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REFINING THE BORDERLINE PERSONALITY DISORDER PHENOTYPE THROUGH FINITE MIXTURE MODELING: IMPLICATIONS FOR CLASSIFICATION

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Borderline personality disorder (BPD) is characterized by considerable heterogeneity. Prior approaches to resolving heterogeneity in BPD pathology have used factor and cluster analytic as well as latent class analysis strategies. These prior studies have been atheoretical in nature, but provide an initial empirical corpus for further sub-typing efforts in BPD. A *model-based* taxonomy for BPD that is supported by evidence from an advanced statistical methodology would enhance investigations of BPD etiology, pathophysiology, and treatment. This study applied finite mixture modeling analysis, in a *model-guided* fashion, to selected dimensions of pathology within a group of well-characterized BPD patients to determine if latent groups are harbored within the disorder.

Subjects with BPD ($N = 90$) were examined on a variety of model-relevant psychopathology dimensions. We applied finite mixture modeling to these dimensions. We then evaluated the validity of the obtained solution by reference to a variety of external measures not included in the initial mixture modeling.

Three phenotypically distinct groups reside within the overall BPD category. Group-1 is characterized by low levels of antisocial, paranoid, and aggressive features. Group-2 is characterized by elevated paranoid features, whereas Group-3 is characterized by elevated antisocial and

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aggressive features. External correlates reveal a pattern of differences consistent with the validity of this proposed grouping structure.

A theory-guided finite mixture modeling analysis supports a parsing of the BPD category into three subgroups. This proposed BPD taxonomy represents an approach to reducing heterogeneity observed among BPD patients and it may prove useful in studies seeking to understand etiologic and pathophysiologic factors as well as treatment response in BPD.

Borderline personality disorder (BPD), with a prevalence of 1.4% (Lenzenweger, Lane, Loranger, & Kessler, 2007), has long been known for its heterogenous phenomenology (Stern, 1939; Knight, 1954; Kernberg, 1967, 1975, 1984; Grinker, Werble, & Drye, 1968). As forms of psychopathology go, BPD is, perhaps, one of the most heterogenous constructs including diverse affective, behavioral, and cognitive features. First explicitly defined in the DSM-III American Psychiatric Association, 1980), the BPD phenotype articulated by the DSM-III and subsequent revisions (DSM-IV; American Psychiatric Association, 1994) is clearly *polythetic* in nature and this is, in part, a driving force behind the considerable heterogeneity observed across patients that receive the diagnosis. Such phenomenological heterogeneity has posed challenges to the reliable and valid assessment of the illness and, importantly, it remains a formidable roadblock to research seeking to illuminate the etiology and basic pathophysiology of the disorder. The presence of a great deal of heterogeneity in the BPD diagnosis also impedes the resolution of viable endophenotypes for the illness (Gottesman & Gould, 2003). Additional, excessive within disorder phenomenolical heterogeneity has slowed progress in the refinement of therapeutic strategies differentially targeted at salient features of the disorder.

The vast phenomenological heterogeneity found among those diagnosed with BPD has led to a number of different approaches, all seeking to bring clarity and resolution to this classification challenge. Early efforts to resolve phenomenological heterogeneity in BPD relied on exploratory factor analytic (EFA; Clarkin, Hull, & Hurt, 1993; Sanislow, Grilo, & McGlashan, 2000) or cluster analytic techniques. The EFA work sought to reduce the wide variety of BPD symptoms/features to a smaller number of broad BPD factors, whereas the clustering work sought to determine if there were meaningful sub-types of BPD. All of this work, which proved highly valuable in setting the stage for more recent efforts, was atheoretical in nature, which means the statistical approaches were not constrained or guided by any form of substantive theory or model. EFA results of BPD symptom features revealed that the *DSM* BPD diagnostic criteria were characterized by three broad underlying dimensions, affective disturbance, identity disturbance, and impulse dyscontrol (Clarkin et al., 1993; see also Sanislow et al., 2000). Seeking to classify people rather than variables, Grinker et al. (1968) reported a seminal cluster analytic study of psychological fea-

tures of 51 BPD patients and argued for a clustering solution that divided patients into four groups/sub-types (Group-1—on the psychosis border, Group-2—core borderline, Group-3—affectless, defended, as-if, and Group-4—on the neurosis border). The clustering approach of Grinker et al. (1968) yielded other possible solutions and therefore the reported four-group sub-type scheme was not regarded as definitive by those authors. These empirical solutions provided a necessary and logical first step in efforts to bring order to the vast array of symptoms and signs that are known to suggest BPD, particularly as outlined by the DSM system.

The heterogeneity of the BPD phenotype has increasingly become the focus of energetic research, stimulated in part by increased interest in the underlying nature of BPD and related putatively causal processes involving both neurobehavioral processes and genetic influences (e.g., Cloninger, Svrakic, & Przybeck, 1993; Depue & Lenzenweger, 2001, 2005; Livesley et al., 1998). For example, a recent confirmatory factor analysis (CFA) of BPD features (Sanislow et al., 2002) yielded fascinating results consistent with either one-factor or three-factor models. The one-factor model (CFA normed fit index = .95) could be taken to suggest[1] a common underlying pathological process/construct, whereas the three-factor model (CFA normed fit index = .95) was highly consistent with the earlier EFA solutions suggesting affective disturbance, identity disturbance, and impulse dyscontrol as identifiable latent dimensions. There have also been renewed efforts to classify individuals into BPD sub-types. An interesting recent study by Bradley, Conklin, & Westen (2005) employed *Q*-factor analysis to discern types in practice network clinician ratings of adolescent patients and they found evidence for four sub-types (or sub-groups) of BPD among the girls they studied, however the statistical effectiveness of *Q*-factor analysis in resolving types has been questioned (Waller & Meehl, 1998). Notwithstanding this statistical issue, their (Bradley et al., 2005) four BPD subtypes are well worth considering and were described as high functioning internalizing, histrionic, depressive internalizing, and angry externalizing BPD. In this context, we also note several studies have examined the latent class structure of BPD diagnostic criteria in *mixed* psychiatric samples consisting of a wide range of psychopathology beyond

1. Sanislow et al. (2002) expressed their enthusiasm for the three-factor model in the CFA study as it provided a statistically significant improvement in model fit relative to the unidimensional (one-factor) model. However, given the well-known relationship between the chi-square statistical test and sample size, the evidence suggesting an improvement in model fit for the three-factor vs. one-factor model must be weighed against the sample size ($N = 668$).

2. Moreover, although latent class analysis can be viewed as the analytic method for dichotomous data broadly analogous to finite mixture modeling (which uses continuous data as noted), we believe that LCA discards valuable quantitative data as it does require only categorical input. We see the individual difference variation on our input variables as highly valuable, in this case as in most instances in psychopathology research, and we prefer such information to be available to an analysis. Additionally, the local independence assumption of LCA is often not met for highly covarying symptom dimensions, constraints which limit the resolving power of the method; finite mixture modeling does not require local independence.

BPD (Fossati et al., 1999; Thatcher, Cornelius, & Clark, 2005; Clifton & Pilkonis, 2007), however such studies have diminished probative value for questions regarding heterogeneity *within* BPD itself.[2]

Where do these studies leave us with respect to parsing the phenomenological heterogeneity of BPD? Each of these studies offers a useful stepping stone in the pathway toward a principled approach to classifying BPD. Although the EFA/CFA factor analysis studies are not directly comparable to the more typological efforts (e.g., clustering; *Q*-factor analysis), the weight of the evidence suggests that BPD is not likely to be characterized or understand as a homogenous disorder at the latent level. This alone, is important as it suggests that the heterogeneity is probably genuine, not merely an artifact of samples or analytic techniques, and still represents a classification challenge. In our view, an important feature that needs to be brought to this work is a *theoretical model* that could be used as a guide in informing future statistical efforts aimed at parsing heterogeneity of symptoms and signs in BPD. As noted earlier, all prior efforts were *atheoretical*, including the CFA work that assessed the fit of the previously obtained EFA solutions. Several of the studies restricted themselves to the actual DSM BPD diagnostic criteria as input indicator variables for analysis, others used a wide range of input variables that were not selected on any theoretically-guided a priori basis. We suggest a useful advance in these classification efforts might be found in coupling an explicit theoretical model with a statistical approach. Moreover, we think that attempting to parse BPD patients as defined by the DSM system, but using variables other than the diagnostic criteria themselves might provide useful leverage on this problem. Such an effort would clearly build upon the prior studies in this area.

We also note that from the statistical point of view, improvements in analysis might be found in some of the more recently developed methods. Unfortunately, neither factor nor cluster analytic procedures are well suited to the classification problem at the heart of the BPD heterogeneity question, namely "are there different types of BPD patients?" For example, is it well-known that factor analytic strategies organize indicator features into larger dimensional composite variables, but do not really allow one to sort individuals into meaningful subgroups. Cluster analysis routines, despite their intuitive appeal, vary considerably in their classification accuracy and, unfortunately, often fail to detect clear-cut subgroups (Golden & Meehl, 1980). Moreover, in most applications cluster analysis approaches rely on impressionistic approaches to the determination of the optimal number of clusters in a solution (Lenzenweger, Jensen, & Rubin, 2003), not unlike determining the number of factors in a scree plot of eigenvalues.

In short, the BPD phenotype remains characterized by a daunting degree of phenomenological heterogeneity and a way forward in refining this phenotype is needed. We argue that efforts seeking to refine the BPD phenotype would proceed best with (a) a clinically informed model that makes

unambiguous predictions regarding the clinical phenomenology of putative BPD subtypes and (b) a well-principled statistical methodology that is not subject to the limitations of previously employed techniques. Use of a theoretical model to guide variable selection for analyses seeking to parse individuals that carry the BPD diagnosis could potentially yield a more clinically meaningful set of variables for analysis as well as subject the theoretical model itself to empirical scrutiny.

In brief, our theoretical model derives from the extensive clinical experience with and substantive considerations for BPD developed by Kernberg (Kernberg, 1984; Kernberg & Caligor, 2005). Kernberg's diagnostic classification system for the personality disorders, which combines dimensional and categorical features, hypothesizes that all severe personality disorders, including BPD as defined by DSM-IV, exist at what is termed the borderline level of personality organization (BPO). Therefore, we emphasize that we are discussing predictions regarding BPD specifically as it is manifested within the BPO realm. Importantly, Kernberg has explicitly predicted that BPD-related psychopathology can be parsed into two well-delimited sub-types, namely high vs. low level borderline personality organization (BPO), distinguished by a higher level of aggressive affect in low level BPO BPD patients. Moreover, an auxiliary theoretical conjecture suggests that within the low-level BPO, one should see a further separation of BPD patients at that level as a joint function of broadly defined internalizing vs. externalizing personality style (with a particular focus on paranoid cognitive tendencies), externalizing aggression, and explicitly antisocial behaviors. The hypothesis regarding the separation of BPD patients as a function of low vs. high level BPO as well as the hypothesis of further differentiation of those BPD patients within the low-level BPO realm, suggests a taxonomy that is, at minimum, composed of two BPD classes with a three-class BPD model as highly plausible.

The proposed BPD sub-types should vary in the manner in which, broadly, aggression is manifested (namely, directed at either the self or others) and this variation is revealed specifically via configural relations among paranoid, antisocial, and aggressive features (i.e., outwardly expressed vs. not expressed outwardly). Translating Kernberg's theoretical conjectures into plausible phenomenological-clinical entities, one might find a BPD subtype that is higher level and is relatively free of paranoid features, antisocial behaviors, and externalized aggression. Within the low level BPO range, one might find a BPD subtype characterized by relatively high level of paranoid features (highly inwardly directed), but comparatively low levels of externalizing aggression and antisocial features. Finally, also within the low level BPO range, a third BPD subtype characterized by relatively high levels of extraversion, antisocial behavior, and externalizing aggression. We note in the first low level group, a paranoid clinical posture is maintained and aggression is presumably tightly contained via this psychological adaptation, whereas in the second low level group aggression predominates the presentation and is visible through antisocial behavioral

enactments suggestive of a breakdown in control over aggression and erosion of moral values. In sum, Kernberg's model predicts at least two classes of BPD patients within the BPO phenotypic space, with three classes being a plausible alternative possibility. In reviewing this model, we seek to clearly articulate our theoretical model that will be used to guid our statistical analyses and we emphasize we will not seek to analyze the DSM BPD criteria, but rather the clinical dimensions explicated by Kernberg's model as a basis for sub-typing BPD patients.

How best to proceed statistically with this challenge? Clearly, although methods such as factor analysis and cluster analysis have a solid statistical basis, they do not offer the strongest tools for this classification problem. A viable alternative statistical approach to heterogeneity in BPD can be found in a statistically well principled procedure known as *finite mixture modeling* (Titterington, Smith, & Makov, 1985; McLachlan & Peel, 2000).[3] Multivariate mixture modeling has seen only modest application in psychopathology research (e.g., Lenzenweger, McLachan et al., 2007). What is it that finite mixture modeling does statistically? In brief, finite mixture modeling seeks to resolve the number of components (consisting of cases) that are intermixed within a larger combined population. As such, it is ideally suited to the problem of heterogeneity in BPD. The approach is statistically superior to conventional clustering routines as it does not make untenable assumptions regarding the shape and covariance structure of latent groups within data as do most clustering methods. A very attractive feature of finite mixture modeling is that it provides a statistical framework (i.e., a likelihood) to evaluate the number of components residing within data. Although regularity conditions do not hold for the likelihood ratio test (LRT) statistic for tests on the number of components to have its usual null distribution of chi-squared in finite normal mixture models, it can be bootstrapped to provide *p*-values. Also, there is much empirical evidence to suggest that two information criterion measures provide useful guides as to the number of components in a normal mixture modeling approach (McLachlan & Peel, 2000) and we employ them as supplements to the bootstrapped LRT. These two supplemental information criteria are the Akaike Information Criterion and the Bayesian Information Criterion.[4]

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3. We do not view this as the forum for an extended introduction to finite mixture modeling in an effort to conserve space. Rather we would suggest that interested readers should consult established sources that contain extensive detail. Useful technical introductions can be found in Titterington et al. (1985) and McLachlan and Peel (2000); a conceptual introduction with special relevance for psychopathology can be found in Lenzenweger, McLachlan, and Rubin (2007). We also forego an extended discussion of statistical fine points that bear upon technical matters, such as local solutions in mixture modeling. These issues are well covered in the sources noted previously as well as in recent papers (e.g., Hipp & Bauer, 2006).

4. We are aware of other approaches to determining the number of components in finite mixture modeling analyses. For example, there is the Lo, Mendell, and Rubin (2001) test, however that test was originally developed for use with equal covariances. We note we estimated our models using unrestricted covariances. The Lo et al. (2001) remains to be investigated for unrestricted covariances. There is also the method of posterior predictive checks/*p*-values,

The manner in which finite mixture modeling is similar to or different from other latent structure analysis techniques is likely to be of interest to personality disorders researchers. Given the emerging popularity of taxometric (Meehl, 1995; Lenzenweger, 2004) analytic techniques in psychological science research as a technique for examining latent structure, we note here, briefly, some similarities and differences between taxometric and finite mixture modeling techniques. To begin, for the two class situation, taxometric methods (like latent profile analysis) and finite mixture modeling share a comparable analytic model (Bauer & Curran, 2004), but they are methods designed to answer different substantive questions. Taxometric methods (Meehl, 1995; Meehl & Yonce, 1996; Waller & Meehl, 1998) are typically used to determine whether or not a single quantitative dimension or a two class qualitative latent structure best characterizes the structure underlying observed quantitative data. In distinction, finite mixture modeling is used to determine how many latent normal components best characterize observed quantitative data (Titterington et al., 1985; McLachlan & Peel, 2000). Important differences between the two families of methods are worth noting. Taxometric methods do not yield a data partition if there are no discrete groups embedded in a multivariate distribution, whereas finite mixture modeling yields partitions in the underlying data and fit measures as well as comparison statistics (e.g., resampling based likelihood ratio tests) are used to discern the number of latent components. Finite mixture modeling, importantly, directly reproduces the covariance structure of the data, whereas taxometric methods do not. Furthermore, finite mixture modeling does not assume local independence of indicators within groups, but does explicitly assume normality within groups, an assumption that is used in the estimation of model parameters. Taxometric methods do assume local independence of indicators within groups and the methods do not explicitly assume multivariate normality, even though the latter typically receives little attention in applications. Finally, as presently developed, taxometric methods are really only capable of detecting two latent classes if they exist, whereas finite mixture modeling can detect any number of latent components (i.e., 1, 2, 3, or more). Beauchaine (2003) provides additional useful discussion of the differences between these two families of statistical methods.

One might also ponder a comparison of a popular cluster analysis method, K-means clustering, and finite mixture modeling. The use of K-means clustering is essentially equivalent to fitting mixtures of normal distributions using method-of-moments arguments with a common spherical covariance matrix. That is, it produces spherical clusters of common size. But often in practice the clusters are elliptical in shape and may have different scales or orientations. The latter clusters are allowed under a normal mixture model with component distributions having unrestricted

developed by Rubin and colleagues (Rubin, 1984; Gelman, Meng, & Stern, 1996), which may be appropriate in some analytic contexts.

covariance matrices. With the normal mixture model, the clusters are invariant under change in location, scale, and rotation, of which the first two (that is, invariance under location and scale) are highly desirable. Hierarchical agglomerative methods are considerably dependent on the metric adopted. As noted above, a substantial advantage of finite mixture modeling over K-means and hierarchical clustering methods is that it provides a statistical framework (i.e., a likelihood) to evaluate how many clusters there are in the data.

We sought to determine if the three-group taxonomy proposed by Kernberg could be discerned in a group of well-characterized BPD patients (who present with other forms of Axis II pathology as well) using finite mixture modeling. Furthermore, the current study sought to evaluate the validity of the obtained solution by examining relations of the obtained solution with external correlates of validity.

METHOD

SUBJECTS

The 90 subjects were (male = 7, female = 83) between the ages of 18 and 50 who met DSM-IV-TR criteria for BPD. They were drawn from the greater New York City area, covering areas of New York, New Jersey, and Connecticut. They came from several and varied recruitment streams, including both clinic settings as well as from the community, thus they represented a broad cross-section of BPD patients and were not a highly selected, inpatient treatment-based group of BPD patients. Individuals with comorbid psychotic disorders, bipolar I disorder, delusional disorder, delirium, dementia, and/or amnestic as well as other cognitive disorders were excluded. Those with active substance dependence were excluded, although patients with past substance dependence and past and current substance abuse were included. Patients were screened in telephone interviews for age and other inclusion criteria. Suitable patients were assessed in face-to-face evaluations with trained evaluators. BPD was diagnosed by the *International Personality Disorder Examination* (IPDE; Loranger, 1999) and the symptom ratings were reliable, $ICC(1,1) = .83$), as were the categorical diagnoses (kappa = .64). Exclusion diagnoses were based on SCID interview (First, Spitzer, Gibbon, & Williams, 1996). Despite being drawn from the community, the BPD patients in this study were relatively impaired as evidenced by their global functioning score as well as number of comorbid Axis I and Axis II disorders and probably represents a typically heterogenous sample of BPD individuals as they appear in the population. Importantly for the purposes of this study, these patients are not a rarified group of BPD-only subjects with no other comorbid psychiatric conditions. In fact, mindful that BPO constructs apply to a broad range of PDs, we note the BPD patients in this study often had two or more other Axis II diagnoses and, thus, they represent a more diverse and varied group particularly

well suited for evaluation of BPO-related theoretical constructs. Finally, we note a subset of these subjects were studied in a randomized controlled treatment trial (Clarkin, Levy, Lenzenweger, & Kernberg, 2007), however the current report draws upon baseline individual difference data for all the study subjects prior to participation in that treatment trial.

METHODS

International Personality Disorder Examination (IPDE). The IPDE is the well-known semi-structured interview procedure that assesses both *DSM* and *ICD-10* PD features (Loranger, 1999). The diagnosticians for the initial BPD diagnostic assessments were highly experienced clinicians with considerable exposure to severe Axis II psychopathology. They all received training from A. W. Loranger in the administration and scoring of the IPDE as well as supervision from senior diagnosticians at the study site. As noted, the BPD symptom ratings were reliable, $ICC(1,1) = .83$, as were the BPD categorical diagnoses ($kappa = .64$).

Structured Clinical Interview for DSM-IV (SCID). The SCID is the well-known semi-structured interview for the assessment of Axis I disorders (First et al., 1996). As noted, the clinicians conducting the baseline assessments were highly experienced diagnosticians with extensive inpatient and outpatient experience with severe psychopathology, including Axis I and Axis II conditions.

Inventory of Personality Organization (IPO). The IPO (Clarkin, Foelsch, & Kernberg, 2001) is an 83-item self-report psychometric inventory that assesses five clinical dimensions relevant to the diagnosis of borderline personality organization (Kernberg, 1967, 1975, 1984). The present study used the aggression scale of the IPO in the finite mixture modeling. The IPO exhibits generally excellent psychometric properties (Lenzenweger, Clarkin, Kernberg, & Foelsch, 2001) and shows evidence of construct validity.

External Validity Measures. Kernberg's model also allows predictions regarding the relative ordering of the three proposed BPD sub-groups on relevant dimensions of personality, social functioning, abuse history, and other variables of clinical significance. The following dimensions, assessed using sound psychometric instruments, were used in our model-guided comparisons across the groups revealed in the finite mixture modeling. Social closeness (affiliative positive emotion), negative emotion, and constraint were measured using the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982); recent level of occupational functioning was assessed using the Social Adjustment Scale (SAS; Weissman & Bothwell, 1976); self-reported childhood sexual abuse and childhood physical abuse from the Childhood Trauma Questionnaire (CTQ; Bernstein, Ahluvalia, Pogge, & Handlesman, 1997); cognitive-motor impulsivity (Factor 1) from the Barratt Impulsiveness Scale (BIS; Patton, Stanford, & Barratt, 1995); narcissistic personality disorder dimensional score from the IPDE (Loranger, 1999); identity diffusion as measured by the IPO (Clarkin et al., 2001;

Lenzenweger et al., 2001); and total psychopathy dimensional score from the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996). Additionally, all patients were assessed for the total number of types of social services they had ever utilized.

STATISTICAL ANALYSIS

Finite mixture analysis (Titterington et al., 1985; McLachlan & Peel, 2000) was conducted using the UNIX-based program EMMIX (v. 1.3). Finite mixture modeling analysis seeks to resolve the most likely number of normal components underlying a multivariate array of continuous/quantitative data. In short, it seeks to identify substantially well defined and coherent sub-groups within a larger population of subjects, looking for what is termed a possible *mixture* of sub-groups within the larger overall group. We evaluated the number of components underlying aggression (IPO) and IPDE antisocial and paranoid PD features considered simultaneously. Our a priori prediction, suggested 3 components should underlie this array of clinical features. Therefore, we tested 1 through 4 component models. Evaluation of model fit was done by comparisons of the bootstrapped likelihood ratio (McLachlan & Peel, 2000). We also used the Akaike (AIC) and Bayesian (BIC) Information Criteria as supplemental guides to assess fit. Fit statistics do not always agree with one another with respect to model selection, thus we decided a priori to be guided by the LRT results as well as the preponderance of evidence across the two supplemental fit indices (AIC, BIC).

Does the use of three measures as input indicators in the finite mixture modeling analysis assure that three classes will be found in the data? Certainly, it does not. Here we note it is important to be aware of the fact that a single indicator variable could actually yield evidence of multiple groups harbored within the overall distribution (e.g., Lenzenweger & Moldin, 1990). Moreover, the use of three or more input measures could just as likely identify a single group within a multivariate arrary, i.e., find no evidence of mixture. Thus, one should not assume that the use of three indicators will necessarily yield three groups. Rather, the method is designed to allow one to determine how many groups, if more than one, are harbored within a larger sample of cases defined by multiple quantitative variables.

After resolving the number of components underlying the three variables, we obtained posterior probabilities for component membership for each subject. These probabilities were then used to assign individuals on a case-by-case basis to the component in which they most likely belonged. The component membership was then used to group subjects in subsequent comparisons using other criteria of validity (focused constrast analysis; Rosenthal & Rosnow, 1991). Evaluation for the presence of group differences across independent (i.e., external to the mixture modeling analysis), but clinically relevant, dimensions of personality and pathology

represents an initial effort to validate a proposed grouping or parsing strategy (see Cronbach & Meehl, 1955). Contrast analysis confers greater power and precision than conventional unfocused ANOVA in answering focal questions. Effect-sizes are reported as effect-size $r_{contrast}$.

RESULTS

The demographic and clinical characteristics of the sample are in Table 1. The mean GAF score ($X = 49.49$; $SD = 9.57$) for the sample suggests serious symptoms and/or serious impairment in one area of functioning as defined by the DSM-IV-TR. Moreover, there was a substantial amount of Axis I comorbidity present in the sample as would be expected. The average number of Axis II disorders, including BPD ($X = 2.49$, $SD = 1.13$; range 1 to 6 Axis II diagnoses), suggests the presence of substantial and reasonably anticipated Axis II comorbidity. Comorbid Axis II PD diagnoses in the 90 BPD patients included paranoid (27%), schizotypal (3%), antisocial (21%), histrionic (24%), narcissistic (18%), avoidant (31%), dependent (11%), and obsessive-compulsive (14%). The presence of extensive Axis II comorbidity in these 90 BPD-diagnosed patients suggests that our sample contained a range of Axis II pathology, above and beyond BPD, and, thus, provides important clinical variation within the patient sample consistent with Kernberg's BPO formulation. In short, the patients for this sample represented a wide range of severe impairment as well as substantial Axis I and Axis II comorbidity.

We note that the IPDE antisocial and paranoid PD dimensional scores and IPO aggression values did not differ significantly across men and

TABLE 1. Sample Characteristics

N	90
Sex	83 Female, 7 Male
Age (yrs)	31.06 (7.81)
Ethnicity	
White (Caucasian)	67.8%
African-American	10.0%
Hispanic	8.9%
Asian-American	5.6%
Mixed/Other Ethnicity	7.7%
Education	
Less than High School	3.3%
High School/GED	7.8%
Some College	31.1%
Associates Degree	6.7%
BA, BS (4 Years College)	32.2%
Graduate/Post-Graduate	18.9%
Global Functioning Scale (GAF) M (SD)	49.49 (9.57)
Axis I Disorders (lifetime)	
Major Depression	47.8%
Anxiety Disorder (any)	55.6%
Substance Dependence (any/including alcohol)	14.4%
Eating Disorder (anorexia/bulimia)	21.1%
Total Axis I Disorder Diagnoses (any) *M* (*SD*)	3.80 (2.72)
Total Axis II Disorder Diagnoses *M* (*SD*)	2.49 (1.13)

women. These values were standardized (z-scores) to ensure a commensurable metric across the three measures. We note that the distributions for these variables were not excessively skewed, thus overextraction of components due to skewness was not a concern (McLachlan & Peel, 2000).

To test the two-class and three-class possibilities predicted by Kernberg's model, we conducted EM-based finite mixture analyses 1 through 4 normal component models (unrestricted covariance matrices). Thus, one statistical analysis allows us to test the primary high vs. low level conjecture as well as the possibility that the low-level disorders will parse into two classes. The results for these model fits are contained in Table 2 based on 100 bootstrap replications as recommended by McLachlan and Peel (2000). However, we also note that our mixture modeling results/solutions were similar for 200, 500, and 1000 LRT draws/replications. Table 2 results indicate that a three component model yielded the best fit according to likelihood ratio comparison test. The AIC suggested improvement in fit might be found with a four group model, however both the LRT and BIC did not support a four group model. The consistency of the LRT and AIC fit statistics versus the BIC led us to conclude the evidence favors a three component (group) model. Thus, it appears that three discernible groups are commingled within the overall distribution of paranoid and antisocial PD features as well as aggression within BPD subjects. Mixing proportions for each of the three components were .403, .263, and .333, respectively. The estimate for the correct allocation rate for each component is .94, suggesting accurate placement of cases.

An important assumption in finite mixture modeling with normal components is that the underlying components resolved indeed have relatively normal distributions. The distributions of IPDE antisocial and paranoid PD features as well as IPO-aggression scores were relatively normal fashion within each of the three groups identified by the mixture modeling analysis (all Kolmogorov-Smirnov *Z* tests were nonsignificant).

Individual cases were then assigned to one of the three groups using the posterior probabilities. This parsing yielded: Group-1 = 36 (40%), Group-2 = 25 (27.8%), and Group-3 = 29 (32.2%). This membership provided a basis for group comparison of the subjects on other variables of interest.

TABLE 2. Summary of Finite Mixture Modeling Fits for Antisocial and Paranoid Personality Disorder Features and Aggression Scores in 90 Borderline Personality Disorder Patients

g	Log likelihood	–2 logλ	*P*	AIC	BIC
1	–379.70	—	—	777.39	799.89
2	–358.51	42.37	.01	755.02	802.52
3	–338.59	39.85	.01	735.18	807.67
4	–324.84	27.50	.21	727.68	825.17

Note. g = number of components (groups). –2 logλ = comparison of the log likelihood statistics. AIC = Akaike Information Criterion; BIC = Smaller values for the AIC and BIC indicate better model fit.

First, we examined the levels of paranoid and antisocial PD and aggression values across the three groups in order to determine whether or not the observed groups actually conform to the pattern predicted by Kernberg's (1984; Kernberg & Caligor, 2005) model. The three groups did differ substantially with respect to paranoid PD, antisocial PD, and aggression. The quantitative statement (i.e., focused linear contrasts) of the model predictions are shown as contrast (lambda) weights in the left side of the top panel in Table 3. As seen in the top panel of Table 3, Group-1 contains individuals who showed comparatively low levels of paranoid, antisocial, and aggressive features relative to the other two groups. Whereas Group-2 revealed, as hypothesized, significantly higher levels of paranoid features and comparatively lower levels of antisocial and aggressive features. Finally, Group-3 revealed significantly higher levels of both antisocial and aggressive features. All three linear contrasts were statistically significant and associated with large effect sizes (effect-size *r*'*s* > .37). We, therefore, designated Group-1 as nonaggressive/nonparanoid/nonantisocial, Group-2 as paranoid/nonaggressive/nonantisocial, and Group-3 as aggressive/antisocial/nonparanoid. We emphasize these statistical differences to show that the differences across the 3 groups are not trivial in nature; rather, the phenotypic space has been parsed in a manner that revealed relatively large differences. If the three groups did not differ on these three input variables, one might justifiably wonder if a 3 group parsing was worth the effort and offers any substantive leverage.

TABLE 3. Clinical Features and External Validation Measures in Three Groups of Borderline Personality Disorder Patients (*N* = 90)

Measure	Contrast Weights G1 G2 G3	Group 1 (*n* = 36) *M* (*SD*)	Group 2 (*n* = 25) *M* (*SD*)	Group 3 (*n* = 29) *M* (*SD*)	*t*	*p*	ES r
Clinical Features							
Paranoid PD (*z* IPDE)	1, –2, 1	–.93 (.47)	1.17 (.55)	.15 (.53)	12.90	.0001	.81
Antisocial PD (*z* IPDE)	1, 1, –2	–.57 (.51)	.08 (.93)	.64 (1.12)	4.53	.001	.44
Aggression (*z* IPO)	1, 1, –2	–.24 (.76)	–.49 (.42)	.72 (1.21)	5.51	.001	.51
External Validation Measures							
Social Closeness (MPQ)	1, –2, 1	47.13 (11.06)	37.42 (7.71)	41.29 (10.29)	2.89	.005	.30
Social Function (SAS)	–2, 1, 1	4.56 (1.16)	4.96 (1.10)	5.34 (.86)	2.63	.01	.27
Social Services Usage	1, 1, –2	.86 (1.38)	.76 (1.20)	.83 (1.75)	.24	.81	.03
Sexual Abuse (CTQ)	1, –2, 1	7.83 (5.36)	12.44 (1.60)	9.14 (4.09)	2.85	.005	.29
Physical Abuse (CTQ)	–2, 1, 1	8.19 (3.92)	10.76 (5.34)	10.10 (4.98)	2.21	.03	.23
Impulsivity (Barratt F1)	1, 1, –2	29.67 (6.36)	30.40 (4.44)	32.88 (6.42)	2.13	.04	.22
Constraint (MPQ)	1, 1, –2	161.31 (9.94)	157.16 (11.70)	150.97 (17.71)	2.73	.008	.28
Narcissism (IPDE)	–2, 1, 1	5.75 (4.39)	6.44 (4.47)	7.66 (4.52)	1.35	.18	.14
Psychopathy (PPI)	1, 1, –2	343.42 (31.55)	366.91 (37.21)	388.24 (38.66)	4.07	.001	.40
Negative Emotion (MPQ)	–2, 1, 1	60.51 (9.83)	64.99 (10.41)	66.84 (11.82)	2.35	.02	.24
Identity Diffusion (IPO)	1, 1, –2	65.08 (11.55)	65.68 (13.12)	73.90 (14.11)	2.92	.004	.30

Note. Group 1 = nonparanoid/nonaggressive/nonantisocial; Group 2 = paranoid/nonaggressive/nonantisocial; Group 3 = antisocial/aggressive/nonparanoid. Contrast weights refer to lambda weights for the focused linear contrasts implemented in ANOVA. The *t*-values are reported for the focused contrast. Degrees of freedom for all t = 87, except for the PPI which is 85. The *p*-values are based on a two-tailed test of statistical significance. ES-*r* indicates effect-size *r*. ES *r* = "effect size" *r*, which is interpreted as follows: .10 = small effect, .24 = medium effect, and .37 = large effect (Rosenthal & Rosnow, 1991, p. 446).

Based on Kernberg's (Kernberg & Caligor, 2005) model as well as our extensive experience with BPD, we hypothesized, a priori, that systematic differences would exist among the three BPD groups and we translated these hypotheses into focused linear contrasts. We tested a wide variety of external variables in this analysis. We were agnostic as to the causal versus outcome status of some of these variables in relation to BPD. Our primary interest, rather, was in the presence of reliable differences across the three groups. As such, our search for group differences represents an application of the classic criterion and construct validation approach (Cronbach & Meehl, 1955). We note here that if no differences were to emerge in these analyses, then we would have considerably diminished enthusiasm for the parsing strategy we resolved. Space limitations preclude an extensive development of the theoretical basis for each of these contrast analyses, though all are grounded in theory and extensive clinical observation in our group. The important point, to emphasize, is that these predictions were made prior to the constrast analysis, thus making us articulate specific relationships amongst the three BPD groups on this wide variety of external variables. The focused contrast (lambda) weights for the ANOVA are in the left panel of Table 3. Group-2 (paranoid/nonaggressive/nonantisocial) displayed decreased social closeness ($r_{contrast}$ = .30) and increased levels of self reported childhood sexual abuse ($r_{contrast}$ = .29) relative to Groups-1 (nonaggressive/nonparanoid/nonantisocial) and 3 (aggressive/antisocial/nonparanoid). Group-3 (aggressive/antisocial/nonparanoid) displayed significantly lower levels of constraint ($r_{contrast}$ = .28), higher levels of impulsivity ($r_{contrast}$ = .22), more severe identity diffusion ($r_{contrast}$ = .30), and higher levels of psychopathic personality features ($r_{contrast}$ = .40) relative to Groups-1 (nonaggressive/nonparanoid/nonantisocial) and 2 (paranoid/nonaggressive/nonantisocial). Group-1 (nonaggressive/nonparanoid/nonantisocial) displayed better levels of social/work functioning ($r_{contrast}$ = .27), lower levels of negative emotion ($r_{contrast}$ = .24), and lower rates of self reported childhood physical abuse ($r_{contrast}$ = .23) relative to Groups-2 (paranoid/nonaggressive/nonantisocial) and 3 (aggressive/antisocial/nonparanoid). The contrast analyses for the usage of social services and narcissism were nonsignificant. The effect-sizes for the significant contrasts were typically medium to large in magnitude (Rosenthal & Rosnow, 1991).

DISCUSSION

BPD, which is relatively prevalent (1.4%) (Lenzenweger, Lane et al., 2007), is well represented in clinical samples (Zimmerman, Rothschild, & Chleminski, 2005) and known as a disabling illness associated high treatment utilization (Zanarini, Frankenburg, Hennen, & Silk, 2004). Thus, accurate classification of the disorder is necessary to promote effective research into etiology, pathophysiology, putative endophenotypes, and treatment. However, the disorder is extraordinarily clinically heterogenous, which frus-

trates research efforts. This heterogeneity is not merely a reflection of the *DSM* approach to diagnosis, rather has been known for some time (Stern, 1939; Knight, 1954; Kernberg, 1967; Grinker et al., 1968). We sought to the heterogeneity within the BPD construct through the concurrent use of a theoretical model grounded firmly in clinical observation, diagnostics, and long-term treatment experience (Kernberg, 1984; Kernberg & Caligor, 2005) and finite mixture modeling. The results of our finite mixture modeling analysis support the existence of three relatively distinct types of BPD patients, each type revealing a distinctive clinical profile. The three groups vary as a function of paranoid and antisocial features as well as aggression in a manner consistent with the general theoretical predictions from Kernberg's model of borderline personality organization, not merely as a function of BPD severity. In this context it is also worth noting that having three indicator variables as input to the finite mixture modeling does not ensure more than one group will emerge, nor can one assume a particular grouping structure will emerge based on the content of the input variables. Moreover, the three groups display a pattern of relationships with external correlates of personality, psychosocial functioning, reported childhood trauma, and psychopathology that provide a preliminary basis for validation of the sub-types. Group-1 (nonaggressive/nonparanoid/nonantisocial) BPD patients tend to be characterized by less negative emotion, less childhood physical abuse, and better social/work functioning. Group-2 (paranoid/nonaggressive/nonantisocial) BPD patients are phenotypically paranoid, yet nonaggressive, as well as less affiliative (diminished social closeness) and reporting higher rates of childhood sexual abuse. Finally, Group-3 (aggressive/antisocial/nonparanoid) BPD patients are phenotypically antisocial and aggressive as well as are more dyscontrolled (diminished constraint), impulsive, identity diffused, and psychopathic. It may be that the Group-2 (paranoid/nonaggressive/nonantisocial) BPD patients respond to aggressive affects via a paranoid posture and, thus, do not manifest overt externalizing behavioral aggression as measured by our scale, whereas Group-3 (aggressive/antisocial/nonparanoid) patients manifest a breakdown of behavioral constraints with resulting overt behavioral aggression. In this context we want to emphasize that the BPD groups we discovered were not based on the internal structure of the DSM BPD criteria, but rather on the relations to our three indicator variables—in short, we parsed this group of BPD patients according to three measures selected a priori as consistent with Kernberg's model of phenomenology of BPD. Our model-guided work builds, in our view, upon the prior atheoretical/empirical work that utilized factor and cluster analytic strategies.

Refinement of the BPD phenotype will likely facilitate research efforts in BPD at multiple levels of analysis—genomic, neurobiological, endophenotypic, intervention—through enhanced resolution on the putative underlying psychopathological processes. By this we mean dissecting the BPD phenotype into what may be more homogenous and, arguably, meaningful

sub-types may help to advance research efforts beyond reliance on the coarse DSM-system BPD construct. To our minds, assuming BPD likely reflects an emergent process (Depue & Lenzenweger, 2005) in which several neurobehavioral systems interact configurally with environmental inputs to result in phenotypic evidence of disturbance, it is likely that accurate identification of meaningful clusterings in the phenotypic space will provide a clearer window on signal, rather than noise, in the effort to unravel etiology and pathophysiology. To illuminate underlying biological factors in BPD (and PDs; Depue & Lenzenweger, 2005), one must begin with a unit of analysis that is as clean (i.e., homogenous) as possible—where nature may have been carved at its joints—for laboratory efforts.

Is it possible that other variables used in a finite mixture modeling study such as this might have generated different results (i.e., a different subtyping taxonomy)? Clearly, that is a possibility of which we were quite mindful as we conducted this analysis. In order to investigate this possibility, we examined an extensive taxonomy of other measures as input variables and did not find another latent structure taxonomy for BPD beyond a severity of illness parsing. We note these additional analyses were atheoretical in nature and guided by what might be viewed as reasonable ad hoc combinations of input variables. In short, whenever we parsed the data using mixture modeling based on other input variables (even in the absence of a compelling theoretical rationale), the resulting parsing nearly always yielded two groups and provided minimal clinical differentiation among the BPD patients beyond the degree of severity. While severity may serve as the basis for some reasonable differentiation among patients for some research projects, in our view, it does not aid in resolving latent structure in a way that will facilitate research advances. By way of comparison, note how the positive versus negative symptom distinction in schizophrenia has helped to advance research beyond schemes reliant on severity of illness. Finally, increasing the number of predictor variables beyond three, yielded little in terms of resolving power.

We are mindful of several limitations of our study. First, our sample consists largely of women. Thus, although sex differences in the rate of BPD do not exist in the general population (Lenzenweger, Lane et al., 2007), many women are diagnosed with the disorder in clinical settings (American Psychiatric Association, 1994). Therefore, future replication work on our proposed taxonomy should consider constructing samples with equal proportions of males and females, perhaps drawn from the general population. Secondly, our sample was diagnosed according to the DSM-IV definition of BPD, therefore our findings are necessarily conditioned on the presence of DSM-IV BPD and are not necessarily directly applicable to other definitions of BPD or unselected PD samples. Furthermore, we stress that we did not analyze the BPD DSM diagnostic criteria, rather we parsed a sample of BPD patients using a priori selected clinical measures. We also note, in this context, that our 3-group parsing really only relates to BPD. Our study did not address whether these three variables might be useful in dissecting other groups of psychopathology (e.g.,

panic disorder, eating disorder, depression). Finally, we emphasize that our taxonomy, which is theory-based and supported by our finite mixture analyses, may not be the only viable taxonomy for BPD and thus we offer it as a starting point in efforts to refine the BPD phenotype in the search for etiology, endophenotypes (Gottesman & Gould, 2003; Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002; Lenzenweger & Pastore, 2007), and effective treatments (Clarkin et al., 2007) for the disorder. We also readily acknowledge there are dimensional alternatives to the categorical sub-typing approach we pursued (e.g., Cloninger et al., 1993; Depue & Lenzenweger, 2001, 2005; Livesley et al., 1998).

We offer this three sub-group taxonomy for BPD that integrates paranoid and antisocial features as well as aggression as a heuristic. We also advocate the use of finite mixture modeling as a statistically well-principled method for the illumination of latent organization in continuous data. This statistical approach holds considerable value as an objective means for parsing observed heterogeneity in multiple phenotypic indicators in BPD research (cf., Lenzenweger et al., 2003; Lenzenweger, McLachlan et al., 2007). Effective parsing of the phenotypic space associated with BPD may yield meaningful subgroups and these classifications then may aid greatly, efforts seeking to link specific polymorphisms to behavioral or neurocognitive endophenotypes in genomic research (cf., schizophrenia research). Finally, as noted by Lenzenweger, McLachlan et al. (2007) any statistical approach to understanding the latent structure of data will necessarily reveal only part of the story, and cannot conclusively resolve a substantive issue. We believe the substantive discussion regarding the fundamental nature of the latent structure of schizophrenia liability will be informed not only by statistical methods and results such as ours, but by reference to other data from other levels of analysis as well. Thus, we see theory, method, and inquiry at many levels of analysis facilitating our understanding of BPD.

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