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DSM-IV schizotypal personality disorder: a taxometric analysis among individuals with and without substance use disorders in the general population

Bradley T. Kerridge^{a,*}, Tulshi D. Saha^b, and Deborah S. Hasin^{a,c}

^aDepartment of Epidemiology, Mailman School of Public Health, Columbia University, 722 West 168th Street, New York, NY, 10032

^bLaboratory of Epidemiology and Biometry, Intramural Division of Clinical and Biological Research, National Institutes on Alcohol Abuse and Alcoholism, National Institutes of Health, 5636 Fishers Lane, Rockville, Maryland, 20852

^cDepartment of Psychiatry College of Physicians and Surgeons, Columbia University, New York, New York, 10032

Abstract

This study examined the underlying structure of DSM-IV schizotypal personality disorder (SPD) among individuals with and without a substance use disorder. Using a nationally representative sample of U.S. adults, taxometric analyses were conducted on SPD in the total sample and among individuals with and without a substance use disorder. The structure of SPD in the total sample and among individuals without substance use disorders was dimensional (comparison curve fit indices (CCFI): 0.440, 0.365) whereas a taxonic structure was demonstrated among individuals with a substance use disorder (CCFI: 0.679). Taxonicity underlying schizotypy and SPD in prior taxometric research may have been the result of sampling high risk subsamples of the population. Taxometric research on SPD and other personality psychopathology among high risk subgroups of the population can help elucidate the complex etiology of SPD and the role played by comorbid substance use disorders in the expressivity of these disorders.

Keywords

Schizotypal personality disorder; taxometric analyses; substance use disorders; structural analyses; personality disorders

Introduction

Schizotypal personality disorder (SPD) is a schizophrenia spectrum disorder with well-established genetic relationships between SPD and schizophrenia and shared etiological,

*Corresponding Author: Bradley Kerridge, Ph.D., Mailman School of Public Health, Department of Epidemiology, 722 West 168th Street, New York, NY, 10032, Phone: (301) 990-8391, bk2518@columbia.edu.

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neurodevelopmental and cognitive processes underlying their phenotypical similarities (Bedwell & Donnelly, 2005; Kendler, Myers, Torgersen, Neale, & Reichborn-Kjennerud, 2007). According to the Diagnostic and Statistical Manual- Fourth Revision (DSM-IV: American Psychiatric Association, 1994), SPD manifestations include ideas of reference, odd beliefs, unusual perceptual experiences, odd thinking, appearance or behavior, suspiciousness, constricted affect, lack of close relationships and social anxiety.

Modern conceptualizations of SPD and schizophrenia spectrum disorders derive from both categorical and dimensional models. Meehl's (1962, 1989, 1990, 1992, 1995) neurodevelopmental model of schizophrenia describes the role of biopsychosocial risk factors in determining the gradient of expressivity and the existence of clinical and subclinical entities. He argued that a single discrete causal factor produces schizotaxia characterized by neural integrative defects, synaptic disconnectivity and widespread neural aberration. Through social learning, individuals with schizotaxia develop schizotypy, the psychological and behavioral manifestation of latent liability for schizophrenia.

The expressivity gradient of schizotypy ranges from nonpathological to subthreshold presentations, to SPD and then schizophrenia. Meehl's model, however, implies that the structure of liability for schizophrenia is dichotomous; individuals are at risk because of inherited predisposition. By extension, it predicts that schizotypy is dichotomous given the one-to-one correspondence between schizotaxia and schizotypy (Lenzenweger & Korfine, 1992). Schizotypy and SPD are not identical, however, Meehl (1990, 2004) recognized that schizotypy may underlie vulnerability to or encompass SPD. Although schizotypy represents a latent personality organization and SPD comprises observable signs and symptoms, Meehl's model holds that manifestations of schizotypy can include SPD and schizophrenia (Lenzenweger et al., 2005). Thus, insofar as individuals with SPD carry latent liability, they appear to represent a valid putative schizotype (Ahmed et al., 2013).

Purely dimensional views of schizophrenia spectrum disorders also have proponents. According to Eysenck (1993) and Claridge (2006), psychoticism is a normal behavioral variation on a severity spectrum with schizophrenia and related disorders contributing to schizophrenia liability. Gettesman and Shield's (1972) threshold model also posits that the dimensional distribution of schizophrenia (and by extension schizotypy) risk results from additive effects of multiple genetic and environmental factors. At lower levels of expressivity, schizotypy is dimensional, but as risk factors accumulate beyond a threshold, qualitatively distinct entities emerge within the schizophrenia spectrum.

The taxonicity versus dimensionality of SPD is important to classifications of SPD and other categorically defined DSM-IV personality disorders (PDs) that have remained unchanged in the DSM-Fifth Revision (DSM-5: American Psychiatric Association, 2013). Many clinicians and researchers have proposed a dimensional representation of PDs based on the limitations of categorical models including limited construct validity and high rates of diagnostic co-occurrence (Widiger and Simonsen, 2005; Widiger & Trull, 2007). Further, understanding the latent structure of SPD can help elucidate the structure of schizophrenia spectrum disorders without confounding by severe psychiatric symptoms (Lenzenweger & Korfine, 1995). Thus, these questions are salient to psychometric and genetic research on

schizophrenia spectrum disorders (Holzman et al., 1988; Matthysse, Holzman, & Lange, 1986; Meehl, 1962, 1990).

Meehl and colleagues developed taxometric methods to differentiate between taxonic and dimensional constructs (Meehl, 1995a; Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998). Schizotypy, generally defined by selected positive (i.e., perceptual aberration, magical thinking, referential thinking, and hallucinatory tendency) or negative (i.e., social anhedonia, physical anhedonia) features, is one of the most taxometrically studied personality constructs. Most of the sixteen taxometric studies published to date measured a single domain of positive or negative features and were conducted in large undergraduate samples (Blanchard, Gangestad, Brown, & Horan, 2000; Horan, Blanchard, & Gangestad, 2004; Korfine & Lezenweger, 1995; Lezenweger, 1999; Lezenweger & Korfine, 1992; Linscott, Marie, Arnott, & Clarke, 2006; Meyer & Keller, 2001); a few used smaller at-risk samples (e.g., offspring of mothers with schizophrenia) (Erlenmeyer-Kimling, Golden, & Cornblatt, 1989; Golden & Meehl, 1979). Taxonic structures underlying schizotypy were found in all but four of these studies.

In addition to the taxometric studies on schizotypy, four studies investigated the structure of SPD using symptoms approximating DSM-IV criteria (Ahmed et al., 2013; Fossati et al., 2007; Rawlings, Williams, Haslam, & Claridge, 2008; Tyrka, et al., 1995a, Tyrka, Haslam, & Cannon 1995b). Findings were mixed. Two studies, one among high school and university students (Fossati et al., 2007) and the other among children of schizophrenic mothers (Tyrka et al., 1995b) observed taxonicity. Two studies, one among students and health professionals (Rawlings, Williams, Haslam, & Claridge, 2008) and the other among respondents in two general population surveys (Ahmed et al., 2013) revealed dimensionality. Of note, none of these studies measured SPD directly, but reduced the nine DSM-IV criteria into smaller factor analytically-derived scales or used total SPD symptoms as a single scale indicator combined with indicators from other self-report trait measures.

Support for the taxonic structure underlying SPD may reflect sampling design. Most of these studies were conducted among undergraduates who have higher rates of substance use disorders (SUDs: Blanco et al., 2008; Dawson, Grant, Stinson, & Chou, 2005) than the general population (Hasin, Stinson, Ogburn, & Grant, 2007). Investigators have long been interested in the high prevalence of SUDs among individuals with schizophrenia spectrum disorders (Dixon, 1999). Evidence generally supports associations of schizotypy and SPD with substance use, abuse and dependence (Bailey & Swallow, 2004; Chen et al., 2003; Compton, Chien, & Bollini, 2009; Esterberg, Goulding, McClure-Tone, & Compton, 2009) although the nature of the relationship remains unclear. Notwithstanding that SPD and SUDs may both constitute risk factors for schizophrenia spectrum disorders; no taxometric study has examined the structure of SPD separately among individuals with and without SUDs.

The present study was designed, in part, to address sampling issues in prior taxometric research on schizotypy and SPD. Using Wave 2 of the large nationally representative National Epidemiologic Survey on Alcohol and Related Conditions (NESARC: Grant, Kaplan, & Stinson, 2005), the underlying structure of DSM-IV SPD was directly assessed in the total sample and among subgroups with and without SUDs. We hypothesized that SPD

will be dimensional in the total sample and among individuals without SUDs and taxonic among individuals with SUDs by virtue of increased risk of schizophrenia spectrum disorders.

Methods

Sample

Data were derived from participants in the Wave 2 of the NESARC described in detail elsewhere (Grant, Kaplan, & Stinson, 2005). The Wave 1 NESARC was a representative sample of the US population 18 years and older, residing in households and group quarters. Face-to-face interviews were conducted with 43,093 respondents. The NESARC oversampled blacks, Hispanics and individuals aged 18 to 24 years. The overall response rate was 81.0%.

In Wave 2, attempts were made to conduct face-to-face interviews with all Wave 1 respondents. The Wave 2 response rate was 86.7% reflecting 34,653 completed interviews. The cumulative response rate at Wave 2, the product of the Wave 2 and Wave 1 rates, was 70.2%. As in Wave 1, Wave 2 data was adjusted for oversampling and household and person nonresponse. The weighted data were further adjusted to represent the U.S. general population.

All potential NESARC respondents were informed in writing about the nature of the survey, the statistical uses of the survey data, the voluntary aspect of their participation, and the federal laws that rigorously provided for the strict confidentiality of the identifiable survey information. Those respondents consenting to participate after receiving this information were interviewed. The research protocol, including informed consent procedures, received full ethical review and approval from the US Census Bureau and US Office of Management and Budget.

Schizotypal personality disorder

All diagnoses were made using the Alcohol Use Disorder and Associated Disabilities Interview Schedule- DSM-IV Version (AUDADIS-IV: Grant, Dawson, & Hasin, 2004). The AUDADIS-IV is a fully-structured interview designed for administration by experienced non-clinical interviewers. Each DSM-IV SPD symptom question was followed by a question on whether the symptom caused distress or social or occupational dysfunction. A SPD diagnosis required respondents to endorse five of nine SPD symptom items with at least 1 symptom item causing distress or dysfunction. Seventeen symptom items operationalized the nine DSM-IV SPD criteria. Indicator items for the taxometric analyses were the nine dichotomous SPD criteria in addition to a tenth indicator that was the sum of positive responses to the nine distress/dysfunction items.

Test-retest reliability of the AUDADIS-IV SPD diagnoses and dimensional item scales, assessed in a general population sample (Ruan et al., 2008) was good ($\kappa = 0.67$), as were intraclass correlation coefficients for dimensional symptom items (0.71). The validity of SPD was assessed by examining the association between the diagnosis and several Short Form 12v2 mental and social disability scales (Ware, Kosinski, Turner-Bowker, & Gandek,

2001), controlling for other personality disorders, and other comorbid substance use, anxiety and mood disorders. SPD was a highly significant predictor for all SF-12 disability scales (Grant et al., 2004). Respondents with SPD had significantly greater disability and social/occupational dysfunction than respondents who did not have SPD. These results are consistent with a recent study by Samuel et al. (2013) that compared the predictive five-year validity of clinicians naturalistic personality diagnoses (including SPD) with diagnoses from semi-structured interviews and self-report questionnaires. Using psychosocial, interpersonal and recreational functioning measures as independent external criteria, the study showed that clinician's personality disorder diagnoses were less valid than those derived from semi-structured interviews or self-report questionnaires. The results suggested that clinicians use semi-structured interviews or self-report measures to inform personality disorder diagnoses.

Substance use disorders

The AUDADIS-IV assessed DSM-IV criteria for alcohol and drug specific abuse and dependence for 10 drug classes: sedatives, tranquilizers, opiates (other than heroin), stimulants, hallucinogens, cannabis, cocaine (including crack), inhalants/solvents, heroin and other drugs. Alcohol and drug-specific abuse and dependence diagnoses were aggregated to yield the any SUD diagnosis used to classify respondents in this study.

Consistent with DSM-IV, lifetime abuse diagnoses required respondents to meet at least one of four abuse criteria in the year preceding the interview or before and was precluded by a diagnosis of dependence. AUDADIS-IV lifetime dependence diagnoses required respondents to satisfy at least three of seven dependence criteria during the past year or before. Dependence diagnoses before the past year were required to satisfy the DSM-IV time clustering criteria: at least 3 symptoms must have occurred within the same year.

Reliability and validity of the AUDADIS-IV alcohol and drug use disorder criteria and diagnoses ($\kappa = 0.53\text{--}0.79$) are documented in numerous psychometric studies (Chatterji et al., 1997; Grant et al., 1995; Hasin, Carpenter, McCloud, Smith, & Grant, 1997; Muthen, Grant, & Hasin, 1993; Nelson, Rehm, Üstun, Grant, & Chatterji, 1999; Vraiti et al., 1998), including clinical reappraisals by psychiatrists in clinical (Canino et al., 1999) and general population samples (Cottler et al., 1997) in several countries as part of the World Health Organization/National Institutes of Health International Study on Reliability and Validity.

Statistical analyses

Dichotomous indicators—The use of dichotomous indicators was suggested by Meehl as early as 1965. Concern has been expressed (Miller, 1996) that dichotomous input can, in rare circumstances, result in false evidence of a latent taxon or identification of a pseudo taxonic plot (Meehl, 1995a). However, Meehl & Yonce (1996) questioned the theoretical basis for this concern, noting that dichotomous output indicators are formally identical to the preferred quantitative output because the general covariance mixture theorem is distribution free, holding for any pair of real numbers, including those taking on only two values. This concern has also been rebutted by Monte Carlo work showing that taxometric procedures can be used confidentially with dichotomous data with demonstrated reliability and validity (Ruscio & Ruscio, 2004a). Further, a comprehensive review of taxometric studies conducted

prior to 2011 showed no tendency for taxometric procedures using dichotomous indicators to yield higher rates of taxonic findings (Haslam & Kim, 2002; Haslam, Holland, & Kuppens, 2012).

Base rates—Taxometric analyses require that the taxon must be sufficiently large to be identified if indeed it exists. Meehl's rule of thumb for the estimated taxon base rate (proportion of taxon members in a sample) was 0.10 (Meehl & Yonce, 1996). However, recent monte carlo simulation studies have shown that the absolute number of taxon members may be more important than the base rate (Ruscio, 2009; Ruscio & Ruscio, 2004b). These studies indicated that a taxon sample sizes between 200 and 300, with taxon base rates between 0.0025 and 0.01 were suitable to establish a valid taxon when the total sample size of the study exceeded 10,000 (as in this study).

Indicator validity and nuisance covariance—Indicator validity in taxometric analyses is expressed as the difference in indicator variable means within the taxon and complement groups, standardized by the pooled within group variance, commonly known as Cohen's *d* (Cohen, 1992; Ruscio & Ruscio, 2004a; 2004b). Indicators with a mean separation of approximately 1.2 standard deviations or greater should effectively differentiate taxon and complement groups (Beauchaine & Beauchaine, 2002; Meehl, 1995a). Potential nuisance covariance unexplained by group membership is assessed by examining the average within group (taxon, complement) correlations among indicators. Monte Carlo simulation studies suggest that taxometric methods are robust to moderate nuisance covariance on average within group (taxon and complement) correlations among indicators of < 0.30 (Meehl, 1995b; Ruscio, Ruscio, & Carney, 2011). In contrast, indicator variables in the total sample should be positively associated if the indicators represent multiple facets of a target construct (Ruscio & Ruscio, 2008; Ruscio et al., 2011).

Indicator validities and nuisance covariances were estimated using DSM-IV SPD diagnostic status as an a priori classification (Beauchaine, 2007; Ruscio, 2009). Taxometric analyses used the same a priori diagnostic classification allowing class membership to be defined.

Taxometric Analyses—Three taxometric procedures were conducted to examine the structure of SPD in the total sample and among those with and without a SUD using Ruscio's (2010b) TaxProg programs in the R statistical program language. The first was the mean above minus below a cut (MAMBAC) procedure (Meehl & Yonce, 1994). The MAMBAC procedure starts with assigning one indicator (i.e., diagnostic criterion) to the role of input variable and another indicator (i.e., diagnostic criterion) the role of output variable, with each indicator serving in turn as the input indicator with each other indicator serving as output indicator. A cut is then made on the input indicator distribution. Each cut divides the data into two groups, cases falling above and below the cut score. The average score on the output variable is then calculated for each group. The mean differences are plotted along the y-axis of the MAMBAC graph, with the number of cases plotted along the x-axis. Next, the cut is made further up the input indicator and the difference between groups is again calculated. The cut continues to move progressively higher until it reaches the top of the distribution. MAMBAC curves show how the mean differences for cases above and below the cut vary with location of the cut points. We selected 50 cuts at equally-

spaced intervals between cases, beginning and ending 25 cases from either extreme. This procedure was repeated for each input-output indicator pair, yielding 90 curves associated with 10 SPD indicators ($n(n-1)$). Each set of curves were averaged to produce a single aggregate MAMBAC curve. Generally, taxonic constructs produce peaked MAMBAC curves whereas dimensional structures appear concave up or non-peaked (Meehl & Yonce, 1994). All MAMBAC procedures used 10 internal replications to smooth the shape of the MAMBAC curve (Ruscio & Ruscio, 2004a).

The second taxometric procedure was Maximum Eigenvalue MAXEIG (Meehl and Yonce, 1996). MAXEIG tests whether indicators of a construct covary because of a mixture of two underlying groups (taxonicity) or loadings on a common latent factor (dimensionality). In the MAXEIG procedure, two indicators in turn served as output indicators while the remaining indicators served as a composite input indicator. MAXEIG examines the association between output indicators within subsamples of cases ordered along an input indicator that forms the x-axis of the MAXEIG graph. Each indicator serves as input indicator while all other indicators serve as composite output indicators. The first (largest) eigenvalue of the covariance matrix of the output indicator is then calculated and plotted along the y-axis of the MAXEIG graph for each subsample, producing 10 (n) curves associated with the SPD analyses which were averaged. Cases were divided into 25 ordered fixed sized subsamples that overlapped 90.0% with adjacent subsamples as shown on the x-axis. Similar to MAMBAC, 10 replications were used. For prototypical categorical data, the MAXEIG curve is expected to be peaked whereas a non-peaked MAXEIG curve suggests dimensionality (Waller & Meehl, 1998).

The third taxometric procedure, L-Mode, conducts a principle axis factor analysis on the indicator variables and identifies the latent structure by calculating factor scores for all cases using a one-factor latent variable model (Ruscio, 2010a). The resultant L-Mode graph is the factor score density plot. For categorical data, the prototypical L-Mode graph is bimodal, each mode corresponding to a unimodal distribution of factor scores. For dimensional data, the prototypical L-Mode graphs show unimodal distributions of factor scores.

Taxometric graphs often deviate from their prototypical shape due to factors like levels of indicator validity, rendering interpretation difficult. It is therefore recommended to simulate via bootstrapping one or more sets of taxonic and dimensional comparison data using the same taxometric analyses used for the research data, in this case 100 data sets for taxonic and 100 for dimensional structures. Importantly, the resulting simulated datasets account for the mean, standard deviations, and indicator correlations of research data and differ only in latent structure (Ruscio & Ruscio, 2004a). Once the comparison data are generated and plotted, a comparison curve fit index (CCFI) quantifies the similarity of taxometric graphs yielded by the research data to those derived from simulated taxonic versus dimensional data (Ruscio & Ruscio, 2004a, 2004b). A CCFI value of 0.50 supports equally good fit with simulated dimensional and taxonic models, values from 0.0 to 0.50 support dimensionality and values from 0.50 to 1.0 support taxonicity.

Results

Indicator validity, nuisance covariance and base rates

As shown in Table 1, indicator validity was acceptable, the average Cohen's d exceeding 1.2 in all models. With one exception, validity of individual indicators associated with each model also exceeded 1.2. One to two of the ten SPD indicators associated with each of the six models failed to meet, but closely approximated this standard (Cohen's $d = 0.77$ – 1.18). Nuisance covariance was also acceptable (Table 1), with very low average indicator correlations in both taxon and complement groups but higher ones in the total sample ($r = 0.23$ – 0.26) for each model. Taxon base rates were 0.04, 0.07 and 0.03 for the total sample and among respondents with and without SUDs, respectively.

Taxometric analyses

Taxometric curves generated by MAMBAC and MAXEIG procedures for SPD for the total sample and among those with and without SUDs appear in Figures 1 and 2 (insert Figures 1 and 2 about here). Average CCFIs across the three taxometric procedures for the total sample and among respondents without a SUD were 0.440 and 0.365, respectively, reflecting dimensionality of SPD. However, among individuals with a SUD, the CCFI of 0.679 indicated taxonicity. As depicted in Figures 1 and 2, research data for the total sample and among respondents without a SUD fit the simulated dimensional comparison data well while the data for respondents with a SUD better fit the simulated taxometric comparison data. Consistent with MAMBAC and MAXEIG procedures, only L-Mode curves (not shown) associated with the subgroup with a SUD were bimodal, indicating taxonicity. L-Mode curves were unimodal for the total sample and among respondents without a SUD reflecting dimensionality underlying SPD in these groups.

Discussion

The underlying latent structure of SPD was clearly dimensional in the total sample and among respondents without any lifetime SUD. These findings are consistent with those of Ahmed and colleagues' (2013) taxometric analyses of the total NESARC sample using a reduced number ($n=3$) of factor-analytically derived scales of SPD symptom items. Conversely, SPD was clearly taxonic among individuals with a SUD. The taxonicity among respondents with a SUD is consistent with most prior taxometric studies of SPD (Blanchard, Gangstead, Brown, & Horan, 2000; Fossati, Raine, Borroni, & Maffei, 2007; Horan et al., 2004; Korfine & Lenzenweger, 1992; Linscott, Marie, Arnott, & Clarke, 2006; Meyer & Keller, 2001) that were largely conducted among undergraduates who are similarly at high risk for SPD and other schizophrenic spectrum disorders because of their SUD, or studies of children of schizophrenic parents who are at-risk because of hereditary predisposition (Erlenmeyer-Kimling, Golden, & Cornblatt, 1989; Tyrka et al., 1995b).

The dimensionality of SPD in the total sample and among individuals without a SUD is consistent with Esysenck's (1993) and Claridge's (2006) fully dimensional models in which a dimensional distribution of schizotypy represents a continuum of expression ranging from

non-disordered to subclinical manifestations to schizophrenia spectrum disorders, including SPD.

That SPD was taxonic among individuals with a SUD is consistent with Meehl's model suggesting that schizotypy corresponds to a neural integrative defect referred to a schizotaxia predisposed by a single discrete causal factor giving rise to the taxonic nature of schizophrenia and by extension its spectrum related disorders. Although specific etiologies can give rise to taxa, so can thresholds on latent continua (Gottesman & Shields, 1972) that may better explain the present results. That is, at lower levels of expressivity, SPD is dimensional, but as multiple genetic and environmental factors accumulate beyond a threshold among individuals at high risk of schizophrenia spectrum disorders the qualitatively distinct condition of SPD or related spectrum disorder emerges.

As previously noted, most taxometric studies on schizotypy or SPD have been conducted among undergraduates. The majority have found evidence for a nonarbitrary boundary between those with and without schizotypy or SPD. Unlike general population samples, undergraduates are at high risk of schizotypy and other schizophrenia spectrum disorders, including SPD, by virtue of their greater risk of SUDs relative to other age subgroups of the population. This contention is borne out in the taxonic findings of SPD among the subsample of NESARC respondents with a SUD. The demonstration of a latent categorical structure underlying DSM-IV SPD among individuals with SUDs does not imply the detection of a natural kind or entity. As cautioned by Meehl (2004), all that can be concluded with confidence is that the data appear to derive from a population in which there is a latent statistical discontinuity large enough to be noticeable. Although most taxometric studies have yielded evidence of categorical latent structures, it is unclear whether this evidence derives from research designs incorporating samples at high risk for schizophrenia spectrum disorders rather than disease etiology. Further taxometric analyses contrasting general population samples and subgroups at high risk for SPD and other schizophrenia spectrum disorders, including but not limited to those with SUDs, appears warranted. Taxometric analyses among high risk subgroups of the general population will certainly require much larger samples than those in most research conducted to date.

Meehl (2001) and others (Brown & Barlow, 2001; Widiger & Clark, 2000) predicted that research, including taxometric studies, would ultimately provide the evidence base for incorporating dimensional elements into classification systems such as DSM-5 in which disorders are defined exclusively as categories. Many researchers consider personality psychopathology as dimensional or as extreme ends of a continuum of normal personality variation (Coghill & Sonuga-Burke, 2012; Livesly, Schroeder, Jackson, & Jang, 1994; Widiger & Costa, 2005). Recent appeals have also been made to reorganize PDs along broad personality dimensions have included SPD (Cheilewski & Watson, 2008; Tackett, Silberschmidt, Krueger, & Sponheim, 2008). Debate over the role of dimensions in DSM-5 has also included recommendations to incorporate dimensional information while retaining categorical diagnoses (Helzer, Bucholz, & Gossop, 2007; Kraemer, 2007). This study's results suggest that purely categorical or purely dimensional representations may not adequately describe SPD. Rather, categorical representations reflecting the qualitative distinct entity of SPD emerging from the accumulation of risk factors (e.g., co-occurring

SUD, parental schizophrenia) beyond a threshold should be supplemented by dimensional components representing normal behavioral variation on a severity spectrum with schizophrenia spectrum disorders and contributing to schizophrenia liability.

The current study contributes to the taxometric literature on schizotypy and SPD through the use of a large general population sample, empirically supported consistency tests, examination of the structure of DSM-IV SPD and assessment of nonarbitrary boundaries of DSM-IV SPD among a high risk subgroup of individuals with a SUD. Limitations are noted. Like most prior taxometric research utilizing self-report measures of schizotypy or SPD, this study is subject to mono-operational bias (Shadish, Cook, & Campbell, 2002). Golden and Meehl (1992) note that taxometric analyses are ideally performed on indicators from independent domains such as self-report and neurocognitive performance, neurophysiological and other endophenotypes associated with SPD. This strategy is much less vulnerable to artifacts potentially inherent in measures operating at a single level of description and may more clearly reflect processes more closely related to SPD etiology.

In summary, this study found that DSM-IV SPD (identical to DSM-5 SPD) demonstrated dimensionality in the total sample and among individuals without a SUD. However, SPD appears taxonic among individuals with a SUD. Future methodologically stringent research on SPD and schizotypy among individuals with SUDs holds great promise in enhancing our knowledge of the latent structure and etiology of SPD, schizotypy and other schizophrenia spectrum disorders and the role of SUDs in the liability for these disorders.

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Total sample **Respondents with substance use disorders** **Respondents without substance use disorders** **Figure 1.**

Average MAMBAC curves: DSM-IV Schizotypal personality disorder in the total sample and among respondents with and without substance use disorders. Thick lines show the actual data, thin lines show the results for the corresponding comparison data that contain a gray band (when visible) that spans ± 1 standard deviations from the mean at each data point on the curve.

Total sample **Respondents with substance use disorders** **Respondents without substance use disorders** **Figure 2.**

Average MAXEIG curves: DSM-IV Schizotypal personality disorder in the total sample and among respondents with and without substance use disorders. Thick lines show the actual data, thin lines show the results for the corresponding comparison data that contain a gray band (when visible) that spans ± 1 standard deviations from the mean at each data point on the curve.

Table 1

Taxometric Parameter Estimates and Fit Statistics: DSM-IV Schyzotypal Personality Disorder among Respondents with and Without Substance Use Disorders

Model	Average nuisance covariance			Cohen's <i>d</i>	Supplied base rate	CCFI			Mean CCFI
	Total Sample	Taxon	Complement			MAMBAC	MAXEIG	L-Mode	
Total respondents (N = 34,653)	0.23	-0.01	0.13	2.033	0.045	0.567	0.337	0.384	0.440
Respondents with substance use disorder (N = 12,084)	0.25	-0.01	0.13	1.810	0.068	0.863	0.706	0.469	0.679
Respondents without substance use disorder (N = 22,569)	0.22	-0.02	0.13	2.197	0.031	0.472	0.301	0.321	0.365