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TAXOMETRIC EVIDENCE FOR THE DIMENSIONAL STRUCTURE OF CLUSTER-C, PARANOID, AND BORDERLINE PERSONALITY DISORDERS

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[◂]Au: Degrees?

Despite a lively debate about the dimensional vs. categorical nature of Personality Disorders (PDs), direct empirical tests of the underlying structure are missing for most PDs. Taxometrics can be used to investigate whether latent structures are categorical or dimensional. We investigated the latent structure underlying Avoidant, Dependent, Obsessive-Compulsive, Depressive, Paranoid, and Borderline PD by means of three types of taxometric analyses. SCID-II based DSM-IV PD criterion scores from 1,816 patients from Mental Health and Forensic Institutes, and 63 nonpatients, were analyzed with three types of taxometric analyses. MAMBAC, MAXEIG, and L-MODE taxometric analyses were applied on multiple criteria sets, constituted both on theoretical grounds and randomly. Assumptions for taxometric analyses were generally met. All but two of the 78 taxometric analyses indicated greater evidence for a latent dimensional structure, with better fit of empirical data to dimensional than to taxonic simulations; mean Comparative Curve Fit Index (CCFI) = .23, SD = .09. Only two analyses yielded ambiguous evidence (CCFI in the .40–.60 range) and none indicated taxonic structure.

Following the classification of Personality Disorders (PDs) on a separate axis in the DSM-III (American Psychiatric Association, 1980), a central debate on the conceptualization of PDs has focused upon whether or not PDs are extreme positions on underlying dimensions, or qualitatively different from normal personality (Haslam, 2003; Widiger & Clark, 2000). It has been argued that categorical approaches are more suitable for clinicians

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(Haslam, 2003; Rounsaville et al., 2002; First et al., 2002; Livesley, 2003; Widiger & Clark, 2000; Widiger & Mullins-Sweatt, 2005). Categories can be used in a pragmatic sense whether the underlying structure is dimensional or not. But, the use of categorical diagnoses in the DSM has created the impression that the *underlying* structure of PDs is categorical; i.e., that there is a qualitative, and not just a quantitative difference between people with a specific PD and those without it. Categories assume the existence of boundaries, discrete or fuzzy, between normality and abnormality. This categorical thinking influences both the theoretical and the clinical views we have about PDs. For instance, the idea that people with a PD cannot profit from psychological treatments because of a fundamental flaw in their character was until recently very common. Moreover, whether we view PDs as essentially dimensional or categorical, influences our research methodologies and how we interpret empirical findings. For example, if the underlying construct is dimensional, diagnostic thresholds are essentially arbitrary. If so, a small change can have a great impact on prevalence estimates. Moreover, according to some authors, defining PDs as categories with arbitrary boundaries leads to the idea that the large numbers of comorbid diagnoses indicate that patients have many discrete psychiatric diseases, whereas a dimensional understanding of psychopathology would suggest that patients differ in their profiles (Haslam, 2003; Widiger & Samuel, 2005).

Conceptual problems, as well as practical and ideological arguments have dominated the debate. There has been a lack of direct empirical tests comparing categorical and dimensional models, although results from factor-analytic, longitudinal, and validity studies have been interpreted as generally more supportive of dimensional than of categorical conceptualizations (Blackburn, Logan, Renwick, & Donnelly, 2005; Durbin & Klein, 2006; Hong et al., 2005; Pukrop & Krischer, 2005; Saulsman & Page, 2004; Skodol et al., 2005; Ullrich, Borkenan, & Morneros, 2001). The structure of PDs has therefore been considered as perhaps the most crucial unresolved issue in the study of PDs (Endler & Kocovski, 2002). Until recently statistical procedures for such comparisons, known as taxometrics, were difficult to apply and had to rely on subjective judgements about the shape of curves (Waller & Meehl, 1998). Recent advancements have solved these problems (Ruscio, Ruscio, & Meron, 2007). In brief, taxometrics refers to a group of statistical procedures developed to test whether empirical data indicate that a construct should be viewed as a dimension or as a taxon (i.e., a latent class differing in kind from its complement class). A number of procedures is available to check datasets for the distribution of latent constructs underlying observed manifestations on characteristics that indicate dimensionality vs. taxonicity. By simulation methods and application of fit indices, objective tests of taxonicity versus dimensionality are now available (Ruscio et al., 2007). In general it is recommended to use several methods to check for consistency. If all methods yield the same outcome, the evidence is much stronger than the

outcome of one method, because different methods are based on different assumptions and procedures.

Previous studies found evidence for dimensionality in Borderline PD (Ayers, 2000; Rothschild, Cleland, Haslam, & Zimmerman, 2003;[1] Simpson, 1994; Trull, Widiger, & Guthrie, 1990). Meehl (2001) commented that the evidence in the Trull et al. study was not unequivocal as the lack of a central peak might have been caused by low base rate of Borderline PD and inchworm tests lacked. Evidence for taxonicity of schizotypy and schizotypal PD was found in many studies (Haslam, 2003; Blanchard, Gangestad, Brown, & Horan, 2000; Horan, Blanchard, Gangestad, & Kwapil, 2004; Korfine & Lenzenweger, 1995; Lenzenweger, 1999; Lenzenweger & Korfine, 1992; Tyrka et al. 1995). Evidence for taxonicity of Antisocial PD and psychopathy was mixed, with four studies reporting taxonic structure (Ayers, 2000; Harris, Rice, & Quinsey, 1994; Skilling, Harris, Rice, & Quinsey, 2002; Vasey, Kotov, Frick, & Loney, 2005; Edens, Marcus, Lilienfeld, & Poythress, 2006) and three studies dimensional structure (Edens et al., 2006; Marcus, John, & Edens, 2004; Marcus, Lilienfeld, Edens, & Poythress, 2006). Most PDs have not been investigated at all in this respect. Given the discussion whether the PD construct in the DSM-V should remain categorical or become dimensional, (Rounsaville et al., 2002; First et al., 2002; Widiger & Simonsen, 2005), there is an urgent need to investigate the latent structure of most PDs.

The aim of the present study was to investigate the underlying structure of six common PDs in a large mixed sample of nonpatients, and patients with and without PDs who were assessed with the SCID-II for DSM-IV (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The PDs investigated by us were Avoidant, Dependent, Obsessive-compulsive, Depressive, Paranoid, and Borderline PD. These PDs were sufficiently common in our sample to allow using taxometric procedures.

METHOD

PARTICIPANTS

Data were available for 1,879 people (1,245 women). Sixty-three were nonpatients, who participated in various studies. Of the 1,816 patients, 1,803 sought treatment at mental health centers in the Netherlands (RIAGG-Maastricht; Psychiatric Hospital Vijverdal, & Dr. Poelsoord, Maastricht; GGzE, Eindhoven; Psychiatric Hospital Valerius, Amsterdam; Parnassia, The Hague; Vincent van Gogh Institute, Venray) and Belgium (CGG-Hasselt, CGG-Munster, Medical Centre St. Jozef Kortenhoef, Psychiatric Hospital Rekem, Psychiatric Hospital Ziekeren). Thirteen patients were incar-

1. A review by Haslam (2003) concluded that the Rothschild et al. (2003) data supported taxonicity of BPD. This was based on preliminary analyses. The final analyses supported a dimensional structure, as published by Rothschild et al. (2003).

cerated in prisons or forensic hospitals in Belgium (Antwerpen, Brugge, Gent, Rekem). The mean age was 33.03 (SD 10.75). Based on SCID-II scores, there were 400 Avoidant, 74 Dependent, 222 Obsessive-Compulsive, 40 Passive-Aggressive, 175 Depressive, 95 Paranoid, 11 Schizotypal, 11 Schizoid, 7 Histrionic, 7 Narcissistic, 169 Borderline, and 33 Anti-Social PDs diagnosed in the sample; 1,142 (60.8%) of the sample did not meet a PD diagnosis. None of the nonpatients received a PD diagnosis. Data was collected as part of standard assessment or as part of specific research projects. In the latter case, participants gave written consent after full explanation of the study.

MATERIALS

We used the Dutch version of the Structured Clinical Interview for DSM-IV Axis-II Personality Disorders (SCID-II; First et al., 1997; Weertman, Arntz, & Kerkhofs, 2000). For research purposes, cut-off points were not used and all criteria of all PDs were assessed. The very few "inadequate information" (?) ratings were recoded to "absent or false" (1) ratings. Axis-2 criteria were assessed by 46 interviewers, trained by our research group. In a test-retest study with two different interviewers we found adequate ICC's (md = .76) for PD trait scores (Weertman, Arntz, Dreessen, Van Velzen, & Vertommen, 2003). In a second study 151 taped SCID-II interviews were rated twice by independent raters from our pool of raters. ICC's were excellent for sumscores of the six pertinent PDs: Avoidant PD .90, Dependent PD .92, Obsessive-Compulsive PD .89, Depressive PD .95, Paranoid PD .85, Borderline PD .95 (median .91, range .85&ndash.95; Lobbestael, Arntz, & Bernstein, 2008). Of five of these PDs cell sizes allowed to estimate Cohen's Kappa for diagnostic interrater agreement: Avoidant PD .83, Dependent PD .83, Obsessive-Compulsive PD .87, Depressive PD .94, Borderline PD .91. Cronbach alpha's of the PD sumscores in the present study indicate sufficient homogeneity of the scales, Avoidant .82, Dependent .70, Obsessive-Compulsive .70, Depressive .77, Paranoid .71, Borderline .87, with all criteria having adequate item-rest correlations. We checked the factorial validity by confirmatory factor analysis using structural equation modeling (Jöreskog & Sörbom, 2002). The fit of the criteria to the 6 pertinent PDs was good, Comparative Fit Index = .94, Standardized Root Mean Square Residual = .053. Correlations corrected for attenuation between latent factors varied from .22 to .71, between raw sumscores from .21 to .55. PD sumscores were therefore sufficiently independent.

STATISTICAL ANALYSES

The following three taxometric methods were used, so that consistency over methods could be checked.

First, Mean Above Minus Below A Cut (MAMBAC; Meehl & Yonce, 1994) is a procedure that is based on the idea that if taxa exist, there must be

an optimal cutting score on a quantitative property of these taxa. This cutting score minimizes the number of false-positive and false-negative classifications. In absence of taxonic structure, an optimal cutting score does not exist. The procedure uses one input indicator of the construct, and one output indicator. The mean difference on the output indicator between subsamples left and right to scores of the input indicator is plotted as function of the latter. If the data are taxonic a peaked curve is expected, whereas no peak is expected for dimensional data. We performed MAMBAC using 50 equally-spaced cuts located 25 cases from each end of the input indicator; variables served in all pairwise input/output indicator combinations.

Second, MAXimum EIGenvalue (MAXEIG; Waller & Meehl, 1998) examines inter-indicator association within ordered subsamples of cases to test whether the indicators covary due to a mixture of latent taxa or due to the indicators' shared loadings on a latent dimension. Within a subsample that contains a relatively pure group of either taxon or complement members, there is little association between indicators. In contrast, associations among indicators is high in subsamples that contain a mixture of groups because taxon members tend to score high and complement members score low on the indicators. MAXEIG uses an input indicator of the construct, and two or more output indicators. The approach is multivariate: the largest eigenvalue of the covariances of the output indicators is used as association parameter. As for MAMBAC, taxonic data are expected to yield a peaked curve and dimensional data are not. We performed MAXEIG using 50 windows that overlapped 90% with one another; each variable served once as input indicator, with all other variables serving as output indicator.

Third, Latent Mode (L-MODE; Waller & Meehl, 1998) is based on factor analysis. By plotting the distribution of individuals' scores on a single latent factor calculated through factor analysis L-MODE seeks to differentiate taxonic and dimensional latent structure. Taxonic data are expected to yield bimodal score distributions, whereas dimensional data are expected to yield unimodal score distributions.

Calculations were done in R (R Foundation for Statistical Computing, Vienna, Austria; http://www.r-project.org/) with Ruscio's taxometric programs (http://www.taxometricmethod.com/). To interpret results, 10 samples of dimensional and 10 samples of taxonic comparison data were generated using an approach grounded in the bootstrap; for the latter, cases were assigned to groups using a base-rate classification method (Ruscio, in press◂; Ruscio et al., 2007). For these procedures indicator sets have to be constructed. For MAMBAC the minimum number of indicators is two, for MAXEIG it is three, L-MODE needs multiple indicators. Ruscio, Haslam, & Ruscio (2006) suggest constructing criterion sets based on empirical, theoretical, or content-related arguments. For each PD we therefore constituted two sets of three or four indicators each by combining criteria on the basis of the criteria's content. Themes of the theoretical indicator

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sets are described in the Appendix. The two theoretical sets for Paranoid PD were based on Bernstein and Useda (2006). For Borderline PD, empirically derived factor models constituted the basis for the sets (Sanislow et al., 2002; Clarkin, Hull, & Hurt, 1993; Hurt et al., 1990; Morey, 1991; Livesley & Schröder, 1991; Zanarini, Gunderson, Frankenburg, & Chauncey, 1989). We also constructed three sets per PD by randomly assigning the criteria to indicators. The minimum number of variables per indicator was set at 2. The 1–2-3 SCID-II criterion ratings were used to approach continuity better than with dichotomous scores. Table 1 presents the indicator sets. For MAMBAC, each indicator served as output variable, with the sum of the other two as input variable. For MAMBAC and MAXEIG, we also used each of the PD criteria (for MAXEIG, all pairs of criteria) as output indicator(s), with the remaining criteria scores summed as input indicator, to get a more continuous input indicator. For MAMBAC and MAXEIG, all

TABLE 1. Overview of the Indicator Sets

	Indicator 1	Indicator 2	Indicator 3	Indicator 4[1]
Avoidant PD				
Theoretical 1	1, 2	3, 6	4, 5, 7	
Theoretical 2	2, 7	1, 3	4, 5, 6	
Random 1	6, 3	7, 4	1, 2, 5	
Random 2	2, 4	1, 3	5, 6, 7	
Random 3	2, 6	5, 7	1, 3, 4	
Dependent PD				
Theoretical 1	1, 2	3, 5	4, 6	7, 8
Theoretical 2	1, 2, 4	3, 5	6, 7, 8	
Random 1	1, 7	2, 8, 6	3, 4, 5	
Random 2	3, 4	1, 7, 8	2, 5, 6	
Random 3	2, 5, 6	1, 3, 8	4, 7	
Obsessive-Compulsive PD				
Theoretical 1	1, 2	3, 5	4, 6, 7, 8	
Theoretical 2	1, 6	3, 5, 7	2, 4, 8	
Random 1	1, 6, 3	7, 2, 4	5, 8	
Random 2	4, 8	6, 7, 2	1, 3, 5	
Random 3	1, 6, 2	3, 8, 7	4, 5	
Depressive PD				
Theoretical 1	2, 7	3, 5	1, 4, 6	
Theoretical 2	1, 2, 7	3, 5	4, 6	
Random 1	4, 5	2, 7	1, 3, 6	
Random 2	2, 4	1, 6	3, 5, 7	
Random 3	6, 7	2, 5	1, 3, 4	
Paranoid PD				
Theoretical 1	1, 2	3, 4, 7	5, 6	
Theoretical 2	2, 7	1, 3	4, 5, 6	
Random 1	2, 7	1, 5, 3	4, 6	
Random 2	4, 3, 7	6, 2	1, 5	
Random 3	5, 3	2, 4, 7	6, 1	
Borderline PD				
Theoretical 1	2, 3, 7, 9	4, 5	1, 6, 8	
Theoretical 2	1, 2, 3	4, 5, 8	6, 7, 9	
Random 1	1, 3, 9	5, 6, 8	2, 4, 7	
Random 2	3, 4, 8	1, 2, 9	5, 6, 7	
Random 3	1, 6, 9	2, 3, 5	4, 7, 8	

Note. Numbers indicate the DSM-IV number of the specific PD criterion.
[1]Only for Dependent PD one of the sets consisted of four indicators because of theoretical (content) reasons.

permutations were analyzed. Ruscio's programs yield aggregated output of the results of all permutations. For L-MODE, computations were done on the separate DSM-IV PD criterion scores.

The number of 10 bootstraps seems small, but a recent study (Ruscio & Kaczetow, under review) indicates that this suffices. In the first place, it should be noted that the number of bootstrap samples (B) needed, depends on the nature of the application. For example, to construct a *CI*, it's not unusual to use $B = 1{,}000$ or more so that the tails of an empirical sampling distribution can be defined well. On the other hand, to estimate the *SE* of a statistic, it's not unusual to use $B = 25$ to 200. To the extent that the statistic in question is fairly stable from sample to sample, for example if N is large, low values of B suffice to estimate its variability. In the context of taxometrics, multiple bootstrap samples are used only to calculate a series of M values that constitute an averaged curve. Also, each of the B curves is itself an averaged curve across a series of analyses generated by using indicators in all possible configurations. In sum, because taxometric studies usually have fairly large N (and the present study has certainly a large N) and what's being obtained is an average of already averaged curves, it should not be too surprising that a small value of B suffices.

Secondly, the results from the Ruscio and Kaczetow (under review) study for different levels of B underscores the fact that it makes little difference how large B is. Across all 25,000 target data sets in the simulation study, an increase in B from 10 to 25 or 50 did not yield a statistically significant increase in the accuracy of the CCFI as calculated for MAXEIG analyses: At $B = 10$, the CCFI threshold of .50 correctly identifed taxonic vs. dimensional structure 92.4% of the time, and at $B = 50$ accuracy was 93.0%.

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Third, if one uses too small a value of B in any bootstrap application, the results would be affected to a greater extent by sampling error. This would not introduce a systematic bias, but it would make it more difficult to obtain consistent results across any series of analyses. Thus, if the present results turn out to be consistent, it indicates that B could not have been problematically low.

The following checks were done to assess whether the data were adequate for taxometric analyses (Meehl, 1995; Ruscio et al., 2006). (1) The putative taxon sample sizes should be large enough. In the case of $N = 300$ (the suggested minimum for taxometrics) it is recommended to have at least 10% ($n = 30$) of the sample to be putative taxon members. For larger N's not the relative but the absolute sample size is important (Ruscio et al. 2006). We restricted our analyses to PDs with $>n \geq 70$; (2) The (nuisance) correlations between indicators within putative taxon and complement groups should be lower than .30. As can be seen in Table 2, this requirement was met in most sets. Only 14 (9%) of the 156 correlations were >.30, most of them very near .30; (3) The indicator validities should be large enough, i.e. Cohen's $d > 1.25$ when putative taxon and complement groups

are compared on the indicator. Table 2 shows this is the case for 76 (97.4%) of the 78 indicator sets. The two lower *d*'s (1.11, 1.17) were very near 1.25; (4) The sample should not be specifically selected on the putative taxon membership, as this may lead to pseudotaxonicity. E.g., a mixed sample of BPD patients and controls not meeting any BPD criterion might yield invalid conclusions, as bimodality is built in. We used an unselected patient sample, with 63 nonpatients added, in which distributions of number of PD criteria were not affected by biased sampling. Inspection of criterion sumscores frequency plots did not indicate obvious bimodality; (5) The indicators should be quasi-continuous. We analysed composite scores of PD criteria (MAMBAC and MAXEIG); (6) The choice of indicators should be valid. By using SCID-II PD criteria scores we stayed as close as possible to the constructs as defined by the DSM-IV. Semi-structured clinical interviews like the SCID-II are more valid than questionnaires or clinical impressions; (7) Indicators should be nonoverlapping. Because we used DSM-IV criteria, content overlap between the observed variables was minimal; in constructing the theory-based indicator sets, we attempted to avoid higher order content overlap between the sets; (8) Simulations should demonstrate that taxonic and dimensional latent constructs can be distinguished given the characteristics of each data set. All parallel analyses of taxonic and dimensional comparison data yielded distinguishable results.

Results were evaluated as follows. First, by visual inspection of plots yielded by the original data, and visual comparison of them with simulated taxonic and dimensional plots. Parallel analyses of comparison data can be informative because the expected curve shapes for taxonic and dimensional data are ideals that can be influenced by many data characteristics. Second, results were evaluated by checking consistency of base rate estimates within and across methods. Lack of consistency may indicate dimensional structure, and high consistency may indicate taxonic structure (Waller & Meehl, 1998; Ruscio et al., 2006; see Ruscio, 2007, for empirical study of this consistency test). Third, results were evaluated by calculating the Comparison Curve Fit Index (CCFI), an objective measure that quantifies the relative similarity of results for the empirical data to those of taxonic and dimensional comparison data (Ruscio et al., 2007). The CCFI gives an indication to what degree the observed distribution fits more with dimensional simulation (<.5), with CCFI = 0 as perfect dimensional fit, or more with taxonic simulation (>.5), with CCFI = 1 as perfect taxonic fit. CCFI = .50 denotes equivalent evidence. In a Monte Carlo study, Ruscio and Kaczetow (under review) found evidence for high accuracy (larger than .95 in most of the data conditions they investigated) of the CCFI when outside the .40–.60 region. Thus, in the present study we interpreted a CCFI <.40 as indicating dimensionality; a CCFI >.60 as taxonicity; and a CCFI in the .40–.60 range as ambiguous. Several rigorous studies show that the use of comparison data, when quantified using the CCFI, identify taxonic and dimensional data with an impressive degree of validity that

TABLE 2. Author: Please Provide Table Title

	Indicator Validity (*d*)		Indicator Correlations within:				Base Rates of:			Fit of Data with Simulation of:		
			Taxon Group		Complement Group		Taxon Group		Averaged Curve	Taxon	Dimension	Comparison
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*RMSR*	*RMSR*	*CCFI*
Avoidant PD												
MAMBAC												
Model 1 (th1)	2.41	0.43	–0.11	0.22	0.39	0.14	0.11	0.14	0.05	0.19	0.03	0.15
Model 2 (th2)	2.40	0.27	0.06	0.04	0.08	0.11	0.32	0.03	0.31	0.20	0.03	0.12
Model 3 (r1)	2.40	0.17	–0.12	0.12	0.34	0.11	0.15	0.13	0.15	0.15	0.03	0.17
Model 4 (r2)	2.45	0.25	0.04	0.05	0.11	0.11	0.30	0.06	0.29	0.43	0.04	0.08
Model 5 (r3)	2.30	0.15	0.13	0.05	0.04	0.06	0.39	0.09	0.38	0.41	0.05	0.11
Summed criteria	1.67	0.19	0.02	0.09	0.06	0.07	0.30	0.08	0.29	0.26	0.03	0.11
MAXEIG												
Model 1 (th1)	2.18	1.30	0.36	0.11	0.12	0.20	0.23	0.11	0.21	0.11	0.02	0.15
Model 2 (th2)	2.37	0.40	0.11	0.04	0.14	0.22	0.24	0.09	0.23	0.12	0.03	0.20
Model 3 (r1)	2.24	0.08	0.15	0.14	0.13	0.21	0.26	0.07	0.24	0.12	0.02	0.16
Model 4 (r2)	2.40	0.13	0.03	0.02	0.21	0.14	0.24	0.02	0.24	0.10	0.03	0.20
Model 5 (r3)	2.34	0.30	0.14	0.05	0.13	0.12	0.26	0.05	0.25	0.10	0.03	0.25
Summed criteria	1.71	0.19	0.01	0.09	0.07	0.08	0.27	0.12	0.23	0.07	0.03	0.27
L-MODE												
All criteria	1.59	0.24	0.06	0.08	0.02	0.05	0.43	NA	NA	NA	NA	NA
Dependent PD												
MAMBAC												
Model 1 (th1)	1.84	0.15	–0.04	0.21	0.03	0.05	0.22	0.05	0.20	0.27	0.05	0.15
Model 2 (th2)	1.97	0.24	–0.16	0.16	0.01	0.04	0.22	0.16	0.21	0.44	0.04	0.08
Model 3 (r1)	2.31	0.48	–0.04	0.11	0.07	0.05	0.19	0.01	0.19	0.32	0.04	0.10
Model 4 (r2)	2.57	0.36	–0.13	0.13	0.20	0.07	0.10	0.09	0.03	0.19	0.06	0.23
Model 5 (r3)	2.23	0.09	0.03	0.05	0.07	0.04	0.22	0.03	0.22	0.34	0.02	0.06
Summed criteria	1.45	0.31	–0.01	0.13	0.04	0.05	0.18	0.03	0.18	0.23	0.07	0.24
MAXEIG												
Model 1 (th1)	2.27	0.53	–0.09	0.20	0.13	0.06	0.10	0.04	0.08	0.10	0.04	0.30
Model 2 (th2)	1.85	1.82	0.12	0.31	0.14	0.07	0.17	0.01	0.16	0.10	0.05	0.35
Model 3 (r1)	2.00	0.83	0.01	0.52	0.27	0.01	0.13	0.03	0.12	0.12	0.06	0.33
Model 4 (r2)	2.01	1.73	0.31	0.07	0.21	0.02	0.15	0.04	0.13	0.14	0.04	0.24
Model 5 (r3)	2.16	0.86	0.10	0.25	0.27	0.08	0.12	0.02	0.12	0.12	0.04	0.26
Summed criteria	1.48	0.30	–0.02	0.14	0.04	0.06	0.17	0.20	0.08	0.08	0.02	0.20
L-MODE												
All criteria	1.17	0.15	0.02	0.12	–0.02	0.02	0.50	NA	NA	NA	NA	NA

Obsessive-Compulsive PD												
MAMBAC												
Model 1 (th1)	2.21	0.04	–0.12	0.16	0.19	0.13	0.17	0.16	0.11	0.32	0.06	0.16
Model 2 (th2)	2.23	0.03	–0.16	0.16	0.22	0.12	0.15	0.14	0.11	0.34	0.07	0.16
Model 3 (r1)	2.04	0.33	–0.06	0.13	0.05	0.13	0.25	0.02	0.25	0.19	0.05	0.20
Model 4 (r2)	1.86	0.27	–0.04	0.14	–0.001	0.09	0.33	0.02	0.33	0.22	0.04	0.15
Model 5 (r3)	1.97	0.22	–0.10	0.08	0.04	0.09	0.25	0.07	0.24	0.26	0.03	0.10
Summed criteria	1.11	0.36	0.02	0.09	–0.01	0.04	0.41	0.38	0.00	0.17	0.06	0.26
MAXEIG												
Model 1 (th1)	1.90	0.19	–0.003	0.03	0.05	0.10	0.28	0.04	0.29	0.09	0.04	0.31
Model 2 (th2)	1.82	0.16	0.12	0.14	0.10	0.23	0.33	0.11	0.37	0.08	0.04	0.35
Model 3 (r1)	2.10	0.13	–0.08	0.08	0.18	0.13	0.19	0.08	0.19	0.07	0.02	0.23
Model 4 (r2)	1.99	1.33	0.03	0.13	0.23	0.12	0.20	0.10	0.19	0.11	0.04	0.28
Model 5 (r3)	1.90	0.85	–0.08	0.33	0.24	0.10	0.21	0.10	0.20	0.09	0.03	0.24
Summed criteria	1.28	0.33	–0.03	0.10	0.04	0.07	0.23	0.16	0.16	0.07	0.02	0.20
L-MODE												
All criteria	1.31	0.52	0.03	0.07	–0.002	0.06	0.499	NA	NA	NA	NA	NA
Depressive PD												
MAMBAC												
Model 1 (th1)	2.17	0.12	–0.08	0.05	0.07	0.08	0.30	0.02	0.30	0.25	0.06	0.19
Model 2 (th2)	2.12	0.14	–0.08	0.07	0.06	0.09	0.31	0.05	0.32	0.18	0.06	0.25
Model 3 (r1)	2.24	0.31	–0.08	0.13	0.18	0.14	0.23	0.04	0.23	0.22	0.04	0.17
Model 4 (r2)	2.24	0.09	0.01	0.11	0.06	0.06	0.32	0.07	0.32	0.16	0.03	0.17
Model 5 (r3)	2.32	0.18	–0.19	0.36	0.39	0.13	0.08	0.14	0.00	0.21	0.04	0.17
Summed criteria	1.52	0.29	0.31	0.10	–0.03	0.10	0.24	0.11	0.25	0.13	0.04	0.24
MAXEIG												
Model 1 (th1)	2.06	1.50	0.18	0.17	0.23	0.10	0.25	0.06	0.26	0.12	0.04	0.25
Model 2 (th2)	1.95	0.39	0.05	0.14	0.11	0.14	0.32	0.11	0.30	0.12	0.04	0.27
Model 3 (r1)	1.95	1.51	0.25	0.22	0.31	0.19	0.22	0.08	0.23	0.13	0.03	0.21
Model 4 (r2)	2.11	0.17	0.04	0.27	0.25	0.10	0.24	0.11	0.20	0.12	0.02	0.12
Model 5 (r3)	2.16	1.15	0.19	0.04	0.25	0.13	0.25	0.06	0.24	0.09	0.02	0.21
Summed criteria	1.48	0.23	–0.02	0.12	0.05	0.11	0.28	0.15	0.33	0.06	0.04	0.37
L-MODE												
All criteria	1.33	0.38	0.05	0.12	–0.01	0.04	0.499	NA	NA	NA	NA	NA
Paranoid PD												
MAMBAC												
Model 1 (th1)	2.30	0.63	–0.03	0.25	0.05	0.08	0.19	0.05	0.18	0.39	0.05	0.12
Model 2 (th2)	1.47	0.42	0.13	0.12	–0.06	0.02	0.44	0.49	0.00	0.31	0.07	0.18
Model 3 (r1)	2.27	0.34	–0.08	0.29	0.06	0.12	0.17	0.05	0.17	0.36	0.06	0.15
Model 4 (r2)	2.55	0.38	–0.04	0.14	0.10	0.10	0.14	0.04	0.14	0.18	0.05	0.21
Model 5 (r3)	1.60	0.39	0.03	0.24	–0.06	0.01	0.38	0.55	0.00	0.38	0.08	0.17
Summed criteria	1.94	0.77	–0.03	0.11	0.08	0.10	0.09	0.09	0.00	0.16	0.05	0.25

(*continued*)

TABLE 2. Continued

	Indicator Validity (*d*)		Indicator Correlations within: Taxon Group		Indicator Correlations within: Complement Group		Base Rates of: Taxon Group		Base Rates of: Averaged Curve	Fit of Data with Simulation of: Taxon	Fit of Data with Simulation of: Dimension	Fit of Data with Simulation of: Comparison
	M	***SD***	***M***	***SD***	***M***	***SD***	***M***	***SD***	***M***	***RMSR***	***RMSR***	***CCFI***
MAXEIG												
Model 1 (th1)	2.39	1.08	0.07	0.18	0.17	0.20	0.11	0.04	0.11	0.09	0.04	0.31
Model 2 (th2)	2.47	1.41	0.01	0.41	0.16	0.09	0.09	0.02	0.09	0.07	0.05	0.43
Model 3 (r1)	2.08	1.64	0.19	0.06	0.18	0.03	0.11	0.04	0.12	0.11	0.04	0.29
Model 4 (r2)	2.44	0.76	0.05	0.18	0.19	0.15	0.10	0.03	0.10	0.13	0.05	0.29
Model 5 (r3)	2.27	1.29	0.05	0.24	0.14	0.14	0.09	0.03	0.09	0.09	0.04	0.31
Summed criteria	1.78	0.64	–0.03	0.13	0.05	0.09	0.12	0.11	0.08	0.07	0.02	0.22
L-MODE												
All criteria	1.32	0.50	0.04	0.11	–0.02	0.03	0.498	NA	NA	NA	NA	NA
Borderline PD												
MAMBAC												
Model 1 (th1)	3.24	0.12	0.14	0.14	0.22	0.07	0.18	0.02	0.18	0.21	0.03	0.14
Model 2 (th2)	3.08	0.10	0.18	0.07	0.13	0.04	0.20	0.02	0.20	0.12	0.08	0.396
Model 3 (r1)	3.52	0.26	0.02	0.12	0.41	0.05	0.11	0.09	0.11	0.13	0.05	0.29
Model 4 (r2)	3.25	0.23	0.16	0.06	0.18	0.03	0.18	0.02	0.18	0.12	0.04	0.24
Model 5 (r3)	3.60	0.81	–0.03	0.09	0.53	0.03	0.07	0.12	0.00	0.14	0.06	0.31
Summed criteria	2.08	0.33	0.07	0.09	0.09	0.06	0.18	0.06	0.18	0.10	0.04	0.27
MAXEIG												
Model 1 (th1)	3.26	0.19	0.13	0.06	0.36	0.07	0.11	0.01	0.11	0.09	0.04	0.29
Model 2 (th2)	3.11	1.41	0.25	0.19	0.36	0.02	0.10	0.01	0.10	0.07	0.04	0.36
Model 3 (r1)	3.36	0.91	0.16	0.11	0.43	0.05	0.11	0.01	0.11	0.08	0.04	0.31
Model 4 (r2)	3.31	0.07	0.08	0.07	0.36	0.01	0.10	0.01	0.10	0.08	0.05	0.396
Model 5 (r3)	3.28	0.91	0.26	0.22	0.42	0.05	0.10	0.02	0.10	0.05	0.05	0.50
Summed criteria	2.38	0.44	0.01	0.09	0.20	0.07	0.10	0.05	0.08	0.07	0.02	0.22
L-MODE												
All criteria	1.67	0.26	0.15	0.09	0.01	0.03	0.499	NA	NA	NA	NA	NA

Note. Results aggregated for all permutations (MAMBAC, MAXEIG). For each PD five models with 3 or 4 indicators consisting of summed PD criterion scores were run with MAMBAC and MAXEIG procedures. The first two models (th1, th2) were based on content considerations; the other three were based on randomly derived criteria groupings (r1-r3). MAMBAC and MAXEIG were also run with each (pair) of the criteria as output indicator(s), and the other criterion scores summed as input indicator ("Summed criteria"). The L-MODE procedure was run on all available criteria of each PD. If applicable, weighted fit indices are reported.

Abbreviations: PD, personality disorder; NA, not applicable; RMSR, root mean square residual; CCFI, comparative curve fit index.

surpasses that of several commonly used taxometric consistency tests (Ruscio, 2007; Ruscio & Marcus, 2007; Ruscio et al., 2007). Because there may be small taxa among the PDs, it is important to note that each study of the CCFI showed that it performed at least as well with the lowest taxon base rates studied as with higher values. L-MODE output was only evaluated qualitatively by inspecting curves and comparing them to simulated curves, and interpreting base rate estimates, as no reliable fit indices have been identified so far for L-MODE. When maxima could be located at either end of the MAXEIG graphs, but were not visually detectable, we executed inchworm consistency tests (Waller & Meehl, 1998).

The L-Mode procedure provides several estimates of the taxon base rate (Waller & Meehl, 1998). For the present study one estimate was derived following the guidelines of Waller and Meehl (1998). When the two base rate estimates derived from the location of the two modes in the distribution of estimated factor scores were close to .50, their average was used. When the base rate was less than .50, the estimate from the upper mode was used; when the base rate was greater than .50, the estimate from the lower mode was used.

RESULTS

The results are presented in Table 2. For each PD, MAMBAC, and MAXEIG summary graphs of all first theoretical sets and L-Mode summary graphs of all criteria are presented in Figures 1 and 2.[2] Observed patterns can be compared to taxonic and dimensional simulations.

Avoidant PD. Visual inspection of the graphs favored dimensional structure. Inchworm tests gave no evidence for a hidden taxonic structure. Figure 1, left column, shows graphs of MAMBAC and MAXEIG analyses with taxonic and dimensional simulations of the first theoretical set, and L-Mode graphs of all criteria. MAMBAC analyses yielded divergent mean taxon base rate estimates, range .11–.39 (Table 1). The base rate estimates from the MAXEIG procedure were more consistent across the different sets (mean .25, range .23–.27), overlapping with the base mean base rate estimate of all MAMBAC analyses (.26). Note that DSM-IV defined avoidant PD was present in 21.3% of the cases. L-MODE yielded a much higher base rate estimate however (.43). RMSRs and CCFIs (range .08–.27) pointed unequivocally to dimensional structure. None of the indices favored a taxonic structure.

Dependent PD. The graphs suggested dimensional structure. Inchworm tests gave no evidence for hidden taxonicity. Figure 1, mid column, shows graphs of MAMBAC and MAXEIG analyses with taxonic and dimensional simulations of the first theoretical set, and L-Mode graphs of all criteria. MAMBAC analyses yielded somewhat divergent mean taxon base rate esti-

2. All graphs can be inspected at http://www.epp.unimaas.nl

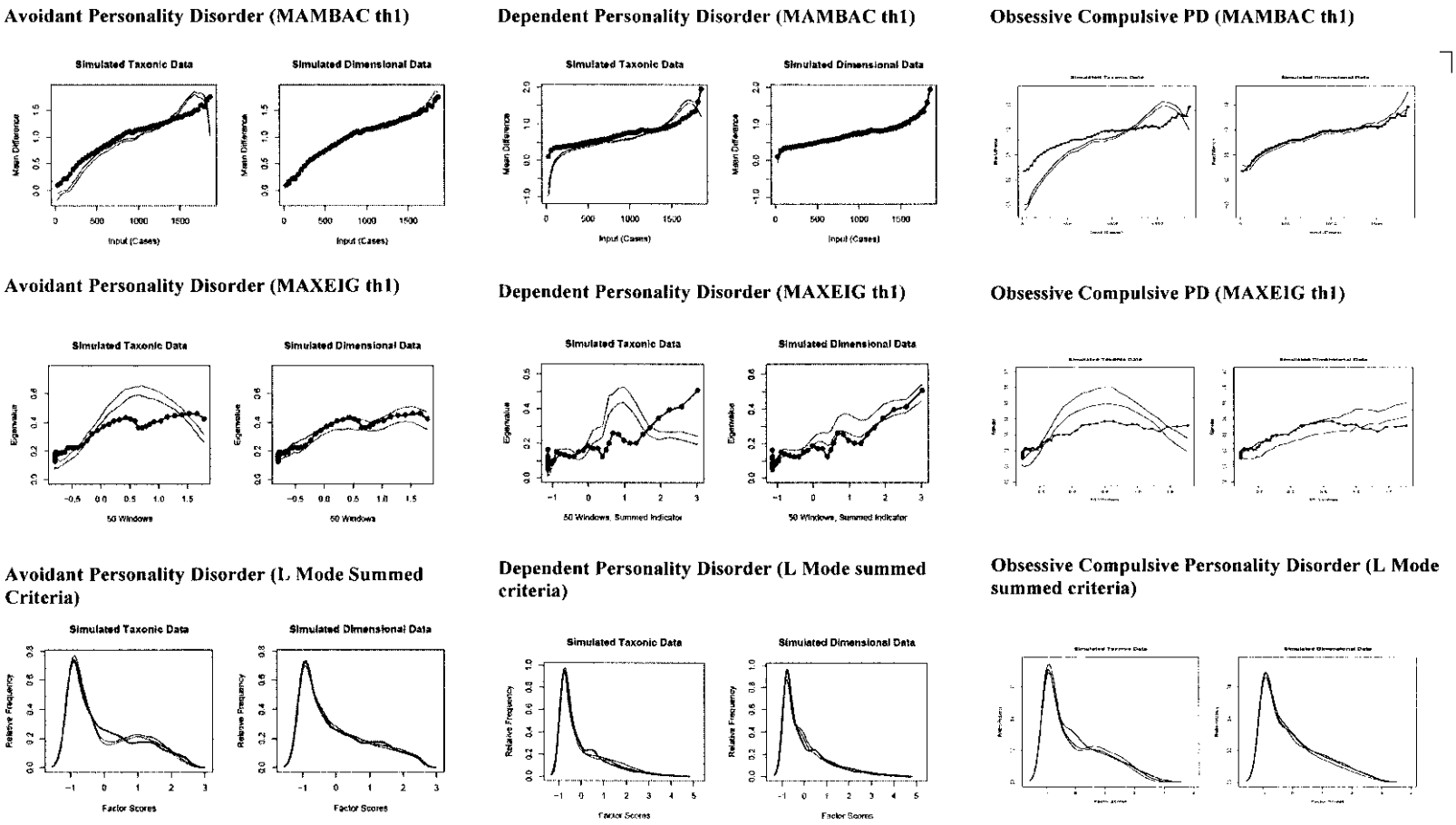

FIGURE 1. Observed (bold) and simulated curve ranges of MAMBAC and MAXEIG procedures for the first theoretical indicator sets and of L-Mode for all criteria of Avoidant PD (left column), Dependent PD (middle column) and Obsessive-Compulsive PD (right column). Observed curves are averaged across results of all permutations of indicator sets (MAMBAC, MAXEIG). For both taxonic and dimensional structures, 10 simulations were done. Observed curves resemble simulated dimensional curves better than simulated taxonic curves.

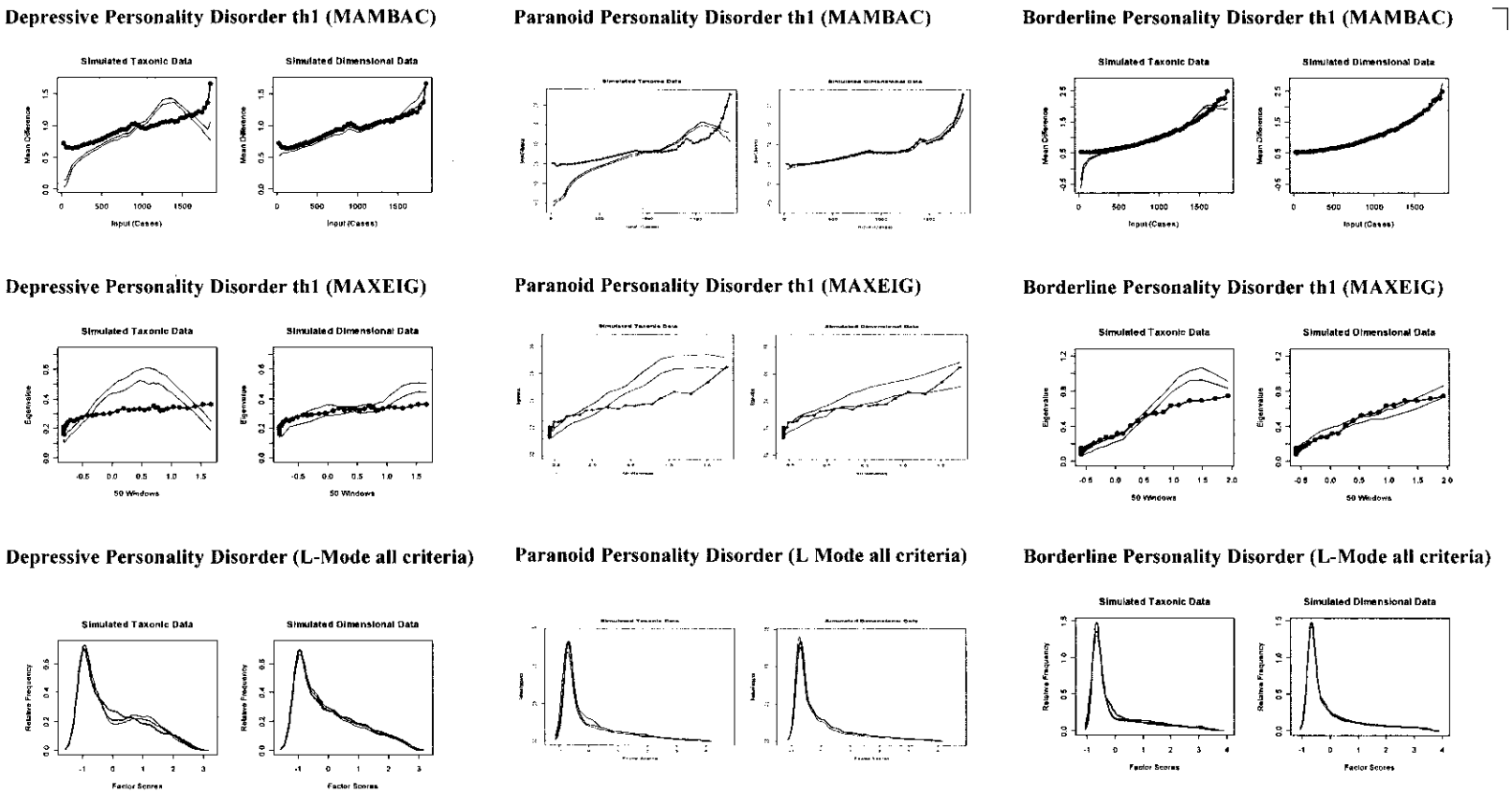

FIGURE 2. Observed (bold) and simulated curve ranges of MAMBAC and MAXEIG procedures for the first theoretical indicator sets and of L-Mode for all criteria of Depressive PD (left column), Paranoid PD (middle column) and Borderline PD (right column). Observed curves are averaged across results of all permutations of indicator sets (MAMBAC, MAXEIG). For both taxonic and dimensional structures, 10 simulations were done. Observed curves resemble simulated dimensional curves better than simulated taxonic curves.

mates, range .10–.22 (Table 1). The base rate estimates from the MAXEIG procedure were more consistent (mean .14, range .10–.17). L-MODE yielded a much higher base rate estimate (.50). All RMSRs and CCFIs (range .06–.35) favoured dimensional structure.

Obsessive-Compulsive PD. The graphs suggested stronger fit with dimensional than with taxonic simulations. Inchworm tests gave no evidence for a hidden taxonic structure. Figure 1, right panel, shows graphs of MAMBAC and MAXEIG analyses with taxonic and dimensional simulations of the first theoretical set, and L-Mode graphs of all criteria. MAMBAC analyses yielded highly divergent mean taxon base rate estimates, range .15–.41 (mean = .26; Table 1). The base rate estimates from the MAXEIG procedure were a bit more consistent (mean .24, range .19–.33). DSM-IV defined Obsessive-Compulsive PD was present in 11.8% of the cases, much less than the estimates of these analyses. L-MODE yielded a much higher base rate estimate however (.499). RMSRs and CCFIs (range .10–.27) suggest dimensional structures.

Depressive PD. Visual inspection of the graphs unequivocally favoured dimensional structure. Inchworm tests gave no evidence for a hidden taxonic structure. Figure 2, left panel, shows graphs of MAMBAC and MAXEIG analyses with taxonic and dimensional simulations of the first theoretical set, and L-Mode graphs of all criteria. MAMBAC analyses yielded divergent mean taxon base rate estimates, range .08–.32 (Table 1). Again, base rate estimates from the MAXEIG procedure were more consistent (range .22–.32), the mean (.26) consistent with the mean of 6 MAMBAC analyses (.25). Depressive PD according to DSM-IV was present in much less cases (9.3%). L-MODE yielded a much higher base rate estimate (.499). RMSRs and CCFIs (range .12–.37) all indicated dimensional structures.

Paranoid PD. Visual inspection of simulation output graphs indicated good discrimination of taxonic and dimensional structures, and higher resemblance of observed data results with dimensional than with taxonic simulations. Inchworms tests did not indicate hidden taxonicity. Figure 2, mid column, shows examples. Estimated base rates within and across procedures diverged (Table 1). MAMBAC estimates ranged from .09 to .44, MAXEIG estimates were much lower (mean = .10) and consistent, range .09–.12, whilst L-Mode had a much higher estimate, .498. All but one fit index indicated greater evidence for dimensional than for taxonic structure (CCFI: .12–.31). One CCFI was in the ambiguous range (.43).

Borderline PD. The graphs indicated higher fit with simulated dimensional than with simulated taxonic distributions (Figure 2, right column). Estimated taxon base rates were relatively consistent (MAMBAC mean = .15, range .07–.20; MAXEIG mean = .10, range .10–.11). But, L-Mode had a divergent base rate estimate, .499. DSM-IV defined BPD was present in 9% of the cases. All CCFIs (range .14–.50) favored dimensionality however, with one exception (MAXEIG, random set 3), which pointed at equivocal evidence for taxonic and dimensional structure. Borderline PD sets were a bit hampered by too high within complement group correlations (perhaps

related to the strong internal consistency of the Borderline scale). Those analyses not suffering from this nuisance correlation all supported dimensional structure. In sum, Borderline PD was the only PD where in one analysis equal evidence for taxonicity and dimensionality was found, but the rest of the evidence was in favor of a dimensional structure.

Overall summary. All but two of the 78 analyses yielded greater evidence for underlying dimensional than for taxonic structure. Two analyses yielded indecisive results. The CCFI's had an average (and median) of .23 (*SD* .09; *SE* .011), indicating latent dimensional structure. In sum, the data quite consistently fitted dimensional simulations better than taxonic simulations.

DISCUSSION

Our data were suitable for taxometric analyses, as indicated by indicator-validity, nuisance correlations, and simulations of taxonic and dimensional structures, which yielded distinguishable graphs. Based on visual inspection, variability in base rate estimates, and most importantly on fit with simulations, all but two taxometric analyses indicated that each of the 6 PDs investigated should be viewed more as dimensional than as taxonic. The only two exceptions were one of the 12 CCFIs of BPD, and one of the 12 CCFIs of Paranoid PD, who were in the range of CCFIs that indicate ambiguity with regard to evidence for dimensional vs. taxonic structure. Thus, our results suggest that the six studied PDs should be conceptualized as extreme positions on an underlying dimensional construct. The findings on Borderline PD are in line with findings in four previous studies (Ayers, 2000; Rothschild et al., 2003; Simpson, 1994; Trull et al., 1990). To the best of our knowledge, there have been no earlier taxometric studies investigating the underlying structure of the other five PDs

It is noteworthy that taxometric studies so far have suggested that some PDs are taxonic, whereas others seem dimensional (Haslam, 2003; Rothschild et al., 2003). Taxometric studies quite consistently suggested that schizotypy or Schizotypal PD is taxonic; but divergence about Antisocial PD and psychopathy is reported (Ayers, 2000; Edens et al., 2006; Harris et al., 1994; Haslam, 2003; Marcus et al., 2004, 2006; Skilling et al., 2002; Vasey et al., 2005). The PDs we investigated appear to be dimensional. Perhaps Axis-II in the DSM-V should not be uniform in its use of dimensional or categorical approach, the choice depending on empirical evidence for each PD (Haslam, 2003; Rothschild et al., 2003).

Evidence that a PD has a dimensional nature does not necessarily imply that it can be adequately understood and described by dimensional models of normal personality, although some tend to conclude so (Haslam, 2003; Rothschild et al., 2003; Edens et al., 2006). This is because the PD criteria may not have been incorporated in studies of normal personality, or may be so infrequently reported in samples from normal population, that derived personality models don't adequately represent specific mal-

adaptive features of PDs (Pukrop & Krischer, 2005; Shedler & Westen, 2004). Furthermore, in normal samples more general factors, like neuroticism, might overshadow more subtle dimensions that are informative for clinicians. Sampling strategies strongly influence the degree to which general differences in psychpathology dominate factor analytic studies, or whether more subtle factors in types of psychopathology can be found. Thus, although PDs may be dimensional, PD dimensions might be only detectable in samples with high rates of pathological behaviors. The degree to which normal personality models, like the 5-factor model (Costa & Widiger, 2001; Lynam & Widiger, 2001) can sufficiently cover the type of personality problems clinicians deal with should in our view be a research issue in itself, and not be solely decided on the basis of evidence for dimensionality of PDs. Thus, in our view the present findings should not be taken as *direct* evidence that PDs represent the extremes of normal personality dimensions.

Confirmatory factor analysis demonstrated that criteria of the 6 PDs we investigated fitted very well with a model with each PD representing a separate factor. This is in line with a model in which each PD is represented as a separate dimension (or taxon), and replicates an earlier study with DSM-III-R based SCID-II data (Arntz, 1999). Further studies are needed to test this issue over the full range of DSM-IV PDs.

One limitation of the present study is that the proportion of putative taxon members was rather low when we base the proportions on the usual DSM-IV diagnoses (except for Avoidant PD). There is lack of fundamental research into the issue to what degree taxometric procedures can detect taxa when present in small proportions. Nevertheless, one Monte Carlo demonstration suggests that especially MAXEIG is capable to detect very small taxon proportions (up to .0025) when the taxon sample size is $n = 100$, and the other conditions are favorable (good indicator validity, low nuisance correlations, no indicator skew; Waller & Ross, 1997). Note that five of the six PDs investigated in the present study had sample sizes from very near 100 (Paranoid, $n = 95$) up to 400 (Avoidant, $n = 400$); and four of the six PDs were present in 9% or more of the cases (Avoidant, 21.3%; Obsessive-Compulsive, 11.8%; Depressive, 9.3%; Borderline, 9.0%), so that we should have been capable of detecting taxonicity in at least 5 of the 6 PDs. Only Dependent PD was perhaps problematic in this respect ($n = 74$, 3.9%). Furthermore, not arbitrarily defined taxon boundaries, as DSM-IV thresholds are more or less, but "true" boundaries influence the validity of the analyses. Base rate estimates from the taxometric analyses for all six PDs were well above 10%. In further support of high enough putative taxon sample sizes, the graphs of dimensional and taxonic simulations were clearly distinguishable. Lastly, one empirical study demonstrated that taxometric procedures were capable to detect a taxon with a sample size as low as $n = 35$ in a total sample of $N = 1{,}055$ (Waller & Ross, 1997).

A similar issue is the sample size of the complement class. If not large enough, failure to detect taxa might result. The present data set contained

data from 63 nonpatients and 1,079 patients without PD. Furthermore, the sample sizes of subjects not meeting any (subthreshold) criterion of the 6 PDs were: Avoidant PD: 596; Dependent PD 772; Obsessive-Compulsive PD 522; Depressive PD 553; Paranoid PD 882: Borderline PD: 910. A total of 111 participants did not meet any (subthreshold) criteria of these 6 PDs. Taken together, these figures indicate that the complement classes were large enough to detect taxa.

Another limitation is that taxon studies are only as good as the indicators that are used in the study. Some have questioned whether PDs are well-represented by the DSM-IV criteria. Other criteria might have produced other results.

A third limitation is that taxometrics represent only one means of investigating the latent structure of PDs. Evidence from taxometric studies should be combined with results from other types of studies. Nevertheless, our results converge with results from a wide variety of approaches, including factor analysis (Blackburn et al., 2005), stability studies (Durbin & Klein, 2006), and validity studies (Pukrop & Krischer, 2005; Saulsman & Page, 2004; Skodol et al., 2005; Ullrich et al., 2001).

A fourth limitation is related to the statistical methods we used. Although there is increasing evidence for the robustness and validity of the methods we used, and especially for the use of bootstrap methods to create comparison samples and to calculate the CCFI, these methods are relatively new, and therefore our conclusions are accordingly tentative. More specifically, higher number of bootstrap samples might have lead to somewhat more precise estimates, although a recent study indicated that the increase in accuracy of the CCFI was negligible when the number of bootstrap samples was increased from 10 to 50 (Ruscio & Kaczetow, under review). Future fundamental studies should determine to what degree more drastic increases in number of bootstrap samples result in increased accuracy. On the other hand, Ruscio and Kaczetow (under review) demonstrated that CCFIs outside the .40–.60 range have very high accuracies, above 95% for a variety of sample distribution conditions. As 70 of the 72 CCFIs in the present study were smaller than .40, 2 were in the ambiguous range, and none was larger than .60, our results seem robust in indicating that the underlying constructs are dimensional.

Strong points of our study include the use of clinical interview-based criterion scores, which reduce nuisance correlation caused by response bias (Rothschild et al., 2003) and relate the findings to the DSM-IV construct of PDs. We had a very large sample, varying from nonpatients, to axis-1 patients with none to some PD features, to patients with different kinds and severities of PDs. Care was taken to avoid pseudotaxonicity, which can result when two extreme samples (e.g., nonpatients and PD patients) are combined in the dataset. Furthermore, we assessed both randomly grouped indicator sets and sets constructed on theoretical content considerations, and results were consistent. We also used multiple taxometric procedures to test for consistency. The use of simulations and the

recently developed CCFI, made our analyses more objective as we did not have to rely on subjective decisions about graphs.

A treatment implication of dimensional views on PDs might be that it is not wise to view these PDs as completely different from axis-1 disorders, not amenable, for instance, to psychotherapy. Recent findings indicate that specialized forms of psychotherapy that build upon methods and techniques with demonstrated effectiveness for axis-1 disorders are indeed effective for many PDs (Giesen-Bloo et al., 2006; Leichsenring & Leibing, 2003; Perry, Banon, & Ianni, 1999).

In conclusion, we found strong and consistent evidence for latent dimensions underlying borderline, paranoid, depressive, and cluster-C PDs. Replications are needed, especially for the five PDs that we were the first to taxometrically analyze. More important, the PDs that were not yet taxometrically investigated (Schizoid, Passive-Aggressive, Narcissistic, and Histrionic PD) should be tested. These studies will help to better understand personality pathology, aid diagnoses, and case conceptualizations, and hopefully help to base the DSM-V PD constructs on empirical grounds.

APPENDIX. RATIONALE OF THEORETICAL CRITERIA SETS

AVOIDANT PD

The first theoretical indicator set consisted of indicator 1 = social avoidance (1, 2); indicator 2 = inferiority (3, 6); indicator 3 = embarrassment (4, 5, 7). The second theoretical set of indicator 1 = risk avoidance (2, 7); indicator 2 = contact avoidance (1, 3); indicator 3 = (self)criticism (4, 5, 6).

DEPENDENT PD

The first theoretical indicator set consisted of indicator 1 = decision making (1, 2); indicator 2 = subjugation/self-sacrifice (3, 5); indicator 3 = cannot be alone (4, 6); indicator 4 = abandonment (7, 8). The second theoretical set of indicator 1 = makes others responsible (1, 2, 4); indicator 2 = subjugation/self-sacrifice (3, 5); indicator 3 = needs others for care (6, 7, 8).

OBSESSIVE COMPULSIVE PD

The first theoretical indicator set consisted of indicator 1 = details & perfectionism (1, 2); indicator 2 = productivity (3, 5); indicator 3 = rigidity and norms (4, 6, 7, 8). The second theoretical set of indicator 1 = control (1, 6); indicator 2 = economy (3, 5, 7); indicator 3 = high standards (2, 4, 8).

DEPRESSIVE PD

The first theoretical indicator set consisted of indicator 1 = worthlessness (2, 7); indicator 2 = criticism (3, 5); indicator 3 = pessimism (1, 4, 6). The second theoretical set of indicator 1 = negative mood & self-view (1, 2, 7); indicator 2 = criticism (3, 5); indicator 3 = worry (4, 6).

PARANOID PD

The first theoretical indicator set consisted of indicator 1 = preoccupation with idea that others have bad intentions (criteria 1, 2); indicator 2 = trusting issues (criteria 3, 4, 7); and indicator 3 = anger and grudge (criteria 5, 6). The second theoretical set consisted of indicator 1 = suspiciousness (criteria 2, 7); indicator 2 = abuse expectation (1, 3); indicator 3 = hypersensitivity (4, 5, 6).

BORDERLINE PD

The first theoretical indicator set (see Sanislow et al. 2002;) consisted of indicator 1 = disturbed relatedness (2, 3, 7, 9); indicator 2 = behavioral dysregulation (4, 5); indicator 3 = affective dysregulation (1, 6, 8). The second theoretical set (derived from Clarkin et al., 1993; Hurt et al., 1990; Morey, 1991; Livesley & Schröder, 1991; Zanarini et al., 1989) of indicator 1 = unstable self/relationships (1, 2, 3); indicator 2 = impulsive (self-) damaging behavior (4, 5, 8); indicator 3 = affective problems (6, 7, 9).

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