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CONVERGING EVIDENCE FOR THE LATENT STRUCTURE OF ANTISOCIAL PERSONALITY DISORDER

Consistency of Taxometric and Latent Class Analyses

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The latent structure of antisocial personality disorder (ASPD) and psychopathy has been examined using both Meehl's taxometric method and other latent variable models, yielding results that have not been entirely consistent. However, in each instance, researchers used independent data sets for their analyses, obscuring whether the inconsistent findings resulted from the analytic method or from the study's methodology and data. A data set that had been used in a previous latent class analysis (LCA) of ASPD was examined using Meehl's taxometric procedures. Consistent with the prior LCA, the present analyses also supported a dimensional latent structure for ASPD. These converging findings from nonredundant data-analytic procedures provide additional evidence that ASPD is dimensional and exists along a continuum.

Keywords: antisocial personality disorder; taxometrics; latent structure

The question of whether personality disorders are underpinned by dimensions or discrete categories has been receiving considerable attention, in part because of the upcoming revision of the American Psychiatric Association's (1987) *Diagnostic and Statistical Manual of Mental Disorders (DSM)*; e.g., Widiger & Samuel, 2005; Widiger, Simonsen, Krueger, Livesley, & Verheul, 2005). Recent writings on psychological essentialism suggest that a number of people, especially some mental health professionals, are prone to seeing psychological disorders as natural kinds characterized by "discrete essences" (Ahn, Flanagan, Marsh, & Sanislow, 2006; Haslam, 2000; Lilienfeld & Marino, 1999), much like biologically distinct species. Moreover, research suggests that our minds tend to perceive categories even when they do not exist, in part because these categories simplify and streamline our cognitive processing (Macrae & Bodenhausen, 2000). Nevertheless, as Meehl and Golden (1982) noted, there are

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two major types of categories: those that exist solely in the minds of people (including clinical researchers and practitioners) and those that exist in nature—namely, taxa.

Meehl's taxometric method (e.g., Meehl & Golden, 1982; Meehl & Yonce, 1994) was developed specifically to empirically address the question of the latent structure of mental illnesses, including personality disorders, and thereby resolve the question of whether these conditions are underpinned by genuine categories in nature (taxa) or by dimensions. Numerous studies have used taxometric procedures to investigate whether one widely researched personality disorder—namely, psychopathy—is (a) categorical and qualitatively distinct from less severe forms of antisocial or disruptive behavior or (b) dimensional and existing along a continuum. Most of the recent research in this area (e.g., Edens, Marcus, Lilienfeld, & Poythress, 2006; Guay, Ruscio, Hare, & Knight, 2007; Marcus, John, & Edens, 2004; Murrie et al., 2007; Walters, Duncan, & Mitchell-Perez, 2007; Walters Gray, et al., 2007) has concluded that psychopathy has a dimensional latent structure (but see Vasey, Kotov, Frick, & Loney, 2005, for a recent exception that concluded that psychopathy is taxonic in a juvenile sample). In contrast, less attention has been devoted to examining the latent structure of antisocial personality disorder (ASPD). However, with increasing calls for replacing the Axis II personality disorder categories with dimensions (e.g., Widiger & Trull, 2007), further research on the latent structure of ASPD is warranted.

Although the first published taxometric study (Harris, Rice, & Quinsey, 1994) to use indicators drawn from the Psychopathy Checklist–Revised (PCL-R; Hare, 2003) concluded that there was a psychopathy taxon, closer inspection revealed that it was only items drawn from Factor 2 that yielded this apparent taxon. Because Factor 2 emphasizes antisocial behavior and lifestyle, it was possible to conclude that it is ASPD and not psychopathy that is taxonic. Using an overlapping forensic sample, this same research team (Skilling, Harris, Rice, & Quinsey, 2002) found further evidence of a taxon for antisocial behavior. Skilling, Quinsey, and Craig's (2001) taxometric analysis of antisocial behavior in a sample of boys from urban schools also yielded a low-base-rate taxon of severely antisocial boys. These findings led Haslam (2003) to conclude that the ASPD diagnosis should remain categorical and that “evidence for this taxon is strongest precisely in those aspects of the psychopathy construct that *DSM* embodies, namely, antisocial behavior with a history of childhood conduct problems” (p. 81).

However, using indicators drawn from the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II), Marcus, Lilienfeld, Edens, and Poythress (2006) concluded that ASPD was dimensional. Walters, Diamond, Magaletta, Geyer, and Duncan (in press) examined the latent structure of antisocial personality as assessed by the Antisocial Features scale of the Personality Assessment Inventory (Morey, 2007). They found consistent evidence for a dimensional latent structure both in their large sample of federal inmates and when they divided this sample into subsamples based on gender, ethnicity, and security level. Furthermore, in a review of studies that used latent variable modeling (in contrast to taxometric procedures) to examine the structure of externalizing disorders (including ASPD), Krueger, Markon, Patrick, and Iacono (2005) concluded that the evidence consistently supported a dimensional model. Most notably, Bucholz, Hesselbrock, Heath, Kramer, and Schuckit's (2000) latent class analysis (LCA) of data from the Collaborative Study of the Genetics of Alcoholism (COGA) concluded that ASPD was dimensional.

Thus, whereas studies that used latent class models other than taxometrics to examine the structure of ASPD and related conditions appear to converge on a dimensional structure, those that have used taxometric procedures have yielded inconsistent results, with some

supporting a taxonic structure and others supporting a dimensional structure.¹ Because the studies of ASPD and psychopathy using latent variable models—whether taxometric or otherwise—have used different data sets, the question of whether these methods yield consistent results when applied to the same data remains open. Waldman and Lilienfeld (2001) called for research linking taxometrics to other latent variable models. However, to our knowledge, Lenzenweger, McLachlan, and Rubin's (2007) latent structure analysis of schizotypy indicators using both MAXCOV (maximum covariance; Meehl & Yonce, 1996) and finite mixture modeling (FMM) is the only study to use both Meehl's taxometric method and a type of latent structure analysis with the same data set. In their study, both MAXCOV and FMM yielded a two-class structure.

However, given that the two sets of procedures are based on different models and assumptions; for example, taxometric procedures test whether there is a dimensional structure or two qualitatively distinct groups, whereas LCA can identify multiple groups; and taxometric procedures require unimodal distributions and zero within-group covariances, whereas LCA assumes multivariate normality and local independence (see Lenzenweger et al., 2007), there is the potential that the two sets of procedures will yield divergent structures. The present study applied taxometric procedures to a data set drawn from the COGA study that largely overlaps with the one used by Bucholz et al. (2000) in their LCA. If a series of taxometric analyses yields results that converge with this LCA, then this exercise in "critical multiplicity" (Cook, 1985) will increase our confidence in the conclusion that ASPD has a dimensional latent structure as well as our confidence in conclusions drawn from these nonredundant data-analytic procedures.

METHOD

PARTICIPANTS

The present study included 6,795 participants who were recruited for the larger Collaborative Study on the Genetics of Alcoholism (COGA; Bucholz et al., 2000). Participants were individuals classified by the *DSM-III-R* (APA, 1987) as alcohol dependent, their relatives, and controls. The majority of the participants were Caucasian (74.9%), 16% were African American, 6% were Hispanic, and 2.7% were "other." Approximately 55% of the participants were male ($n = 3,704$). Our sample is about 10% larger than the sample used by Bucholz et al. (2000; $N = 6,322$) because the COGA group continued to collect additional data after they completed their study.

MEASURE

ASPD was assessed using the ASPD section of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994). This interview follows the *DSM-III-R* diagnostic criteria for ASPD and includes questions about childhood conduct problems (e.g., truancy, vandalism) and adult antisocial behavior (e.g., arrests, financial irresponsibility). Bucholz et al. (1994) reported adequate 1-week test-retest reliability ($\kappa = .70$) for the ASPD section of the SSAGA. For diagnostic concordance between the ASPD sections of the SSAGA and the SCID, $\kappa = .71$ (Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999).

Because interviewers were instructed to skip the adult antisocial section of the SSAGA if respondents did not report at least two childhood conduct-disorder symptoms, there were

missing data for a substantial number of cases. Following Bucholz et al. (2000), these cases were excluded from the taxometric analyses. Because taxometric procedures do not permit missing data, other cases with missing data were also excluded. The exclusion criteria in the two studies appear to have resulted in similar exclusion rates: Bucholz et al. (2000) used 63% of the women and 89% of the men from their original data set, and the present study retained 62% of the women and 86% of the men from our original data set.

ANALYSIS

Two taxometric procedures were used: MAMBAC (mean above minus below a cut; Meehl & Yonce, 1994) and MAXEIG (maximum eigenvalue; Waller & Meehl, 1998). For MAMBAC, each indicator served as the output, with the remaining indicators summed to create the input, yielding one MAMBAC graph per indicator. Once cases were sorted on the input indicator, 50 cuts were located between equal numbers of cases. MAXEIG is a multivariate extension of MAXCOV. For MAXEIG, each indicator served as the input, with the remaining indicators serving as the output indicators, yielding one graph per indicator. Once cases were sorted on the input indicator, 200 windows were formed with an overlap of 90% between adjacent windows. To stabilize the shape of each MAMBAC and MAXEIG curve, each analysis was replicated 10 times by shuffling the cases with equal scores on the input indicator and recalculating the output value, with the average values across the 10 replications serving as the final results (Ruscio, Haslam, & Ruscio, 2006).

To help interpret the results for the research data, 10 samples of taxonic and dimensional comparison data were generated for each analysis (Ruscio, Ruscio, & Meron, 2007). Each sample of comparison data reproduced the indicators' distributions and correlations using either a taxonic or a dimensional structural model. To generate taxonic comparison data, cases were assigned to putative groups using the base-rate classification method (Ruscio et al., 2007). The base rate was estimated for each MAMBAC and MAXEIG analysis, using the general covariance mixture theorem for the latter (see Ruscio et al., 2006, Appendix C), so that taxonic comparison data were tailored to each analysis. Submitting comparison data to the same taxometric analyses as the research data affords a visual inspection of whether the results for the research data are more similar to those of the taxonic or dimensional comparison data. Also, this approach can be used to calculate the Comparison Curve Fit Index (CCFI), an interpretational aid that distinguishes taxonic from dimensional data with excellent validity (Ruscio et al., 2006; Ruscio et al., 2007). The validity of this index surpassed that of each of the commonly used taxometric consistency tests to which it was compared, and it appears to work especially well when the putative taxon is small (Ruscio, in press; Ruscio & Marcus, 2007; Ruscio et al., 2007). CCFI values can range from 0 (*strongest support for dimensional structure*) to 1 (*strongest support for taxonic structure*), with a value of .50 representing ambiguous results.

RESULTS

CONSTRUCTION OF INDICATORS

Because most of the individual items were highly skewed, with few affirmative responses, and because indicators based on more than one or two items are preferred for taxometric

analyses (Beauchaine, 2003; Cole, 2004), we combined items to create indicators. We factor analyzed the 35 ASPD items using an oblique (promax) rotation to inform our construction of indicators. A scree test indicated a four-factor solution that accounted for 29% of the variance. The first indicator ($\alpha = .70$) was characterized by an irresponsible lifestyle and consisted of nine adult items (e.g., defaults on debts, quits jobs). The second indicator ($\alpha = .67$) involved violence against other people and consisted of four child items (e.g., fighting) and six adult items (e.g., mugging). The third indicator ($\alpha = .60$) was characterized by destruction of property and consisted of four child items (e.g., vandalism) and two adult items (arson). The fourth indicator ($\alpha = .57$) was characterized by deceitfulness and consisted of four child items (e.g., truancy) and one adult item (lying). Two items (fighting and hitting spouse or offspring) loaded on Factors 1 and 2. Because both involved violence against others, they were included in the second indicator.

Taxometric analyses require the use of valid indicators that are capable of discriminating between members of presumptive taxon and complement groups. Validity estimates were computed by using the SSAGA interviews to determine whether participants met the *DSM-IV* diagnostic criteria for ASPD. Among the individuals included in the analyses, 12.8% met the *DSM-IV* criteria for ASPD. For the four indicators, the average separation between those who met the criteria for ASPD and those who did not was 2.00 standard deviation units, which exceeded the recommended minimum of $d = 1.25$ (Meehl, 1995). Furthermore, nuisance covariance (i.e., high correlations among presumptive members of the taxon or complement groups; Meehl & Golden, 1982) did not appear to be a problem. The average correlation among these four indicators in the entire sample was $r = .42$. In contrast, the average correlation of these four indicators was considerably lower ($r = -.14$) among those participants who are unlikely to be members of an ASPD taxon because their total score on all four indicators was in the lowest quartile. Similarly, among those participants most likely to be members of an ASPD taxon (i.e., total scores in the highest quartile), the average correlation among the indicators was $r = .15$.

TAXOMETRIC ANALYSES

None of the four MAMBAC graphs displayed the inverted U-shape indicative of taxonic structure. Instead all four graphs exhibited a rising cusp on the right side of the graph that could either be indicative of a low base-rate taxon or positively skewed indicators.² The indicators were skewed to a substantial extent (M skew = 1.83), which makes comparison with simulated data especially informative for a proper interpretation of these results (Ruscio & Marcus, 2007; Ruscio, Ruscio, & Keane, 2004). The average of the four MAMBAC curves, juxtaposed with MAMBAC curves for simulated dimensional and taxonic data, is provided in Figure 1. It is clear that the actual data are much more similar to the simulated dimensional data than to the simulated taxonic data, and the CCFI of .30 quantitatively corroborates this visual interpretation. Whereas the taxonic graph peaks on the right side, consistent with a low-base-rate taxon, the graphs for the research data and the simulated dimensional data rise steadily to right-end cusps.

None of the four MAXEIG curves displayed a peak indicative of a taxon. It is apparent from the average of these four curves, when juxtaposed with the simulated data (see Figure 2), that the research data are more similar to the simulated dimensional data than to the simulated taxonic data (CCFI = .26). Whereas the simulated taxonic data yielded a clear peak,

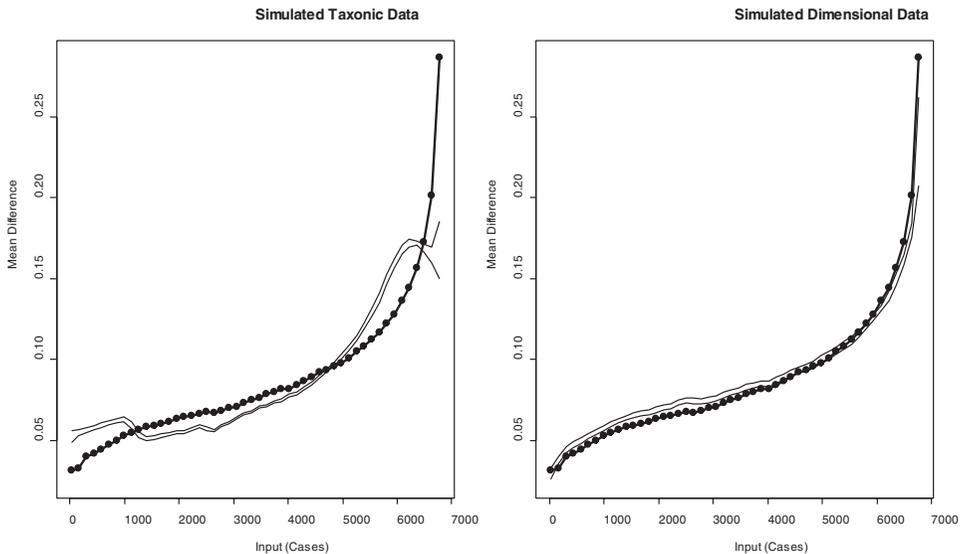


Figure 1: Average MAMBAC Curves for the Research Data, Simulated Taxonic Data, and Simulated Dimensional Data for the Four Indicators Derived Via Factor Analysis From the ASPD Section of the SSAGA Interview

Note. The dark line represents the actual data, and the lighter lines represent one standard deviation greater than and less than the average for each simulated data set.

the research data and the simulated dimensional data both rose steadily across the graph, consistent with a dimensional construct measured with positively skewed indicators (Ruscio et al., 2004).

SUPPLEMENTARY ANALYSES

Additional analyses were conducted, but given space constraints, these results will be reviewed only briefly. Readers may contact the first author for copies of these graphs and results. An L-Mode analysis (Waller & Meehl, 1998) yielded a unimodal graph, as would be expected for a dimensional construct. Two sets of additional taxometric analyses—using MAMBAC, MAXEIG, and L-Mode—were also conducted, separating the sample by sex. All of these analyses yielded results as supportive of a dimensional structure as those presented above.

DISCUSSION

Consistent with other recent taxometric studies of ASPD (Marcus et al., 2006) and the related condition of psychopathy (Guay et al., 2007), the present study yielded consistent evidence that ASPD has a dimensional latent structure. These results from a nonforensic sample complement the findings from prior studies that used data primarily from offender samples (i.e., prison inmates and individuals court-ordered into residential treatment). Because the two studies used data drawn from the same larger study, it is encouraging that

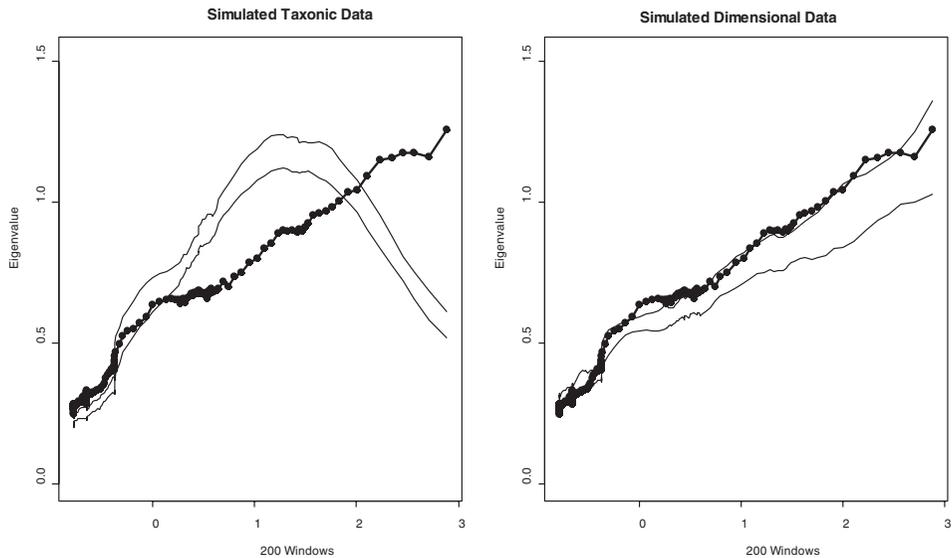


Figure 2: Average MAXEIG Curves for the Research Data, Simulated Taxonic Data, and Simulated Dimensional Data for the Four ASPD SSAGA Factor Scores

Note. The dark line represents the actual data, and the lighter lines represent one standard deviation greater than and less than the average for each simulated data set.

the present taxometric results were highly consistent with those from Bucholz et al.'s (2000) LCA.

Prior taxometric articles concerning ASPD and psychopathy that have yielded dimensional results (e.g., Edens et al., 2006; Guay et al., 2007; Marcus et al., 2004, 2006; Walters, Diamond, et al., 2007) have already discussed many of the implications of a dimensional latent structure for the understanding, measurement, and etiology of these disorders. For example, dimensional results suggest that ASPD is not due to a single dichotomous causal factor, such as presence versus absence of early physical abuse. In terms of diagnosis, these findings suggest that unless there is a clear pragmatic or clinical justification for setting a diagnostic cutoff (e.g., blood sugar levels greater than a specific value are associated with increased morbidity and mortality justifying the cut score for diagnosing type 2 diabetes), antisocial personality is best treated as a continuous dimension and not as a categorical diagnosis. Similarly, assessment instruments for measuring antisocial personality that are designed to provide a continuous range of scores are likely to be more informative than ones that dichotomize ASPD as either present or absent.

One of Meehl's primary reasons for developing his taxometric method was to inform the development of an empirically based taxonomy of psychopathology (e.g., Meehl, 2001). Instead of nosologists deciding whether a disorder was taxonic or dimensional based on their preferences for categories or continua, Meehl (1999) anticipated that the taxometric method would provide an empirical answer to these debates. However, there has been some skepticism about the value of this approach (e.g., Krueger et al., 2005) and little reference to taxometric studies in the recent calls for developing a dimensional classification of personality disorders (e.g., Widiger et al., 2005; Widiger & Trull, 2007).

Although the findings from the present study support a dimensional structure for ASPD and we agree with Meehl's (1992) speculation that most psychiatric diagnoses will prove to have a dimensional structure, we also strongly believe that findings from taxometric studies can contribute to the development of an evidence-based diagnostic system. There is, for example, some evidence that schizotypal personality disorder is taxonic (e.g., Lenzenweger, 1999) and a wholesale revision of Axis II from categories to dimensions risks throwing out the baby with the bathwater. Instead systematic evidence from studies that assess latent structure using a variety of methods—both taxometric and latent variable modeling—can best inform future diagnostic systems.

Two of the primary limitations of the present study stem from the COGA database and thus are shared with Bucholz et al. (2000). Because the COGA database included a disproportionate number of individuals with alcohol abuse problems, it is open to the criticism that some of the antisocial behavior reported by the respondents might have been a consequence of their substance abuse. Thus, the endorsement of antisocial acts by substance-abusing individuals who do not actually have ASPD may have obscured the identification of an ASPD taxon. However, separate taxometric analyses of data from samples of prison inmates and individuals who were court-ordered into residential substance-abuse treatment facilities yielded highly similar, unambiguously dimensional results (Marcus et al., 2006). Still, future research examining the latent structure of ASPD in a large nonforensic sample of individuals who do not abuse substances would be a useful complement to extant literature. The second limitation derives from the SSAGA's skip-out structure. These interview rules eliminated a large number of cases that would probably have been classified as members of the complement group if ASPD had a taxonic latent structure. However, given that the vast majority of the current sample (87%) did not meet the diagnostic criteria for ASPD, it seems likely that there were sufficient complement members to have identified a taxon had one been present. In addition, the consistency of the present findings with those of studies that did not use a skip-out structure (e.g., Krueger et al., 2005; Marcus et al., 2006) bolsters our confidence in the current findings.

The present study appears to be one of the first to follow Waldman and Lilienfeld's (2001) suggestion that researchers compare taxometric procedures with other latent variable techniques using real data. The COGA sample, like all samples, has its limitations, but the findings of Bucholz et al. (2000) have been cited frequently (at least 19 times in the past 7 years) as evidence for the dimensional structure of ASPD, and the present findings corroborate this inference from these data. This represents a promising convergence between taxometric procedures and LCA and suggests an area for further exploration with other clinical data sets (e.g., studies that use latent variable models to reanalyze data sets from taxometric studies).

NOTES

1. However, the apparent inconsistency in the results across taxometric studies may be attributable to important, but insufficiently appreciated, methodological and conceptual differences. When methodological safeguards are implemented to minimize these weaknesses, clearer and more consistent findings of dimensional structure have resulted (see Edens et al., 2006; Guay et al., 2007; Lilienfeld, 1998; Marcus et al., 2004; Marcus et al., 2006; and Murrie et al., 2007, for detailed critiques).
2. Copies of these and all subsequent graphs not presented in the article are available on request from the first author.

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