency (days)	354	51	348	56	0.11	.764
Education (years)	13.1	2.1	14.8	2.2	0.79	.045
Schizotypy probability	0.86	0.20	0.02	0.03	—	—
IQ	105.5	14.4	109.4	12.5	0.28	.456
GAF score	57.2	9.0	68.4	11.3	1.09	.008
Current diagnoses (count)	1.5	0.5	1.3	0.5	0.43	.262
Past other diagnoses (count)	1.3	1.1	0.5	0.7	0.85	.034
Current psychoactive medications	2.3	1.1	1.7	0.7	0.66	.093
Attention						
CPT hit rate	16.6	9.7	24.0	6.4	0.90	.012
CPT discriminability	1.48	1.18	2.01	1.07	0.48	.110
Memory						
LM percent retention ( <i>T</i> score)	9.3	4.6	11.5	2.7	0.58	.068
LM total recall, delayed ( <i>T</i> score)	6.8	2.8	10.6	2.7	1.36	.001
SRT total recall	103.3	22.9	114.6	17.5	0.56	.076
SRT random LRT	17.5	14.8	16.9	17.9	−0.03	.463
Executive functioning						
WCST total errors	38.9	12.5	41.6	12.6	0.21	.285
WCST perseverative errors	38.3	14.0	42.0	17.6	0.23	.271
Negative symptoms						
SANS scores ≥ 3 (count)	4.6	3.4	2.4	3.2	0.65	.048

Note: GAF, Global Assessment of Functioning; CPT, continuous performance test; LM, logical memory subtest; SRT, Selective Reminding Test; LRT, likelihood-ratio test; WCST, Wisconsin Card Sorting Test; SANS, Scale for the Assessment of Negative Symptoms; *d* = Cohen's effect size index.

(power = 0.53); the power to detect large effects in chi-square tests was 0.75. Directional hypotheses were tested with 1-tailed *t* tests. The sequentially rejective correction for multiple testing<sup>35</sup> was used within families of hypotheses (negative symptoms, neuropsychological impairment, GAF). Box-Cox transformation was used to correct significant skew or kurtosis.

## Phase 2 Results

Eight participants in each group did not meet the PASAT practice performance criterion. Consequently, PASAT data were excluded from analyses. Compared to the control group, the schizotypy group had more moderate or more severe negative symptoms, a lower CPT hit rate, and poorer total story recall (table 2). Schizotypy was associated with poorer GAF, more past other diagnoses (ie, disorders other than those which were current), and a trend toward polypharmacy. Differences on IQ, number of current diagnoses, and other measures of attention, memory, and executive function were not significant (table 2).

The clinical schizotaxia negative symptom criterion was assessed with the SANS. The neuropsychological impairment criterion was evaluated using the CPT-IP hit rate and discriminability, the WMS-III logical memory percent retention and total delayed-recall scale scores, the SRT total recall and random long-term retrieval scores,

and the WCST perseverative and total error *T* scores. In the control group, 74% met either the negative symptom criterion or the neuropsychological impairment criterion, but none met both (table 3). In contrast, 50% of the schizotypy group met both criteria. That is, 7 individuals were diagnosed with clinical schizotaxia and all were in the schizotypy group,  $\chi^2 = 6.86$ , *df* = 1, *n* = 28, *P* = .009.

## Discussion

We sought to test for and validate a discrete schizotypy class within self-selected psychiatric patients, none of whom were excluded on the grounds of psychosis. Analyses of self-reported disorganized thought, social fear, hallucinations, and disrupted thought provided evidence of a latent class boundary demarcating ~40% of the sample. Follow-up of a subset of participants after a year showed that half of those in the schizotypy class, and none in the complement, met criteria for clinical schizotaxia. Compared to the complement, the schizotypy group had poorer attention and verbal memory, more negative symptoms, poorer global functioning, and more extensive psychiatric histories. Thus, the findings suggest a meaningful nonarbitrary boundary demarcates schizotypy within the population affected by mental disorder. Membership in this class predicts greater clinical impairment and more enduring psychiatric morbidity.

**Table 3.** Percentages Meeting Clinical Schizotypia Criteria and Diagnostic Thresholds

Domain, Criterion	Schizotypy ( <i>n</i> = 14)		Control ( <i>n</i> = 14)		<i>w</i>	<i>P</i>
	Severe <sup>a</sup>	Mild <sup>b</sup>	Severe <sup>a</sup>	Mild <sup>b</sup>		
Impaired attention	50	21	7	50		
Impaired memory	64	14	36	14		
Impaired executive function	43	21	36	14		
Neuropsychological criterion	79		43		0.37	.122
Negative symptom criterion	64		21		0.43	.056
Clinical schizotypia diagnosis	50		0		0.58	.009

Note: *w* = Cohen's effect size index for chi-square tests (large *w* = 0.5; medium *w* = 0.3).

<sup>a</sup>Severe = percent with severe (or severe and mild) impairment ratings on one or more indices.

<sup>b</sup>Mild = percent with mild (but no severe) impairment ratings on one or more indices.

The schizotypy class prevalence is similar to that observed by Golden and Meehl<sup>13</sup> in a nonpsychotic sample. Since that seminal report, the balance of taxometric findings points toward schizotypy having a class structure.<sup>36,37</sup> Two taxometric studies show the validity of the class division in biological risk samples. In offspring assessed in childhood and adolescence, schizotypy class membership is associated with parental diagnostic status (eg, schizophrenia, affective disorder, control) and predicts psychiatric hospitalization and schizophrenia and related outcomes in early adulthood and later life.<sup>10,11</sup> Taxonicity has also been observed using cognitive and oculomotor risk indices as indicators.<sup>12</sup> In this case, class membership predicted greater self-reported schizotypal features as well as a family history of schizophrenia but not of bipolar or other disorders.

There is limited basis for comparison of the current findings with those from latent structure studies of schizophrenia-spectrum indicators in clinical samples. Most studies of latent structure within clinical samples have restrictive inclusion criteria, such as presentation with psychosis,<sup>20,38–40</sup> thereby introducing demarcations that may or may not correspond with underlying population boundaries.<sup>20</sup> These studies, which have samples that range from just over 100 participants to over 1000, commonly report evidence of class structures, sometimes broadly corresponding to DSM divisions or to conceptual distinctions among different forms of psychosis or schizophrenia. Our findings are consistent with Fossati et al's<sup>41</sup> observation that schizotypal personality disorder was taxonic, with the class comprising 23% of consecutive referrals to a psychiatric clinic.

### Specific Limitations

Several limitations may affect the interpretation of the findings. The Phase 1 sample size is smaller than conventional recommendations for taxometric analyses of schizotypy. A small *n* may reduce precision of the base rate estimate. However, the smallness itself does not

negate the findings, just as low statistical power is relevant only when deciding whether absence of significant evidence is likely due to an inadequate test sample. The minimum *n* required for taxometric analysis is not uniform but depends on the expected class prevalence and the stability of the analysis coefficient (eg, covariance, eigenvalue, slope, difference). The fact that the MAMBAC simulations provided discrimination of dimensional and taxonic samples of the same *n* also speaks to the adequacy of the current sample. The Phase 2 *n* is adequate because the theoretical problem demands that any effects should be large. That said, there is risk that a small sample is less representative of the sampling population than a larger sample. So, it is reasonable to question the generalizability of the validation findings. Similarly, generalizability may be affected by reliance on a self-selected sample, unsupervised completion of the TPSQ by patients, and the absence of any measure to detect disingenuous responding.

Several factors affect the use of clinical schizotypia as a validity criterion. Whereas in most studies of clinical schizotypia, diagnosis has required a history of schizophrenia in a first-degree biological relative and been excluded where there is evidence of psychosis,<sup>42</sup> we did not apply these criteria. The negative symptom and neuropsychological assessments each include measures of impaired attention creating a degree of redundancy between the criteria. Moreover, some question whether impaired attention should properly be regarded as a negative symptom.<sup>43</sup>

### Obstacles to Resolving the Dimension vs Taxon Question

Meehl's is just one of a number of theories of schizotypy but it is typically the focus of taxometric studies. Findings from taxometric studies of schizotypy are not uniform. Most findings appear consistent with the taxonomic model whereas fewer appear to favor the dimensional model.<sup>4,44–49</sup> Reconciling these findings is not

straightforward. Here, we consider briefly what may be the most likely factors contributing to discrepancies in the literature.

The first set of factors is concerned with validity. Empirical studies on the structure of schizotypy reflect aspects of 2 overlapping traditions. First, top-down hierarchical strategies, such as exploratory factor analysis, are widely employed to understand the heterogeneity of schizophrenia. In this tradition, researchers explain variance in all-inclusive sets of indicators; and both syndrome expansion and magnification create more variance, which leads to elaboration of the structure that is identified (eg, Peralta et al<sup>50</sup> vs Peralta and Cuesta<sup>51</sup>). Second, dimensional accounts of personality have been established by factor analytic studies of language, which in turn are founded on the presupposition that lexicon evolution reflects fundamental personality structure.<sup>52</sup> In this tradition, higher order phenotypes (eg, openness, conscientiousness, extraversion, agreeableness, neuroticism) derive from analysis of descriptions of behavior and subjective experiences without reference or linkage to neurobiological underpinnings of these behaviors or experiences.

These traditions are not well suited to testing Meehl's schizotypy for several reasons. Critically, the taxonicity hypothesis applies to schizotaxia.<sup>53</sup> Schizotaxia and its proximal effects (cognitive slippage, aversive drift) create taxonicity in indicators that may not be specific to schizotypy (eg, anhedonia). Also, the schizotaxia-schizotypy-schizophrenia model is not a syndromic framework because a specific pathogenic process is identified, namely, an integrative neural defect. Consequently, to be appropriate, analyses of schizotypy need to: (a) include a variety of indicators linking the integrative neural defect with the end products of the defect, the latter being described in lexical indicators and (b) proceed in a manner that permits the identification and refinement of both the underlying construct and the set of valid indicators of it.<sup>2,54</sup> This is why Meehl<sup>54</sup> described MAXCOV as a "taxonomic search method" (p. 200). Practically, this requires the use of nonlexical indicators in taxometric analyses, the use of indicators derived across multiple levels (signs and symptoms, cognition, neurophysiology, etc.), and the use of indicators that are hypothesized to be directly sensitive to the underlying pathogenic process. Additionally, refinement requires the reduction of variance through the elimination of variables that do not behave as expected, a common practice in early taxometric work.<sup>11,13</sup>

It is possible to arrive at the same conclusion about validity and pragmatic implications about identification and refinement without reference to Meehl's theory. Consider these. First, the challenge of clarifying schizotypy parallels the challenge of clarifying schizophrenia. For the latter, there is no doubt that clarification will depend on identifying key biological and nonbiological processes and the links these have with signs and symptoms,<sup>55,56</sup> and that structural analyses of lexical indicators

alone will add relatively little. The same must be true of schizotypy. It is difficult to see how inferences about the latent structure of a pathogenic process can be derived solely from lexical descriptions of epiphenomenal products of that process.<sup>57,58</sup> This lexical hypothesis problem also applies to observer ratings, which introduce unavoidable bias, including bias based on diagnostic tradition.<sup>20,37</sup> Second, it is well recognized that, if there is more than one, the mechanisms that underlie different phenotypes of schizotypy may give rise to different latent structures.<sup>48</sup> Indeed, this has been observed with schizotypy<sup>17</sup> and depression.<sup>59</sup> Third, many theories of schizophrenia identify a single pathogenic process or final common pathway.<sup>37</sup> There is no reason for thinking that, in respect of a particular process or pathway, all downstream functions of the organism are equally affected and all measures of these functions are equally sensitive to the defect.<sup>3</sup>

The second set of factors is methodological in focus. We think each of the following, which are common in the extant evidence base, has the potential to obscure the true latent structure of schizotypy: (a) homogeneity of measurement method contributes to covariation between indices they generate. In latent structure analysis, such method variance emerges as a latent dimensional structure.<sup>60</sup> (b) Structures derived from item parcels—an indicator composed from a set of items—can differ from structures derived from the items comprising the parcel.<sup>61</sup> This may explain why analyses based on broad composite scores<sup>46,48</sup> yield different results to those obtained from indicators from the same or similar measures.<sup>9</sup> (c) The boundary conditions for taxometric methods are not well understood. Item skewness may create the appearance of a small-class taxon in some but not all situations.<sup>62,63</sup> Conversely, in unpublished simulations, we have found that wide variation in endorsement rates (item difficulty), which has been an objective in the development of some schizotypy scales,<sup>64</sup> can mask taxonicity. (d) There are many simple reasons that indicators may not be appropriate for inclusion in analyses,<sup>13,22,65</sup> including that they are sensitive to other classes (eg, sex). If items are not screened to remove those that will clearly not be sensitive to a nonarbitrary scale point<sup>66</sup> that divides schizotypes from nonschizotypes, the likelihood of detecting the true latent structure will be reduced.

Given these considerations, designs that are built implicitly or explicitly on the lexical hypothesis and all-inclusive indicator sets are likely to have limited utility in refining understanding of the structure of schizotypy. The current trend toward extensive and sophisticated analyses must be complemented by careful consideration of the indicators that are subjected to them. Few if any of the obstacles described here can be overcome through analysis alone.

These issues also affect our interpretation of the findings we report from the general psychiatric cohort. The absence of nonlexical indicators is an obvious weakness

of the current study. Whether this is compensated by validating the class structure using objective indices that others have used as indicators in taxometric analysis (eg, CPT discriminability)<sup>12</sup> is unclear. The indicators were item parcels identified using principal components analysis of item-level data. We have not subjected TPSQ items to taxometric analyses and so do not know whether the items identify the same structure as the parcels used here. Indicators retained in the final taxometric iteration (disorganized thought, social fear, hallucinations, disrupted thought) were not a broad representation of schizotypy features. Psychometric explanations should be sought before considering whether the excluded variables are not indicators of a schizotypy taxon.<sup>67</sup> For example, we note that the excluded indicators tended to have greater skewness than those that were retained (table 1). Nevertheless, ad hoc analyses (not reported) showed that the schizotypy class had higher scores on all of the excluded indicators except solitary pursuits, which did not differ between groups.

#### *Is the Unaffected-to-Schizophrenia Spectrum Dimensional or Categorical?*

We think this question is too simplistic. It belies the degree of challenge presented by the aforementioned problems, leads to the wrong sort of research, and risks pre-emption of proper examination, rejection, or reformulation of Meehl's taxonicity hypothesis. Instead, consider the following questions.

1. What features comprise the schizotaxia phenotype? Although Meehl posed this question in 1989,<sup>2</sup> we are not aware of any study where this has been addressed.
2. What inheritance patterns generate latent taxonicity? Meehl never proposed that schizophrenia arises from a single dominant schizophrenia gene. Rather, his *schizogene* hypothesis specifies a *schizotaxia-gene*. Notwithstanding this and its merits, in the relationship between genetic load and outcome, the degree of departure from linearity required to generate phenotype taxonicity is not known.
3. What gene-environment interactions or nongenetic mechanisms may generate taxonicity? Taxonicity need not stem from genetic variability nor is it necessary that the distribution or population structure of a phenotype behave in the same way as the multivariate distribution of underlying genotypes. Alternatively, could proximal pathogenic mechanisms—such as dopamine dysregulation, aberrant salience, reinforcer processing, or cognitive slippage—create taxonicity through non-linear response functions?
4. Do class prevalence rates vary according to family history or with environmental risk exposures?
5. How are dimensional polygenic potentiators and their effects disentangled from the influence of a taxonic

schizotaxia? The more distal indicators are from schizotaxia, the greater the contribution of polygenic potentiators to variance within a distribution.

Progress in understanding the latent structure of schizotypy depends on satisfactory and compelling answers to these and similar questions. Inconsistencies in the literature signal need for greater clarity. Some of these inconsistencies may reflect sample construction and problematic application of taxometric procedures to data sets.<sup>4,20</sup> However, the more basic challenges are disambiguation and better measurement of the schizotaxia-schizotypy phenotype.

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