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Is antisocial personality disorder continuous or categorical? A taxometric analysis

DAVID K. MARCUS[1]*, SCOTT O. LILIENFELD[2], JOHN F. EDENS[3] AND NORMAN G. POYTHRESS[4]

[1] *Department of Psychology, University of Southern Mississippi, Hattiesburg, MS, USA*; [2] *Department of Psychology, Emory University, Atlanta, GA, USA*; [3] *Department of Psychology, Southern Methodist University, Dallas, TX, USA*; [4] *Department of Mental Health Law and Policy, University of South Florida, Tampa, FL, USA*

ABSTRACT

Background. Although the DSM-IV-TR is organized into discrete disorders, the question of whether a given disorder possesses a dimensional or a categorical latent structure is an empirical one that can be examined using taxometric methods. The objective of this study was to ascertain the latent structure of antisocial personality disorder (ASPD).

Method. Participants were 1146 male offenders incarcerated in state prisons ($n=569$), or court-ordered to residential drug treatment ($n=577$). Participants were interviewed using the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) to assess ASPD symptoms; they also completed the Personality Diagnostic Questionnaire-4 (PDQ-4) ASPD scale. Taxometric analyses were performed to examine whether ASPD is underpinned by a discrete category or a dimensional construct.

Results. Multiple taxometric procedures using two different sets of indicators provided no evidence that ASPD has a taxonic latent structure. Instead, the results were far more consistent with the proposition that ASPD exists on a continuum, regardless of whether it is assessed using a structured interview or a self-report measure.

Conclusions. Evidence that ASPD is dimensional suggests that it is best studied using continuous measures and that dichotomizing individuals into ASPD *versus* non-ASPD groups will typically result in decreased statistical power. The findings are also consistent with a multifactorial etiology for ASPD and with recent attempts to conceptualize ASPD within the framework of extant dimensional models of personality.

INTRODUCTION

A fundamental question in the classification of psychiatric disorders concerns their latent structure. Do these disorders identify discrete conditions (such as influenza) or dimensions that vary quantitatively but not qualitatively from non-pathological conditions (such as essential hypertension)? The authors of the *Diagnostic and Statistical Manual of Mental Disorders* (4th edn, text revision) (DSM-IV-TR; APA, 2000) sidestepped this issue by stating that 'there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder' (p. xxxi). Nonetheless, for largely pragmatic purposes the DSM-IV-TR is organized into discrete disorders.

For much of the history of psychiatric nosology, the question of latent structure has been a source of debate with few data. However,

* Address for correspondence: Dr D. K. Marcus, Department of Psychology, University of Southern Mississippi, 118 College Dr. #5025, Hattiesburg, MS 39406-0001, USA.
(Email: david.marcus@usm.edu)

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taxometric procedures developed by Meehl and colleagues (Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998) allow researchers to test whether a diagnosis has a taxonic (categorical) or a dimensional (continuous) latent structure. Although this research is still relatively preliminary, studies suggest, for example, that schizotypy (Lenzenweger & Korfine, 1992) may be categorical, whereas many forms of depression (Slade & Andrews, 2004) are likely to be dimensional. In all likelihood some DSM-IV-TR disorders will ultimately prove to have a categorical structure, whereas others will be dimensional.

Theorists have often assumed that many, if not most, of the personality disorders have a dimensional latent structure. Attempts to conceptualize personality disorders within the rubric of the five-factor model of personality are consistent with this assumption (Widiger, 2005). Surprisingly, a recent review of taxometric studies of three personality disorders concluded that the research favors a categorical structure for schizotypal, antisocial and borderline personality disorders (Haslam, 2003).

However, recent taxometric research indicates that borderline personality disorder is more likely to have a dimensional latent structure (Rothschild *et al.* 2003). Similarly, the picture may be more complicated for antisocial personality disorder (ASPD), particularly because of the ambiguity regarding the conceptualizations of ASPD and the related construct of psychopathy. Although the accompanying text of DSM-IV-TR asserts that ASPD is essentially synonymous with psychopathy (p. 702), numerous studies indicate that measures of these two conditions are only moderately correlated and exhibit distinctive personality and psychopathological correlates (Harpur *et al.* 1989; Lilienfeld, 1998; Hare, 2003). Whereas ASPD, as defined by the DSM-IV-TR, is characterized by a lifetime history of antisocial behavior (evidence of childhood conduct disorder, repeated unlawful behavior), the core features of psychopathy are a set of personality traits, including callousness, egocentricity and remorselessness, with only remorselessness listed as a diagnostic criterion for ASPD. Moreover, the majority of prison inmates with ASPD do not meet Psychopathy Checklist (PCL) criteria for psychopathy (e.g. Hart & Hare, 1989). Thus, ASPD (unlike the ICD diagnosis of dissocial personality disorder, which appears to be closer to psychopathy in its emphasis on affective and interpersonal deficits) is likely to be a heterogeneous category that includes psychopathic individuals, dissocial individuals influenced by deviant subgroup norms, and a variety of other criminal individuals (Lilienfeld, 1998; Lykken, 1995), making it unlikely that the broad diagnosis of ASPD could be a discrete category.

Taxometric studies by Harris and his associates (Harris *et al.* 1994; Skilling *et al.* 2002) appeared to provide evidence for an ASPD taxon; both studies used similar measures and overlapping samples. However, these studies had significant methodological limitations that increased the risk of 'pseudo-taxonic' results, including (*a*) the failure to include interview data, which is the preferred method of scoring of the Psychopathy Checklist-Revised (PCL-R), (*b*) dichotomizing PCL-R scores instead of using the standard three-point scoring system, (*c*) item-total correlations that were considerably higher than those reported in the PCL-R manual (raising the possibility that the PCL-R may have been scored with an expectation of a psychopathy taxon), and (*d*) the use of large numbers of mentally ill inmates, who may have inadvertently yielded a schizotypy taxon (Lilienfeld, 1998; Edens *et al.* 2006).

Additionally, although much of the evidence for an ASPD taxon comes from the analysis by Harris *et al.* of items drawn from Factor II of the PCL-R, which is the factor that assesses antisocial behavior, several subsequent taxometrics studies failed to find evidence for a taxon for psychopathy or for the construct assessed by PCL-R Factor II (Marcus *et al.* 2004; Edens *et al.* 2006; Guay *et al.* in press). Finally, studies that have used latent class and latent trait models have found that ASPD in particular (Bucholz *et al.* 2000) and externalizing disorders in general (Krueger *et al.* 2005) are best conceptualized as existing along a continuum.

The present study examined the latent structure of ASPD using (*a*) a larger and more appropriate sample than had been used in previous studies, (*b*) two methods of assessing ASPD, namely a well-validated and widely used semi-structured interview and a self-report measure, and (*c*) simulation methods that permit researchers to distinguish between taxonic structures and pseudo-taxonic results that can

result from skewed measures (Ruscio *et al.* in press). These improved methods should provide more definitive conclusions about the latent structure of ASPD.

METHOD

Participants

Participants were 1146 male offenders who were interviewed using the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First *et al.* 1997) as part of a larger NIMH-funded study. A subset of these participants ($n=876$) was used in the Edens *et al.* (2006) taxometric study of PCL-R-defined psychopathy. The participants were incarcerated in state prisons in Florida, Nevada, Utah or Oregon ($n=569$), or court-ordered to residential drug treatment in Florida, Texas, Utah, Nevada or Oregon ($n=577$). Approximately 58% of the sample identified themselves as Caucasian, 34% as African American, and 8% as Hispanic. The mean age was 30·30 years (S.D. = 6·60). Potential participants were selected randomly from lists of individuals who met basic inclusion criteria: Euro-American or African-American, age range 21–40 (inclusive), estimated IQ $\geqslant$ 70 on an IQ screen, spoke English, and were not receiving psychotropic medication for active symptoms of psychosis. The entire protocol took on average 4·5 hours to complete and was administered in two or three sessions. Participants received US$20, except at one agency that did not permit payments. After complete description of the study, written informed consent was obtained from all participants.

Measures

Participants completed an extensive research protocol related to the objectives of the larger project. We describe only those measures relevant to the taxometric analyses.

SCID-II

The ASPD module of the SCID-II (First *et al.* 1997) is a widely used semi-structured psychiatric interview that assesses the DSM-IV criteria for ASPD, yielding both dimensional and categorical scores. The interviewers for this study were clinical psychology graduate students trained by a senior investigator. The ASPD module exhibits adequate levels of inter-rater reliability (mean $\kappa=0{\cdot}72$; Maffei *et al.* 1997; Dreessen & Arntz, 1998). In this study, the inter-rater reliabilities (κ) were 0·74 ($n=50$) for ASPD diagnoses and ICC_1 was = 0·86 ($n=46$) for dimensional ASPD symptom counts.

Personality Diagnostic Questionnaire-4 (PDQ-4) ASPD scale (Hyler, 1994)

The PDQ-4 ASPD scale is a self-report questionnaire consisting of 22 true–false items, one for each ASPD childhood and adult criterion in DSM-IV. The κ coefficient for diagnostic concordance between the PDQ-4 and SCID-II ASPD scales was 0·49 and the receiver operating characteristics (ROC) analyses suggested that this scale could serve as a useful screening measure for ASPD in prison samples (Davison *et al.* 2001). The PDQ-4 ASPD scale provided an alternative (and lower threshold) indicator of ASPD in addition to the SCID-II. In the present sample, PDQ-4 total scores ($\alpha=0{\cdot}85$) correlated 0·55 ($p<0{\cdot}001$) with total SCID-II ASPD scores.

Data analyses

The data were analyzed using a set of taxometric procedures. For taxonic conditions, these procedures detect a qualitative distinction between taxon members and those without the disorder (complement). Dimensional data lack this qualitative difference. The taxometric method applies mathematically distinct procedures to several combinations of indicator variables (i.e. measures of distinct aspects of the condition). These procedures produce a set of graphs and the shape of the graphs should converge on a taxonic or dimensional structure. Each procedure also produces one or more estimates of the base rate of the taxon in the sample. Although consistent base rate estimates between and within procedures were thought to be indicative of a taxonic latent structure, recent simulation studies by Ruscio (unpublished observations) suggest that constructs with dimensional latent structures can also yield consistent base rate estimates.

We used four taxometric procedures to analyze the data: Mean Above Minus Below A Cut (MAMBAC; Meehl & Yonce, 1994), MAXimum COVariance (MAXCOV; Meehl & Yonce, 1996) or MAXimum EIGenvalue (MAXEIG; Waller & Meehl, 1998), and Latent

Mode factor analysis (L-Mode; Waller & Meehl, 1998). In MAMBAC, MAXCOV and MAXEIG, one measure serves as the input indicator and is plotted on the *x* axis. In MAMBAC, cuts are made along an input indicator and the mean scores on the output indicator (another measure of the construct) for those cases above the cut and for those cases below the cut are computed. The difference between these two means is plotted on the *y* axis. Taxonic graphs will produce a single peak, with the location of the peak reflecting the base rate of the taxon. Dimensional graphs will typically appear concave rather than peaked. For the MAXCOV analyses, the graph was segmented into a succession of overlapping windows along the input indicator. The covariances between the other two indicators for each window were plotted on the *y* axis. MAXEIG, a multivariate extension of MAXCOV, uses eigenvalues instead of covariances. If taxonic, the covariance (or eigenvalue) should be maximal in the window most evenly divided between members of the taxon and complement and the graph peaks at this cut. The graph will appear concave, flat or irregular if the construct is dimensional. Fifty windows with 0·90 overlap were used for the analyses in the present study. In L-Mode, all of the indicators are factor analyzed and the distribution of scores on the first principal factor is plotted. If the construct is taxonic, the graph will be bimodal, whereas a unimodal graph is more consistent with a dimensional interpretation (for a detailed description of these procedures see Waller & Meehl, 1998; Ruscio *et al.* 2006).

All taxometric analyses were performed using software developed by Ruscio (2006). Ruscio's programs produce simulated taxonic and dimensional datasets that match the parameters of these data (e.g. indicator correlations, skew, kurtosis). Ten taxonic and 10 dimensional simulations were created for each set of indicators. To aid interpretation of the results, graphs of these simulated datasets can be compared with graphs of the actual data. These simulations are especially useful because skewed (or otherwise non-normal) data can yield ambiguous graphs. In Monte Carlo studies this simulation method has outperformed other methods for interpreting taxometric results (Ruscio *et al.* in press). Ruscio's programs calculate a curve fit index that ranges from 0 (consistent with a dimensional structure) to 0·5 (ambiguous – equally consistent with either structure) to 1 (consistent with a taxonic structure).

RESULTS

Overall, 58% (661) of the participants met the DSM-IV-TR criteria for ASPD. Taxometric procedures are most sensitive when the base rate of the construct in question is about 50%, so if ASPD is taxonic and the DSM-IV-TR diagnostic cut-off approximates to the true cutting score for this taxon, then the present sample should be ideal for identifying this putative taxon. Because our sample included participants from both prisons and residential drug treatment programs, it ran the risk of yielding a 'pseudo-taxon' (e.g. instead of identifying a unique psychiatric disorder, the analyses could be identifying prison inmates). Fortunately, finding that almost identical percentages of prison inmates (57·5%) and drug program participants (57·9%) met the DSM-IV-TR criteria for ASPD makes it unlikely that the taxometric analyses will produce a pseudo-taxon. As a further precaution, we also reran the taxometric analyses separately for the two samples. These analyses were consistent with the results from the entire sample, so only the results from the entire sample are reported below.

There was a moderately high correlation ($r = 0{\cdot}63$) between the SCID-II and the PCL-R total in the present sample, suggesting that although ASPD and psychopathy are related constructs, the relation was far from unity given that they only share 40% of their variance. To put this relationship in context, it is worth noting that this value is similar to the correlation between anxiety and depression (Watson *et al.* 1995), two other related but separable constructs. Therefore, this taxometric analysis of ASPD is non-redundant with our earlier analysis of PCL-R-assessed psychopathy (Edens *et al.* 2006).

The taxometric analyses were performed on three sets of indicators. First, we factor analyzed all 22 items from the SCID-II. Because more than half of the SCID items pertain to childhood conduct disorder and accounted for three of the four factors that were extracted, we also selected individual items from the SCID-II for a second set of analyses. These two strategies for

Table 1. *Four factor-based indicators from the SCID-II Antisocial Personality Disorder Items*

Item	Loading on			
	Factor 1	Factor 2	Factor 3	Factor 4
1. Adult Antisocial				
Unlawful Behavior	**0·59**	0·09	−0·04	−0·03
Deceitfulness	**0·66**	0·13	0·02	−0·09
Impulsivity	**0·51**	0·25	−0·22	0·15
Recklessness	**0·76**	−0·15	0·03	−0·07
Irresponsibility	**0·73**	−0·01	−0·12	0·02
Lacks Remorse	**0·50**	−0·25	0·27	−0·09
2. Childhood Non-assaultive Crimes				
Breaking and Entering	−0·02	**0·56**	0·20	0·03
Lying/Conning	0·21	**0·47**	0·02	0·06
Theft/Forgery	0·10	**0·49**	−0·05	0·23
Running Away	−0·05	**0·70**	−0·03	−0·10
Stay out late	−0·07	**0·70**	0·10	−0·13
Truancy	−0·04	**0·64**	0·07	−0·20
3. Physical Violence				
Childhood Bullying	−0·03	−0·01	**0·51**	0·29
Childhood Fights	−0·01	0·09	**0·69**	0·05
Childhood Weapon Use	−0·01	0·05	**0·69**	0·04
Childhood Cruelty to People	−0·05	−0·02	**0·68**	−0·01
Childhood Robbed/Mugged	−0·04	0·19	**0·63**	−0·10
Adult Fights	**0·39**	−0·05	**0·44**	0·03
4. Childhood Severe Antisocial Behaviors				
Cruelty to Animals	−0·01	−0·27	0·11	**0·74**
Fire Setting	−0·10	0·06	−0·11	**0·76**
Vandalism	0·02	0·23	0·13	**0·44**

$n = 1146$. Loadings of $\geqslant 0{\cdot}30$ are shown in bold.

selecting indicators complemented one another. The factor scores have the advantage of providing more psychometrically sound indicators and a wider range of scores, whereas the individual item indicators placed a greater weight on adult antisocial symptoms. Finally, because the SCID-II is scored by the interviewer and some evidence suggests that rater expectations concerning the latent structure of a construct may influence the results of a taxometric study (Beauchaine & Waters, 2003), a final set of taxometric analyses used participants' self-reported responses to the PDQ-4. Consistency in the results from these three sets of indicators will increase confidence that the analyses revealed the latent structure of ASPD.

Analysis of factor-level SCID indicators

Because indicators that are based on more than one or two items are generally preferred for taxometric analyses (Beauchaine, 2003; Cole, 2004), we created factor scales by using a promax rotation to factor analyze all 22 adult and child ASPD items from the SCID-II. Using the Kaiser criterion (i.e. eigenvalues >1) and scree test, we obtained a four-factor solution that accounted for 43% of the variance (see Table 1). Factor 1 (23·1% of the variance; eigenvalue = 5·07, $\alpha = 0{\cdot}68$) consisted of six adult antisocial items. Factor 2 (8·5% of the variance; eigenvalue = 1·88, $\alpha = 0{\cdot}70$) consisted of six childhood non-violent criminal behaviors. Factor 3 (6·2% of the variance; eigenvalue = 1·36, $\alpha = 0{\cdot}75$) consisted of six items describing bullying or physical assault. Factor 4 (4·9% of the variance; eigenvalue = 1·09, $\alpha = 0{\cdot}47$) consisted of three destructive childhood behaviors. Taxometric analyses require the use of valid indicators that are capable of discriminating between members of a presumptive taxon and complement group. Based on the subsequent MAMBAC, MAXEIG and L-Mode analyses, the average degrees of separation for these four indicators were 1·59, 1·27 and 1·52 standard deviation units, respectively, which exceeded the recommended minimum of 1·25 (Meehl, 1995). In addition, when the sample was divided into participants who met the DSM diagnostic criteria and those who did not, the average indicator validity was 1·33. Finally, there was not a

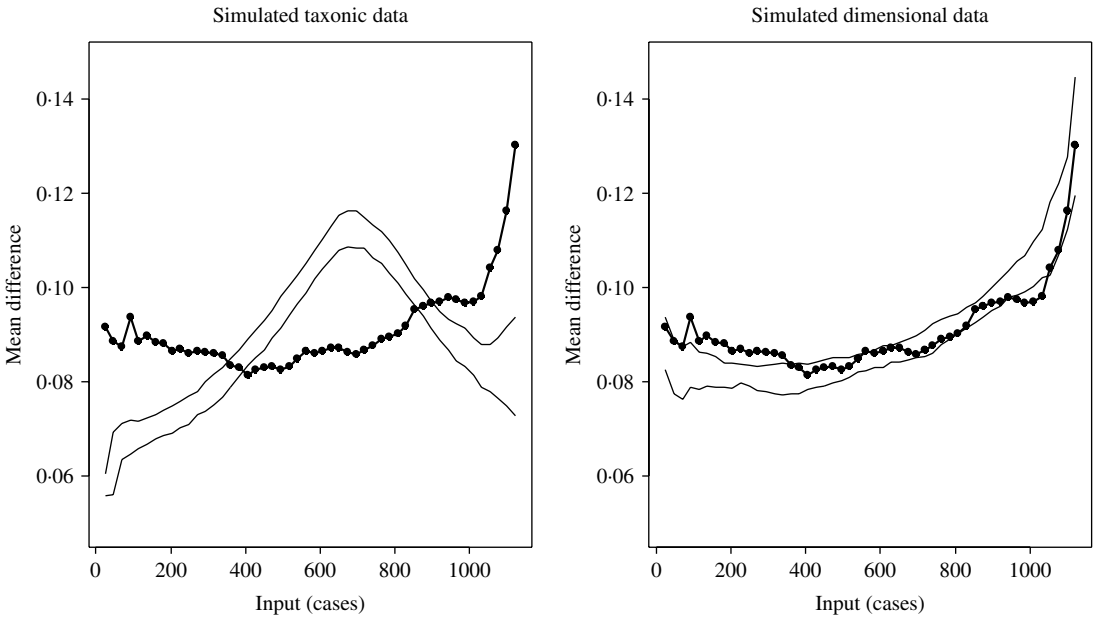

FIG. 1. Average MAMBAC (Mean Above Minus Below A Cut) curves for the research data, simulated taxonic data, and simulated dimensional data for the four SCID-II antisocial personality disorder (ASPD) factor scores. For each curve, 50 cuts were made along the input indicator. The graphs for the simulated taxonic and dimensional data were produced by generating 10 datasets for each latent structure. The dark line represents the actual data and the lighter lines represent one standard deviation above and below the average for each simulated dataset.

problem with nuisance covariance (high correlations among the members of the putative taxon and complement groups) because the average correlation among these four indicators in the entire sample was 0·39, whereas the average correlation among those who met the ASPD criteria (0·18) and those who did not (0·13) was considerably lower.

None of the four MAMBAC curves (each factor score served as the output indicator for one graph with the remaining three factors scores summed to create the input indicator) exhibited the inverted-U shape consistent with a taxonic structure. These curves yielded base rate estimates ranging from 0·32 to 0·55 (mean = 0·42, S.D. = 0·10). The average of these four MAMBAC curves juxtaposed with the graph for the simulated taxonic and dimensional datasets are presented in Fig. 1. In these graphs, the actual data appeared much more like the simulated dimensional data than the simulated taxonic data; the curve fit index (0·12) was also highly indicative of a dimensional structure.

None of the four individual MAXEIG curves displayed the clear single peak indicative of a taxonic structure. These curves yielded base rate estimates ranging from 0·28 to 0·60 (mean = 0·42, S.D. = 0·16). The average of these MAXEIG curves juxtaposed with the graphs for the simulated taxonic and dimensional datasets are presented in Fig. 2. Although the graph of the simulated taxonic data has a clear peak, the MAXEIG graph of the actual data appears flat, as does the graph of the simulated dimensional dataset. The curve fit index (0·17) demonstrates that the actual data were far more consistent with a dimensional structure.

The L-Mode curve for the actual data was unimodal and was similar to the simulated dimensional data (Fig. 3). By contrast, the simulated taxonic data was clearly bimodal. Averaging the base rate estimates from the left (0·13) and right modes (1·0) yielded a 0·57 base rate estimate, and the base rate estimate based on the classification of cases was 0·52. L-Mode base rate estimates of about 0·50 are typical for dimensional constructs (Waller & Meehl, 1998). Overall, the results from the factor scores were consistent with a dimensional latent structure.

Because (*a*) Factor 4 had considerably lower internal consistency than the three other factors,

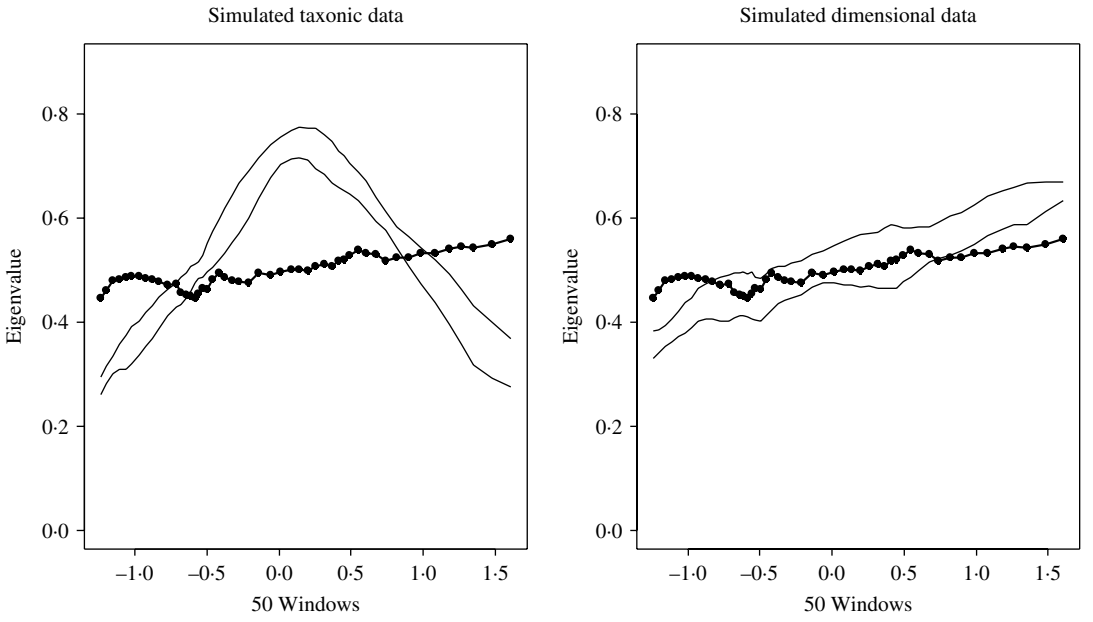

FIG. 2. Average MAXEIG (MAXimum EIGenvalue) curves for the research data, simulated taxonic data, and simulated dimensional data for the four SCID-II antisocial personality disorder (ASPD) factor scores. For each curve, the data were sorted along the *x* axis by the scores on the input indicator and then grouped into 50 subsamples using overlapping windows (0·90 overlap). The graphs for the simulated taxonic and dimensional data were produced by generating 10 datasets for each latent structure. The dark line represents the actual data and the lighter lines represent one standard deviation above and below the average for each simulated dataset.

(*b*) it was the only factor to yield validity indicators less than 1·25, and (*c*) less valid indicators could yield inaccurate dimensional results, all of the taxometric analyses were rerun with Factors 1–3 only. These three indicators displayed strong validity (average validity 1·43, minimum 1·32, when the sample is divided into those who do and do not meet the criteria for ASPD), yet the results of these analyses were also unambiguously dimensional. Alternatively, indicators were also created by forcing the factor analysis into a three-factor solution. The results of these analyses were also clearly dimensional. (Copies of all of these results and all subsequent taxometric analyses that are not provided in this paper are available from the first author.)

Analysis of individual-item SCID indicators

The SCID-II interview for ASPD consists of seven items that assess adult antisocial behavior and 15 items that assess childhood/adolescent conduct disorder. These 15 items were averaged into a single indicator of childhood/adolescent conduct disorder symptoms. This scoring resulted in eight potential indicators. However, SCID-II item 1 (fails to conform to social norms) was dropped because it was highly skewed (present for 90% of the participants) and item 7 (lacks remorse) was eliminated because it had the lowest item-total correlation of the items and in subsequent analyses appeared to have to the lowest item validity coefficient. (Analyses including item 7 also yielded dimensional curves.) Based on the subsequent MAMBAC, MAXEIG and L-Mode analyses, the average degrees of separation for these six indicators were 1·32, 1·37 and 1·22 standard deviation units, respectively, which on average were slightly greater than the recommended minimum of 1·25 (Meehl, 1995).

None of the six MAMBAC curves (each item served as the output indicator for one graph with the remaining five items summed to create the input indicator) exhibited the single clear peak consistent with a taxonic structure. These curves yielded base rate estimates ranging from 0·52 to 0·67 (mean = 0·61, S.D. = 0·05). The

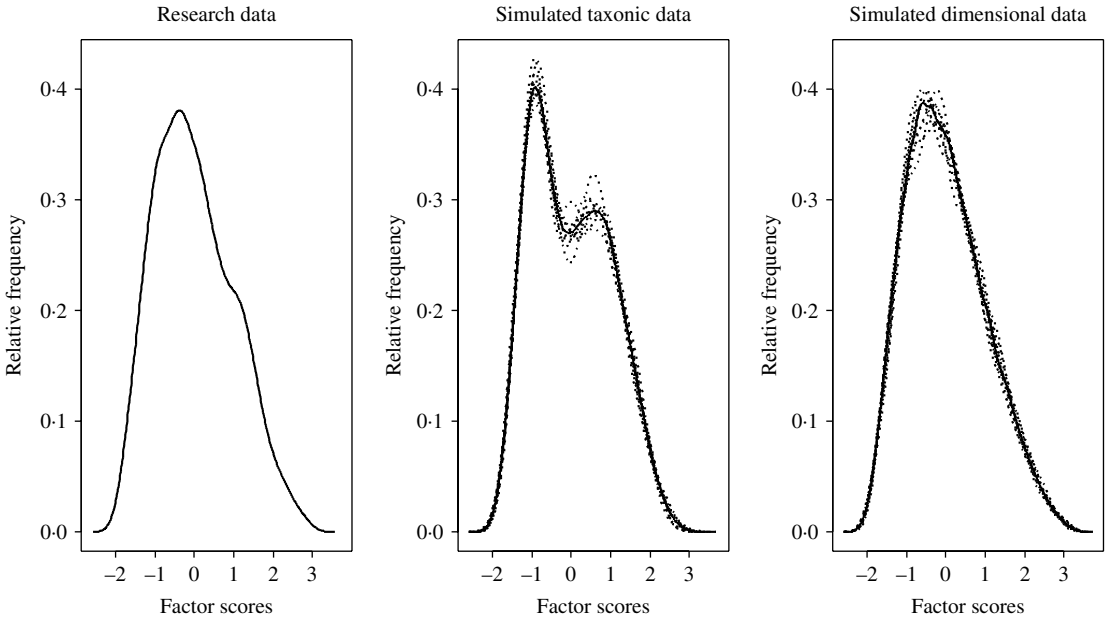

FIG. 3. Latent mode (L-Mode) factor analysis curves for the four SCID-II antisocial personality disorder (ASPD) factor scores, and for the simulated taxonic and dimensional datasets. Each graph displays the frequency distribution of scores on the first factor of a factor analysis of the indicator set. The graphs for the simulated taxonic and dimensional data were produced by generating 10 datasets for each latent structure (broken lines). The solid lines indicate the average of these datasets.

average of these six MAMBAC curves appeared more similar to the simulated dimensional data than to the simulated taxonic data; the curve fit index (0·16) was also indicative of a dimensional structure.

MAXCOV was conducted instead of MAXEIG for the six item-level indicators because each pair of items served as output indicators, with the remaining four items summed to create the input indicator, providing a sufficient range of scores on the *x* axis (in MAXEIG each input indicator would be a single item with a range of 0 to 2). The six indicators yielded 15 MAXCOV curves. Again, none of these individual curves displayed the single peak that would support a taxonic structure. These curves yielded base rate estimates that ranged from 0·05 to 0·92 (mean = 0·67, S.D. = 0·26). Although the graph of the simulated taxonic data had a clear peak, the actual data did not and the curve fit index (0·19) was more consistent with a dimensional structure.

Unlike the L-Mode curve for the simulated taxonic data, which was clearly bimodal, the L-Mode curves for the actual data and for the simulated dimensional data were unimodal. Averaging the base rate estimates from the left (0·00) and right modes (0·76) yielded a 0·38 base rate estimate, and the base rate estimate based on the classification of cases was 0·48.

Because the composite childhood conduct disorder item was the only indicator whose validity dipped below 1·25 based on case assignments from the MAMBAC (1.15) and MAXCOV (1.11) analyses, all of these taxometric analyses were repeated with only the five adult items. These analyses were also clearly dimensional. Overall, the results from the individual items were far more consistent with a dimensional structure.

PDQ-4

We also constructed four indicators using items from the self-report PDQ-4. These four indicators included the same items as the four factor scales from the SCID: Adult Antisocial ($\alpha = 0{\cdot}65$), Childhood Non-assaultive Criminal Behavior ($\alpha = 0{\cdot}73$), Physical Violence ($\alpha = 0{\cdot}78$), and Childhood Severe Antisocial Behavior ($\alpha = 0{\cdot}56$). Based on the subsequent MAMBAC, MAXEIG and L-Mode analyses, the average degrees of separation for these six

indicators were 1·62, 1·39 and 1·60 standard deviation units, respectively. There was little problem with nuisance covariance because the average correlation among these four indicators in the entire sample was 0·42, whereas the average correlation among those whose PDQ-IV met the ASPD criteria (0·13) and those whose did not meet it (0·21) were considerably lower.

All three taxometric methods yielded graphs that were highly consistent with a dimensional latent structure (i.e. the MAMBAC curves were concave, the MAXEIG curves lacked clear peaks, and the L-Mode curve was unimodal). The base rate estimates were 0·45 (S.D. = 0·08) for MAMBAC, 0·38 (S.D. = 0·18) for MAXEIG, 0·55 when averaging the base rate estimates from the left (0·09) and right modes (1·00) from L-Mode, and 0·53 from the L-Mode classification of cases. The fit index for the average MAMBAC curve was 0·09 and the fit index for the average MAXEIG curve was 0·15, both of which are far more consistent with a latent dimensional structure. These analyses were repeated twice more using indicators derived from factor analyzing the PDQ (both those from a three-factor and those from a four-factor solution). These analyses were also clearly dimensional.

DISCUSSION

Using multiple taxometric procedures with two methodologically different sets of indicators, our analyses provided no evidence that ASPD has a taxonic latent structure, at least within correctional and substance abuse populations. Pending replication in other settings (e.g. psychiatric and community populations), our findings are far more consistent with the proposition that ASPD exists on a continuum, whether assessed using a structured interview or a self-report measure.

Haslam (2003) suggested that the extant taxometric research lent support to the inclusion of ASPD, and not psychopathy, as a diagnostic *category* in DSM-IV-R, noting that 'in view of the oft-remarked failure of recent DSM editions of APD criteria to encompass psychopathy satisfactorily, it is interesting that the 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, more recent research using taxometric and latent class methods converges on a dimensional latent structure for the DSM-IV-TR diagnosis of ASPD (present study; Bucholz *et al.* 2000) and for Factor II of the PCL-R, which assesses antisocial behavior (Edens *et al.* 2006; Guay *et al.* in press). Additionally, using latent class analysis, Krueger *et al.* (2005) demonstrated that the broad domain of externalizing disorders appears to lie on a continuum. Therefore, if both ASPD and psychopathy are dimensional, neither should have priority in a categorical diagnostic taxonomy.

There has been growing interest in replacing or supplementing the current categorical approach to diagnosing personality disorders with a dimensional or prototype approach (e.g. Shedler & Westen, 2004; Clark, 2005; Widiger, 2005). The results of this study are consistent with the notion of conceptualizing ASPD as a continuous condition more akin to most forms of Type II diabetes than to dichotomous pathologies such as Type I diabetes. For such dimensional disorders, the relevant issue for researchers and practitioners is not one of establishing the ideal set of criteria for taxon membership, but rather one of determining at what point or under what circumstances deviations along this continuum merit clinical attention (Lilienfeld & Marino, 1995), or result in a 'failure to achieve adaptive solutions to universal life tasks' (Livesley & Jang, 2005, p. 265).

If ASPD proves to have a dimensional latent structure and the evidence continues to demonstrate that schizophrenia spectrum conditions (e.g. schizotypal personality disorder) are taxonic, this combination of findings suggests that revising Axis II of the DSM will not be an easy task. If the current edition is problematic because the diagnoses of personality disorders reflect arbitrary cuts along one or more continuous dimensions, replacing these categories with dimensions would obscure the existence of genuine taxa. Ultimately, an evidence-based diagnostic system for personality disorders may well need to incorporate a 'hybrid' model that accommodates both dimensions and taxa. Needless to say, considerably more research will be necessary to determine which personality disorders are categorical and which are dimensional.

These results also bolster recent attempts to conceptualize ASPD within the framework of extant dimensional models of personality, including temperament (Clark, 2005) or trait frameworks that posit three (Cloninger, 1987; Tellegen & Waller, in press) or five (Costa & Widiger, 2001) higher-order dimensions underlying both normal and abnormal personality. For example, within the five-factor model, ASPD features are associated primarily with low levels of agreeableness and conscientiousness and high levels of certain facets of neuroticism, such as hostility and impulsivity (Miller *et al*. 2003). It is unlikely that these trait models will be sufficient for describing and explaining ASPD, given that many individuals with this configuration of traits do not exhibit this condition. Presumably, these trait dispositions manifest themselves in antisocial behavior only among individuals exposed to certain environmental factors (Lykken, 1995).

A recent criticism of the taxometric literature is that the findings from different studies of the same disorder have often been inconsistent (e.g. Krueger *et al*. 2005; Widiger & Samuel, 2005), making it possible to dismiss the results of this and the Harris *et al*. study as merely another set of contradictory findings. Although the present study is not without limitations (e.g. exclusion of women), there are ample reasons to place confidence in the present findings. First, all of the published research suggesting a taxon for ASPD or psychopathy has been generated by a single research team, whereas dimensional findings have now been published by at least three independent groups of researchers. Second, by including general population inmates and individuals ordered into residential drug treatment, and excluding participants with active psychotic conditions, we minimized the risk of detecting a taxon for schizotypy. Finally, the fact that we detected consistent evidence of a dimensional structure using multiple sets of construct valid indicators for ASPD renders it unlikely that the present results can be attributed to inadequate measurement of this condition.

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DECLARATION OF INTEREST

None.

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