A Taxometric Analysis of the Latent Structure of Psychopathy: Evidence for Dimensionality

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The taxonomic status of psychopathy is controversial. Whereas some studies have found evidence that psychopathy, at least its antisocial component, is distributed as a taxon, others have found that both major components of psychopathy—callousness/unemotionality and impulsivity/antisocial behavior—appear to distribute as dimensions and show little evidence of taxonicity. In the present study, recent advances in taxometric analysis were added to P. Meehl's (1995) multiple consistency tests strategy for assessing taxonicity, and they were applied to Psychopathy Checklist—Revised (R. D. Hare, 2003) ratings of 4,865 offenders sampled from multiple forensic settings. The results indicated that both the individual components of psychopathy and their interface are distributed dimensionally. Both the implications of these results for research in psychopathy and the integration of these findings with previous taxometric studies of psychopathy are discussed.

Keywords: psychopathy, taxometrics, latent structure, antisocial behavior

Psychopathy is a personality disorder that comprises a distinct cluster of emotional, interpersonal, and behavioral characteristics (e.g., emotional detachment, callousness, irresponsibility, impulsivity) and one that is characterized by a disregard for the societal rules and the rights of others (Hare, 1996). Its association with violence (Porter & Woodworth, 2006) and its usefulness as a risk factor in predicting criminal recidivism (Douglas, Vincent, & Edens, 2006) have increased its prominence in the last decade in both criminology and psychopathology. The origins of our current conceptualization of the construct can be traced to Cleckley's (1976) classic description of the syndrome. He delineated its characteristics, however, without addressing the issue of whether it represents the coalescence of extreme manifestations on a number of dimensional traits or constitutes a *taxon* (i.e., a distinct, nonarbitrary entity or class).

The first formalized assessment tool for measuring psychopathy was Hare's (1980) operationalization of the construct in the Psychopathy Checklist (PCL) and its revision, the PCL–R (Hare, 1991, 2003). Although Hare conceptualized the PCL as a way of indicating how closely an individual approximated the "prototypic psychopath" and proposed a PCL–R cutting score of 30 (out of 40) to consider a person sufficiently close to the psychopath prototype, he also recognized that viable arguments could be made for using the PCL–R to obtain dimensional scores.

It behooves the researcher who postulates the existence of a taxon in a particular domain and undertakes taxometrics in pursuit of that taxon to provide adequate theoretical justification for the search (Lenzenweger, 2004). Taxonicity implies both a nonarbitrary latent category and a particular causal structure. The most widely accepted theoretical understanding of taxonicity and the strongest examples of taxa have focused on entities specified by the conjunction of a distinct pathology and etiology (Meehl, 1973, 1992). Even though evidence for specific genetic, biological, and environmental correlates of a syndrome might ultimately be accounted for adequately either by dimensional or taxonic models, the absence of any such etiological evidence should preclude the search for a taxon. Consequently, the theoretical case for hypothesizing the possibility that psychopathy may be distributed as a taxon revolves around evidence for specific genetic, neurobiological, cognitive, and affective covariates of the construct that are consistent with the hypothesis that a specific etiology or specific etiologies may account for it. This evidence makes plausible the search for taxonicity, but does not confirm the existence of a taxon, and is not inconsistent with dimensional explanations.

Two- (Harpur, Hakstian, & Hare, 1988; Harpur, Hare, & Hakstian, 1989), three- (Cooke & Michie, 2001), and four-factor (Hare, 2003; Hare & Neumann, 2006) models of the PCL have all identified two overarching components involving impulsivity–antisocial behavior and affective–interpersonal features. Because

these two components have consistently yielded distinct patterns of correlations with characteristics relevant to psychopathy, different models of specific underlying processes have been proposed for each (e.g., Bloningen, Hicks, Krueger, Patrick, & Iacono, 2005; Fowles & Dindo, 2006; Patrick & Zempolich, 1998). Such models raise the possibility that either or both factors may be distributed as taxa. A brief survey of some of the correlative evidence in the genetic, neurobiological, cognitive, and affective domains provides an adequate justification to support a taxonomic investigation.

There is considerable evidence from adoption and twin genetic studies, including those of twins reared apart, indicating that genetic factors play a significant role in the likelihood that a person will commit a criminal act (Gottesman & Goldsmith, 1994; Grove et al., 1990; Mednick, Gabrielli, & Hutchings, 1984) and also increase the probability that an individual will be diagnosed with antisocial personality disorder (APD) during his or her lifetime (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995; Ge et al., 1996; Lyons et al., 1995). Although behavioral genetic research suggests that the Factor 2 features may have higher heritability than those of Factor 1 (Depue, 1996; Edelbrock, Rende, Plomin, & Thompson, 1995; Krueger, 2000; Mason & Frick, 1994; but see Livesley, 1998, for contrary evidence), there are also data supporting the importance of independent genetic influences in the manifestation of Factor 1 (Patrick, 2003; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003).

A number of neurobiological deficits or anomalies have been identified in both psychopathic criminals and "successful" psychopaths, who have largely avoided contact with the criminal justice system or extended incarceration. For instance, both specific neurological structural features in the amygdala, orbito-frontal cortex, and hippocampus (e.g., Blair, 2004; Mitchell, Colledge, Leonard, & Blair, 2002; Raine, 2001; Raine et al., 2004; Tiihonen et al., 2000) and functional anomalies in the amygdala/hippocampal formation, parahippocampal gyrus, ventral striatum, anterior and posterior cingulated gyri, and fronto-temporal cortex (e.g., Kiehl, Hare, Liddle, & McDonald, 1999; Kiehl et al., 2001; Kiehl et al., 2004; Müller et al., 2003; Völlm et al., 2004) have been linked to the syndrome or its components. Some speculations have been proposed about a comprehensive and integrated mapping of these deficits onto psychopathy (Blair, Mitchell, & Blair, 2005) and onto the two major PCL–R factors by which they are assessed (Patrick, 2003). An understanding of the latent distribution of the PCL–R and its factors could substantially advance the search for such an integration.

Psychopathic individuals have been found to be deficient on a number of cognitive (e.g., Hervé, Hayes, & Hare, 2003; Morgan & Lilienfeld, 2000; Newman & Lorenz, 2003) and affective tasks (e.g., Blair, 2001; Blair et al., 2002; Patrick, 2001; Williamson, Harpur, & Hare, 1991) that map onto the structural features noted above. Psychopaths' difficulty in shifting a dominant behavior when contingencies have been reversed (Newman & Lorenz, 2003) and their difficulties with working memory and other aspects of executive functioning (Morgan & Lilienfeld, 2000; Séguin, 2004) implicate deficits in their orbito-frontal cortex. In contrast, psychopaths' impairments in passive avoidance learning (Newman & Kosson, 1986), dysfunctional response to another's sadness or fear (Blair, 1995), reduced augmentation of the startle reflex by threat primes (Levenston, Patrick, Bradley, & Lang, 2000), impaired aversive conditioning (Raine, Venables, & Wil-

liams, 1996), and deficient processing of fearful expressions (Blair, Colledge, Murray, & Mitchell, 2001) are perhaps better accounted for at the neural level by deficiencies in the functions of the amygdala (Blair, 2004) or in the integration of frontal-limbic processes (Hare, 2003; Müller et al., 2003). Although speculative models to integrate these deficits have been proposed (e.g., Blair et al., 2005; Fowles & Dindo, 2006), unpacking the complexity of the development of the underlying core processes (Séguin, 2004) and mapping onto the specific behavioral patterns in psychopathy have remained elusive.

Such correlations with specific genetic, neurobiological, cognitive, and affective processes suggest the possibility that specific neurobiological deficiencies may be necessary antecedents of psychopathy. Although dimensional models may be proposed to account for such causes (e.g., Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Miller, Lynam, Widiger, & Leukefeld, 2001), a taxonic distribution is a viable theoretical possibility that must be investigated. Resolution of this issue of the latent structure of psychopathy is not only important for developing theoretical models of the etiology and course of psychopathy, but also it has critical implications for determining optimal investigative strategies and for specifying the ideal psychometric qualities of scales constructed both for clinical and dispositional decision making (Krueger, 1999; Meehl, 1992; Ruscio & Ruscio, 2004a). Because of the prominence of PCL–R ratings in risk assessment instruments (e.g., Quinsey, Harris, Rice, & Cormier, 1998) and its widespread use in legal dispositional decisions (Edens & Petrila, 2006), such scaling issues have substantial practical import. Consequently, a number of studies have addressed the problem of psychopathy's latent distribution (Edens, Marcus, Lilienfeld, & Poythress, 2006; Guay & Knight, 2003; Harris, Rice, & Quinsey, 1994; Marcus, John, & Edens, 2004; Skilling, Quinsey, & Craig, 2001; Vasey, Kotov, Frick, & Loney, 2005) but with mixed results. Many of these prior studies have unfortunately suffered from sampling and methodological weaknesses that have limited their ability to provide a definitive answer to this question.

Both of the studies that found no evidence for taxonic latent structures analyzed self-report data. Guay and Knight (2003) conducted a taxometric investigation of the components of psychopathy with the Multidimensional Assessment of Sex and Aggression (MASA; Knight & Cerce, 1999). The MASA is a self-report inventory that covers multiple domains (childhood experiences, family and social relationships, school and work experiences, alcohol and drug use, and sexual and aggressive behavior and fantasies) and was developed to supplement archival records. Participants were 330 sex offenders, 155 generic criminals, and 93 community controls, who had been tested on paper-and-pencil and computerized versions of the MASA. A total of 11 scales measuring conning and superficial charm, emotional detachment and behavioral problems, and impulse control were generated with factor analysis in combination with a Rasch model. The various taxometric techniques (mean above minus below a cut [MAMBAC; Meehl & Yonce, 1994], maximum covariance analysis [MAXCOV; Meehl, 1973], maximum eigenvalue [MAXEIG; Waller & Meehl, 1998]) that were assessed all generated results consistent with a dimensional structure, with no evidence of taxonicity. Marcus, John, and Edens (2004) evaluated a sample of 309 incarcerated offenders (51.6% African American, 37.5% Caucasian, 8% Hispanic, 2.9% other, and 91.3% male) with the Psychopathic Per-

sonality Inventory (PPI; Lilienfeld & Andrews, 1996). The authors performed MAMBAC, MAXEIG, and latent mode (L-Mode; Waller & Meehl, 1998) analyses, and their results showed no evidence of taxonicity. Both studies may have compromised their ability to identify a taxon because of the potential for increased nuisance covariation in self-report data that is produced by response styles and biases. Malingering and positive impression management, dishonesty, lack of insight, semantic aphasia, and negative emotionality are common when studying psychopathy with self-report instruments (Lilienfeld & Fowler, 2006), such as the MASA or the PPI, along with other aspects related to method variance could have artifactually increased the correlation of scales within the taxon, and the complement and thereby reduced the potential to identify a taxon.

Harris et al. (1994) analyzed data collected from 653 mentally disordered participants from a maximum-security institution, who had been adjudicated not guilty by reason of insanity. Although the authors argued that their results provided evidence for a taxon, several methodological ambiguities of their study undermine their conclusion. First, their sample comprised a select, potentially biased group of offenders (Marcus et al., 2004). Second, with the exception of their application of Meehl's (1995) taxometric analyses, most of the statistical procedures used by Harris et al. (1994) lacked empirical support as methods for distinguishing between taxonic and dimensional structures. The validity of their iterative methods approach has not been tested by any Monte Carlo studies, and the distributions of Bayesian probabilities can readily be U-shaped, even when the latent structure is dimensional, especially when a large number of items are used as in the Harris et al. study. Third, their exclusive use of file reviews introduced severe limitations. The data for accurately rating Factor 1 items in archival files is often missing (Hare, 2003). Moreover, insufficient file information can lead raters to score items for which data are missing or inadequate from information on related items (Alpert, Shaw, Pouget, & Lim, 2002), thereby decreasing item covariation within the putative taxon, increasing item correlation across the taxon and complement, and increasing the potential to find a pseudo-taxon. File reports often focus on salient, egregious Factor 2 antisocial behaviors that may make ratings more vulnerable to raters' a priori taxonic (Beauchaine & Waters, 2003) or item contingency (Bolt, Hare, Vitale, & Newman, 2004) biases and may thereby increase the probability of pseudo-taxonicity. In this regard, it is interesting that Harris et al. found evidence for a taxon only in their Factor 2 analyses. Fourth, the authors used an admixed sample in which they used some matching procedures. The alleged taxon might have been an artifact of their sampling strategy. This possibility could have been avoided by rerunning their analyses within samples, or they could have presented a contingency table of sample membership and taxon/complement assignment. Fifth, Harris et al. did not have access to the simulation programs used in the present study, which generate taxonic and dimensional comparison data to analyze as an interpretive aid. Without this, they did not notice the limited range and low values of the covariances that their MAXCOV analyses generated. Consequently, they may not have scaled the ordinate of their graphs appropriately for the interpretation of the curves calculated from their data. A more extended ordinate makes their apparently peaked curve look flat and dimensional.

Later, Skilling et al. (2001) replicated Harris et al.'s (1994) results, with a similar methodology, but this time by analyzing a sample of 1,111 boys. At first sight, their results appear to support the taxonic structure of psychopathy, but, once again, methodological problems plague the study. First, the authors unnecessarily dichotomized their items for MAXCOV, which weakens this procedure (Ruscio, 2000). Second, the authors used the goodness-of-fit index (GFI) to determine the nature of the latent structure. In the two studies that have examined the GFI systematically (Cleland, Rothschild, & Haslam, 2000; Haslam & Cleland, 2002), it has been shown to discriminate poorly between taxonic and dimensional structure. Moreover, because both taxonic and dimensional structures can yield GFIs well above .90, no universally applicable threshold has emerged, even for data that GFI handles well. Examining the consistency of base-rate estimates has intuitive appeal and is widely recommended and practiced, but nobody has ever actually established that taxonic structure does in fact yield more consistent estimates across a realistic range of data parameters. A recent factorial Monte Carlo study (Ruscio, 2006) found that MAMBAC, MAXCOV, and MAXEIG analyses seldom yielded lower standard deviations for taxonic structure than for dimensional structure. More often than not, they were basically the same. Ambiguous MAMBAC and MAXCOV results may be produced by a positive skew of indicators or by low endorsement rates in the case of binary indicators. Rising MAMBAC curves and apparent (but low) peaks toward the right side of MAXCOV curves, highly consistent base-rate estimates, and a high GFI are all as consistent with a latent dimension whose indicators are positively skewed (low endorsement) as they are with a small taxon.

Skilling, Harris, Rice, and Quinsey (2002) used the same participants as the Harris et al. (1994) study to investigate the taxonic structure of APD, PCL–R, and the Child and Adolescent Taxon Scale (CATS), a derived instrument based on eight items associated with the taxon in the Harris et al. article. The authors performed MAMBAC and MAXCOV analyses and concluded that there was evidence supporting a taxon both for APD and for CATS. They failed to report the amplitude of the mean differences in MAMBAC and the covariance scores in MAXCOV. Other problems identified in the Harris et al. article, such as possible rater file review and graph comparison biases, absence of taxon base-rate estimates, the potentially misleading admixed sample, and limited ordinate values also apply to the Skilling et al. study.

Vasey, Kotov, Frick, and Loney (2005) studied a sample of 386 children and adolescents to test the latent structure of psychopathy. The authors used two versions (parent and student) of the Antisocial Process Screening Device (APSD; Frick & Hare, 2001) to assess psychopathic characteristics. Along with evidence of a taxon for broad antisocial behavior, the authors claimed that they had found evidence for a psychopathy taxon. Specifically, with MAXEIG on the five subscales of youth self-report and parent-report APSD, the results produced graphs consistent with a taxonic structure with an average base rate of .08. Their L-Mode analyses also suggested taxonicity with a base rate of .04. Unfortunately, sample composition may easily explain the presence of the alleged taxon. In order to "increase the chances of detecting a psychopathy taxon (should one exist)," the authors Vasey et al. (2005, p. 420) added an extra 60 juvenile offenders to an already heterogeneous

sample of 283 children and adolescents (boys and girls) recruited from middle schools and 43 referred boys with severe emotional, behavioral, or learning problems. Vasey et al.'s two studies of children and adolescents were also limited by their lack of comparison curves for taxonic and dimensional data to help interpret their results. Some of their interpretations of curves as indicating taxonicity are questionable. Moreover, their strongest results came from data in which they combined separate community and clinical samples, increasing the potential for identifying pseudo-taxa (Ruscio & Ruscio, 2004a). As the authors indicated, their best evidence identifies a taxon with a base rate that is too broad for psychopathy and that may be more accurately interpreted as an externalizing syndrome.

Recently, Edens et al. (2006) investigated the latent structure of psychopathy with the PCL–R in a sample of 876 male prison inmates and individuals who were court ordered into residential drug treatment programs (59% Caucasian, 31% African American, 10% Hispanic). The authors used a double strategy in which they conducted taxometric analyses on Hare's (2003) four-factor solution and used the same combination of items (as well as the eight items that correlated most highly with the PCL–R total score in their sample) that had been used by Harris et al. (1994) to produce apparently taxonic results. Their results provided no evidence of taxonicity.

The four studies that profess to offer support for the hypothesis that the latent structure of psychopathy is taxonic do not provide convincing data to support this claim. Consequently, we conducted the present study in which Meehl's (1995) taxometric method was applied to PCL–R ratings of 4,865 offenders sampled from multiple prison settings and that was assessed by both interviews and archival file review. In addition to a large representative sample, the putative psychopathy taxon was sufficiently large to afford powerful taxometric analyses, including the generation of simulated taxonic and dimensional data with which to compare the research data results. Of the sample in the present study, 19% ($n = 927$) had a score of 30 or higher. The present sample also permitted the construction and analysis of several indicator sets with which to assess the latent structure of psychopathy and permitted detailed analyses of subsamples to examine and eliminate potential sampling biases. The sample was subdivided by security level and ethnic status, and separate subsample analyses were computed. Hare's (2003) four-factor solution to the PCL–R was used as the basis of the primary analyses, with composite scores for each factor serving as indicators. Additional analyses were performed with items within each of these four factors—as well as the affective–interpersonal features and impulsivity–antisocial behavior factors—as indicators to assess the taxonicity of each. Thus, the present study addressed the methodological shortcomings of prior studies and allowed a convincing test of the taxonicity of psychopathy and its components.

Method

Participants

The initial sample of participants consisted of 5,408 male prison inmates incarcerated in North American institutions. Data were collected from 15 different samples across Canada (British Columbia, Ontario, and Québec) and the United States (Wisconsin and North Carolina).

Sample 1 comprised 322 male inmates of a federal medium security institution in British Columbia, all serving sentences of 2 years or more. They had volunteered to take part in several research projects. Sample 2 was composed of 121 male inmates of a provincial prison in British Columbia, all serving sentences of less than 2 years. Sample 3 contained 369 male inmates of a minimum security institution in Wisconsin (Kosson, Smith, & Newman, 1990). In Sample 4, 106 male inmates of the Institute Phillipe-Pinel de Montréal in the province of Québec were evaluated with a French version of the PCL–R (Côté & Hodgins, 1991) just prior to conditional release from minimum, medium, or maximum security federal prisons. Sample 5 was composed of 87 inmates of a medium security prison in Kingston, Ontario (Serin, 1991). Sample 6 was composed of 152 African American male inmates (Kosson, Smith, & Newman, 1990). Sample 7 included 60 male inmates from a medium security prison in North Carolina (Johnson, 1990). In Sample 8, 197 male inmates from a federal medium security prison in British Columbia were evaluated with the PCL–R. Sample 9 included 172 male inmates from a federal forensic psychiatric institution in British Columbia. Most were participants in a violent offender treatment program. Participants from Sample 10 were 1,190 male inmates (526 Caucasian and 664 African American) from a state medium security prison in Wisconsin. In Sample 11, 320 male inmates (227 Caucasian, and 73 Native American; 220 sex offenders) from a federal medium security prison in British Columbia that houses violent offenders were interviewed (Porter et al., 2000). Sample 12 was composed of 60 male inmates (rapists) in federal medium and maximum security institutions in Ontario (Brown & Forth, 1997). Sample 13 consisted of 185 male inmates of a medium security institution in British Columbia. Sample 14 was composed of 427 male violent offenders in a federal medium security institution in Ontario (Simourd & Hoge, 2000). Finally, Sample 15 was composed of a representative sample of 1,640 male offenders, admitted to a regional reception and assessment center of the Correctional Service of Canada.

The average age of the 2,300 offenders for whom data were available was 31.1 ($SD = 9.3$; range: 17–81). In general, the reliability of the PCL–R ratings was quite acceptable. Cronbach's alpha was .85, and the intraclass correlation was .86 for a single rating and .92 for the average of two ratings. Intraclass correlation coefficients (ICC) for single ratings (ICC_1) on Factor 1 and Factor 2 of the PCL–R were respectively .75 and .85, and for averaged ratings (ICC_2) they were .85 and .92. For the four facets (Hare, 2003), coefficients ranged from .67 to .84 for single ratings and from .80 to .91 for averaged ratings. To qualify as a PCL–R rater, individuals had to receive proper training in the use of the instrument. For a more detailed description of the sample composition and the rating procedures, see Hare (2003). Participants with missing information on any one of the items ($n = 543$) were excluded, and analyses for the present study were performed on the 4,865 participants with complete PCL–R protocols.

Measures

Psychopathy was assessed with the PCL–R (Hare, 1991). All participants were assessed with the semistructured interview and file information. The PCL–R is a 20-item clinical rating scale that measures interpersonal, affective, and socially deviant features of psychopathy. The mean score on the PCL–R for the entire sample was 21.9 ($SD = 7.9$).

Taxometric Analyses

Two taxometric procedures were performed on each indicator set: MAMBAC and MAXEIG. A third procedure, L-Mode, was performed only on the indicators in the primary analyses, which possessed sufficient variation to render a factor analysis meaningful. These three procedures are based on independent mathematical derivations and therefore can contribute nonredundant evidence of latent structure. Below, we note how MAMBAC and MAXEIG analyses were conducted, both of which involve a number of choice points in their implementation. For detailed descriptions of the logic and the mathematical underpinnings of all three procedures, see the primary publications cited above; on the available options for implementing each procedure and suggestions for making informed choices, see Ruscio and Ruscio (2004b, 2004c).

MAMBAC was performed with composite input indicators (i.e., by selecting one of the k variables to serve as the output indicator and summing the remaining $k - 1$ variables to serve as the input indicator for each of k analyses). To accommodate the constrained indicator response scales, 50 equally spaced cutting scores beginning and ending with at least 25 cases from each end of the input indicator (larger values were used as necessary to stabilize the shapes near the ends of the curves) and 10 internal replications in the calculation of each curve (to reduce the obfuscating influence of cutting between equal-scoring cases, as described in Ruscio, Haslam, & Ruscio, 2006) were used. MAMBAC curves were not smoothed, and full panels of curves for each indicator set were examined to determine whether an averaged curve adequately represented the overall trend. Averaged curves are presented here to conserve space. Panels are available from Jean-Pierre Guay upon request.

MAXEIG was performed with composite input indicators (in the same way, and for the same reason, as in MAMBAC), except for the primary series of analyses. In that instance, there was sufficient response variation to perform MAXEIG in the more traditional manner (i.e., by selecting one of the k variables to serve as the input indicator and by using the remaining $k - 1$ variables as output indicators for each of the k analyses). Each analysis used 50 windows that overlapped 90% with adjacent subsamples and 10 internal replications. Once again, smoothing was not performed, and full panels of curves were examined to ensure that the averaged curve fairly represented the overall pattern of results.

Analyses of Comparison Data

As a supplement to the inspection of taxometric results that can aid interpretation in ambiguous circumstances, bootstrap methods were used. This involves generating samples of taxonic and dimensional comparison data that reproduce the sample size, indicator distributions, and indicator correlations in a sample of research data and then submitting each bootstrap sample to the same taxometric analyses as the research data. Comparison data were generated with an updated version of the algorithm outlined in Ruscio and Ruscio (2004a) and in Ruscio, Ruscio, and Keane (2004) that implements two new features. First, comparison data are allowed to be multidimensional rather than constrained to unidimensionality (in the full sample for dimensional comparison data, within groups for taxonic comparison data). Second, indica-

tor distributions are generated by using a standard bootstrap technique rather than by being copied from the research data. The basic bootstrap approach of resampling, with replacement, treats a sample distribution as an unbiased estimate of the population distribution and draws new samples accordingly. Specifically, N scores for each indicator in a bootstrap sample of comparison data were drawn at random (with replacement) from the original score distribution. Programs including these features have been shown to reproduce indicator distributions and correlations with good precision and negligible bias (Ruscio, Ruscio, & Meron, in press). Simulating taxonic comparison data requires a criterion variable that contains classification codes for each case. The PCL–R cutoff score of 30 that has been typically used for the diagnosis of psychopathy in research studies was used for all analyses. This cutoff was originally generated to maximize the overall hit rate of PCL–R for global judgments of psychopathy (cf. Hare, 2003). It has been used in hundreds of laboratory and applied research studies, and its utility has been supported by its generation and validation of many of the processing and neurological differences discussed in the introductory paragraphs. There are also item response theory data that suggest that the score of 30 indicates the same level of psychopathy across North American male offenders, English male offenders, female offenders, and male forensic psychiatric patients (Hare & Neumann, 2006). Consequently, it constitutes a reasonable cutoff for our taxonomic analyses. Because there might be some differences in offenders whose PCL–Rs were scored only from file reviews and those scored from a combination of file and interview assessments (Hare, 2003), we included in our study only protocols that were rated with both informational sources. We assigned the 927 cases with PCL–R total scores at or exceeding 30 to the putative taxon. We assigned all others to the putative complement. We generated 10 samples of taxonic and 10 samples of dimensional comparison data for each analysis.

An examination of the results for taxonic and dimensional comparison data helps to ensure that apparently dimensional results do not stem from too small a sample size, inadequate representation of putative taxon members, insufficiently valid indicators, problematically high levels of nuisance covariance, or other unacceptable aspects of the data. The extent to which the results for the research data are better reproduced by those within the sampling distribution yielded by analyses of taxonic or dimensional comparison data sets supports the validity of the corresponding structural inference.

As an objective adjunct to the interpretation of curve shapes, we calculated a quantitative index. The fit of the averaged curve for the research data to the averaged curves in the sampling distributions of taxonic and dimensional comparison data sets was assessed by using an approach introduced by Ruscio and Ruscio (2004b) and that was refined by Ruscio et al. (in press). First, fit is calculated for the comparison data generated to represent each structure:

$$Fit_{\text{RMSR}} = \sqrt{\frac{\Sigma (y_{res,data} - y_{sim,data})^2}{N}}, \qquad (1)$$

where $y_{res.data}$ refers to a data point on the curve for the research data, $y_{sim.data}$ refers to the corresponding data point on the curve for comparison data, and N is the number of data points on each curve. Lower values of Fit_{RMSR} reflect better fit, with perfect fit

Table 1
Psychopathy Checklist—Revised (PCL–R) Descriptive Statistics for Subsamples

Indicator set	All subjects		Causian only		African American only		Min. security		Med. security		Max. security		Federal sample	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Summative	21.9	7.9	23.2	7.6	22.8	7.3	23.9	7.9	23.5	7.2	22.6	7.4	19.3	8.2
Affective	3.6	2.1	3.7	2.2	4.1	2.0	4.0	2.4	3.6	2.1	3.6	2.3	3.1	2.2
Antisocial	4.8	2.1	4.9	2.1	4.9	2.0	5.1	2.1	4.8	2.0	4.7	2.0	4.4	2.2
Interpersonal	5.9	2.5	6.4	2.3	5.8	2.2	6.9	2.3	6.4	2.4	6.1	2.4	5.1	2.9
Lifestyle	5.8	2.8	6.1	2.6	5.9	2.6	5.8	2.6	6.3	2.5	6.5	2.6	5.0	3.0
PCL–R Factor 1	8.4	3.8	8.7	3.8	9.0	3.4	9.2	4.0	8.8	3.6	8.3	3.8	7.5	3.9
PCL–R Factor 2	11.7	4.7	12.5	4.2	11.7	4.0	12.7	4.2	12.7	4.3	12.6	4.3	10.1	5.4

Note. Min. = minimum; Med. = medium; Max. = maximum.

represented by a value of 0. Equation 1 is calculated twice, once to assess the fit of the research curves to those for taxonic comparison data ($Fit_{RMSR\text{-}tax}$) and once to assess the fit for dimensional comparison data ($Fit_{RMSR\text{-}dim}$). Then, these two values are integrated into a single comparison curve fit index (CCFI):

$$\text{CCFI} = \frac{Fit_{RMSR\text{-}dim}}{Fit_{RMSR\text{-}dim} + Fit_{RMSR\text{-}tax}}. \qquad (2)$$

CCFI values can range from 0 to 1, with lower values suggesting better fit for dimensional structure and higher values suggesting better fit for taxonic structure. The index is symmetrical about .50 in that this middle value represents equivalent fit for both structures. It is important to note that the CCFI indexes the relative fit of taxonic and dimensional structural models, not the absolute goodness of fit of either model. In a preliminary test of this type of curve fit index (Ruscio, 2004), latent structure was correctly classified with high precision in analyses of the 700 Meehl and Yonce (1994) samples of taxonic and dimensional data. In a Monte Carlo study that included a much broader range of data conditions (Ruscio et al., in press), this index significantly outperformed several of the most popular taxometric consistency tests.

Results

In the first series of analyses, composite indicators were formed in accordance with Hare's (2003) four-factor model; items assigned to each factor (referred to here as *facets*) were summed to yield one indicator a piece.[1] These analyses are primary in the sense that the structure of psychopathy was represented by all four facets: interpersonal, affective, lifestyle, and antisocial. Descriptive statistics for both various subsamples used in the analyses and the entire sample are presented in Table 1. The results for MAMBAC, MAXEIG, and L-Mode analyses of the summative indicators appear in Figure 1. These analyses yielded quite clear and consistent results. Curve shapes were highly consistent with what one would expect for dimensional structure and were quite similar to the curves generated for simulated dimensional comparison data. This same conclusion was also reflected in the CCFI values, which strongly favor a dimensional interpretation (see Table 2 for all CCFI results). Although there was a modest level of consistency among the MAMBAC

base-rate estimates, as well as among the MAXEIG estimates, this was not true of the L-Mode estimates (see Table 3 for summaries of all taxon base-rate estimates). In addition to the notable discrepancies across procedures' estimates, the results were much better reproduced by the dimensional than by the taxonic comparison data. Thus, the totality of evidence suggests that these four theoretically derived composite indicators of psychopathy do not represent a taxonic construct. Rather, individual differences appear more consistent with dimensional structure.

The second series of taxometric analyses was performed to test the latent structure of each of the four theoretical facets of psychopathy. The results for MAMBAC and MAXEIG analyses of indicator sets representing each facet (affective: four indicators; antisocial: five indicators; interpersonal: four indicators; and lifestyle: five indicators) appear in Figure 2. In analyses of the research data, no taxonic peaks emerged, and CCFI values supported—in seven out of eight analyses—an inference of dimensional structure for each facet. Taxon base-rate estimates varied substantially within analyses, and they were generally better reproduced by the dimensional than by the taxonic comparison data. Thus, although the results are not as prototypically dimensional in appearance as in the first series of analyses, no evidence in support of a taxon was obtained for any of the four theoretical facets.

The final series of taxometric analyses examined the latent structure of PCL–R Factors 1 and 2 by using separate sets of indicators to represent each. The results for MAMBAC and MAXEIG analyses of indicator sets representing PCL–R Factors 1 (8 indicators) and 2 (10 indicators) appear in Figure 3. None of the curves for the research data contain the peaks that would be expected of taxonic data, an interpretation supported by CCFI values in three out of four analyses (the exception was an ambiguous value supportive of neither structure). Moreover, taxon base-rate estimates were inconsistent within and across procedures. Results for comparison data suggest that either latent structure could have given rise to such estimates. Hence they are not particularly informative in these analyses. To the extent that the

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[1] A series of preliminary analyses with MAMBAC were performed on all possible combinations of indicators. None of the curves appeared taxonic. These curves are available from Jean-Pierre Guay on request.

MAMBAC
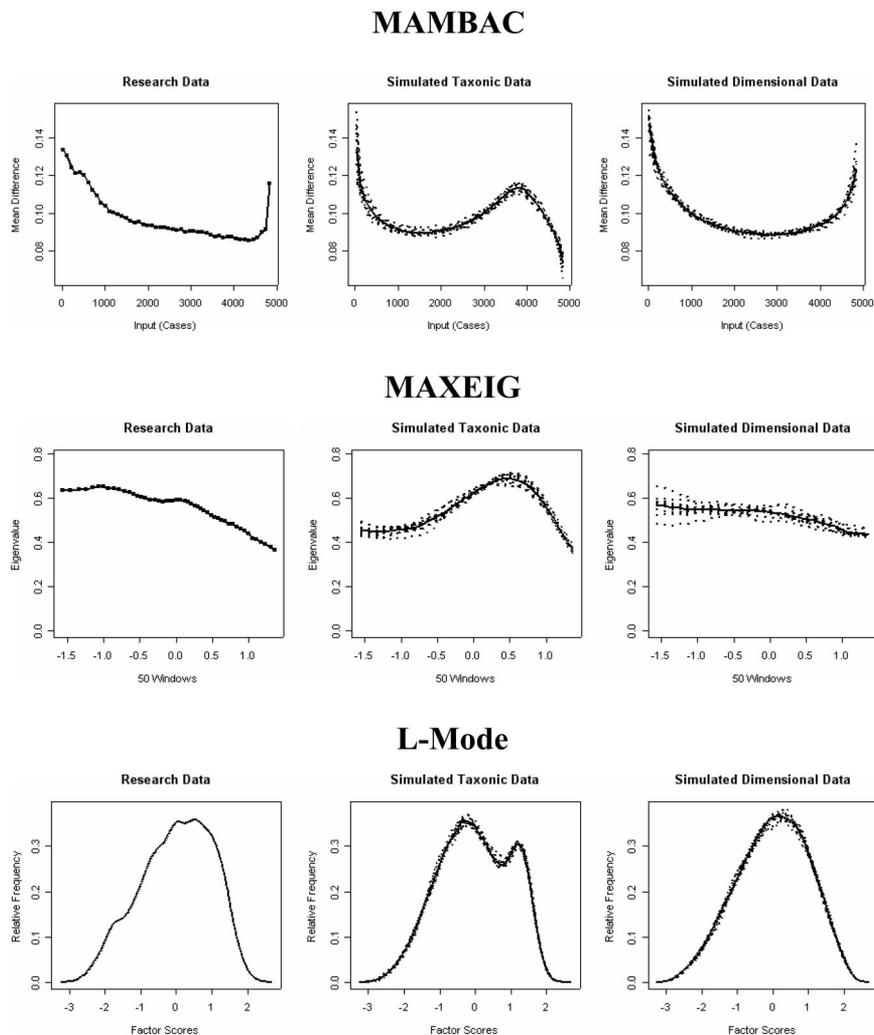
MAXEIG

L-Mode

Figure 1. Mean above minus below a cut (MAMBAC), maximum eigenvalue (MAXEIG), and latent mode (L-Mode) results for the summative indicators. Within each three-panel graph, the first panel shows the averaged curve for the research data, and the second and third panels show the averaged curves for the simulated taxonic and dimensional comparison data. For comparison data, each sample's results are represented by a dotted line, with the average across all samples plotted as a solid line.

results for these two factors hint at latent structure, they suggest a dimensional interpretation. In any event, there is no evidence that either the Affective/Interpersonal (Factor 1) or the Lifestyle/Antisocialty (Factor 2) features of the PCL–R factor are distributed as a taxon.

Follow-up analyses were performed for each indicator set within subsamples consisting of (a) inmates in maximum security prisons, (b) inmates in medium security prisons, (c) inmates in minimum security prisons, (d) African American inmates, (e) Caucasian inmates, and (f) inmates from a representative sample of subjects incarcerated in Canadian federal institutions. These analyses yielded far too many curves to present here, but all CCFI values are provided in Table 2. For each indicator set, results across subsamples were consistent with those in the full sample in supporting an inference of dimensional structure.

Discussion

Summary of Results

Overall, the observed results support a dimensional structure for psychopathy and its components as measured by the PCL–R. Whether considered separately or in combination, none of the analyses provided results consistent with a taxonic structure. The primary analyses—those performed with four theoretically based composite indicators—yielded unambiguously dimensional results, and the follow-up analyses produced results that did not follow the patterns observed for prototypical taxonic or dimensional data. Nonetheless, we believe that the totality of evidence warrants a dimensional interpretation for several reasons. First, none of the MAMBAC or MAXEIG curves generated peaks supportive of a taxonic structure. Second, a curve fit

Table 2
Comparison Curve Fit Index (CCFI) Values for MAMBAC and MAXEIG Analyses

Indicator set	Full sample	Max. security[a]	Med. security[b]	Min. security[c]	African American only	Caucasian only	Federal sample[d]
			MAMBAC analyses				
Summative	**.272**	**.293**	**.325**	**.393**	**.279**	**.369**	**.318**
Affective	**.132**	**.420**	**.294**	**.332**	**.330**	**.294**	**.224**
Antisocial	**.420**	**.313**	**.402**	**.398**	**.361**	**.221**	**.400**
Interpersonal	**.220**	**.422**	**.247**	**.278**	**.414**	**.359**	**.337**
Lifestyle	**.429**	**.448**	**.471**	**.360**	**.356**	**.290**	**.201**
PCL–R Factor 1	**.407**	.604	**.362**	.606	.566	**.499**	**.485**
PCL–R Factor 2	.500	.576	**.472**	**.464**	**.460**	**.459**	**.447**
			MAXEIG analyses				
Summative	**.265**	**.338**	**.339**	**.422**	**.290**	**.345**	**.459**
Affective	**.291**	**.371**	**.431**	**.352**	**.359**	.543	**.423**
Antisocial	.537	**.501**	**.428**	**.426**	**.470**	**.403**	**.499**
Interpersonal	**.194**	**.395**	**.395**	**.379**	**.409**	**.376**	**.256**
Lifestyle	**.340**	**.421**	**.483**	**.480**	**.352**	**.477**	**.198**
PCL–R Factor 1	**.424**	**.438**	**.453**	.503	.506	.565	**.390**
PCL–R Factor 2	**.386**	.508	**.473**	**.408**	**.441**	**.486**	**.472**

Note. CCFI values < .50 support dimensional structure and appear in bold print; CCFI values > .50 support taxonic structure. MAMBAC = mean above minus below a cut; MAXEIG = maximum eigenvalue; Max. = maximum; Med. = medium; Min. = minimum; PCL–R = Psychopathy Checklist—Revised.
[a] Samples 9 and 12.   [b] Samples 1, 5, 7, 8, 11, 13, and 14.   [c] Samples 2, 3, and 6.   [d] Sample 15.

index based on bootstrapped sampling distributions of results for taxonic and dimensional comparison data consistently suggested dimensional structure. Third, taxon base-rate estimates were inconsistent across analyses, and, more often than not, the means and standard deviations of these estimates were better reproduced by the dimensional than by the taxonic comparison data. Fourth, regardless of how the sample was divided, these same results were found.

Table 3
Summary of Taxon Base-Rate Estimates

| Indicator set | MAMBAC | | | MAXEIG | | | L-Mode | |
	Estimates	*M*	*SD*	Estimates	*M*	*SD*	Mode 1	Mode 2
Summative	4	.54	.08	4	.66	.07	.00	.80
Sim. Tax. Data		.65	.07		.43	.02		
Sim. Dim. Data		.54	.04		.62	.06		
Affective facet	4	.95	.10	6	.55	.23		
Sim. Tax. Data		.74	.15		.59	.16		
Sim. Dim. Data		.94	.12		.50	.20		
Antisocial facet	5	.53	.11	10	.68	.24		
Sim. Tax. Data		.56	.05		.61	.15		
Sim. Dim. Data		.51	.07		.56	.20		
Interpersonal facet	4	.56	.21	6	.42	.11		
Sim. Tax. Data		.36	.24		.50	.11		
Sim. Dim. Data		.52	.18		.37	.15		
Lifestyle facet	5	.59	.09	10	.66	.20		
Sim. Tax. Data		.57	.05		.63	.09		
Sim. Dim. Data		.55	.08		.61	.13		
PCL–R Factor 1	8	.60	.11	28	.52	.18		
Sim. Tax. Data		.56	.10		.52	.20		
Sim. Dim. Data		.57	.09		.52	.20		
PCL–R Factor 2	10	.68	.07	45	.69	.20		
Sim. Tax. Data		.60	.06		.60	.17		
Sim. Dim. Data		.69	.14		.61	.18		

Note. MAMBAC = mean above minus below a cut; MAXEIG = maximum eigenvalue; L-Mode = latent mode; Sim. Tax. Data = Simulated taxonic data; Sim. Dim. Data = simulated dimensional data; PCL–R = Psychopathy Checklist—Revised.

Integration of Current Results With Previous Studies

These results are consistent with the results found by Guay and Knight (2003) with the MASA, by Marcus et al. (2004) with the PPI, and by Edens et al. (2006) with the PCL–R, but they differ from the four studies that identified taxonic distributions. It is likely that the methodological problems in the four studies that found evidence for a taxon, delineated in the introductory paragraphs and addressed in the present study, are responsible for the differences in the results. The present study counters the problems of these prior studies by accessing a larger and more representative sample and by analyzing subsamples in detail to ensure that particular selection criteria did not bias results. Both interview and archival file review were used in arriving at PCL–R scores, and only those offenders for whom ratings on all items were possible were included, guaranteeing a more complete and detailed coverage of both Factor 1 and Factor 2 content and guarding against the biasing tendency for raters to use related items to arrive at judgments for items with insufficient information. Finally, more sophisticated analytic procedures were introduced, including the comparison of simulated taxonic and dimensional comparisons generated from the same sample and quantitative analyses of curve fits to reduce some of arbitrariness of the interpretation of the results.

Interpretations of the Dimensional Results

The clear and strong indications of the dimensionality of psychopathy can be interpreted in several ways. These range from the acceptance of psychopathy as dimensional and possibly an extreme on one or more normative personality traits, to questioning the PCL–R as the appropriate assessment to uncover a taxon, to considerations about the homogeneity of psychopathy and the potential multiplicity of its underlying core processes. We consider each of these alternative perspectives in turn.

Psychopathy as a dimension. Consistent with the results of these taxometric analyses, we might conclude that both the latent structure of psychopathy and its core factors may best be interpreted as dimensionally distributed. A shortcoming of taxometric analysis is its failure to parameterize any particular alternative dimensional model (Krueger, 2006). Consequently, the disconfirmation of a taxonic model does not suggest or corroborate any particular alternative model, but rather is simply consistent with research that supports the hypothesis that personality disorders in general may be best conceptualized as distinct configurations of extreme scores on personality traits, affective and cognitive competences, or neurobiological processes that exist on a continuum with normal functioning (e.g., Widiger, 1993; Widiger & Costa, 1994). Using the perspective of the five-factor model (FFM; McCrae & Costa, 1990, Lynam (2002) has recently presented data that support the application of a dimensional conceptualization to psychopathy. He argued both from expert-generated FFM psychopathy prototypes and correlations of the FFM with measures of psychopathy that the psychopath could be described as low on all facets of Agreeableness, and low in the dutifulness, deliberation, and self-discipline facets of Conscientiousness. Mixed results emerged for Neuroticism and Extraversion. For Neuroticism, whereas the facets of anxiety, self-consciousness, and perhaps vulnerability and depression were low for the psychopath, the facets of impulsivity and angry hostility were high. The Extraversion facet of excitement seeking was high for the psychopath, but the facets of warmth and perhaps positive emotions were low. Lynam (2002) concluded that psychopathy could best be conceptualized by extreme scores on a collection of FFM personality traits. More generally, Bishopp & Hare (2005) have reported that a multidimensional scaling analysis of the data set used in the present study provided support for a multidimensional structure within the PCL–R, corroborating the hypothesis that psychopathy can be viewed as an extreme variant of multiple dimensions of personality. Psychopathy might also be conceptualized within the framework of an "externalizing spectrum" of personality and psychopathology (Krueger, 2006).

Conceiving of psychopathy as a dimension carries several implications about optimal strategies for studying the disorder. It argues for moving away from extreme group designs that attempt to distinguish psychopaths from nonpsychopaths or from trichotomizing PCL–R scores and assuming that discrete groups have been formed for comparison purposes (Lilienfeld, 1994) and toward dimensional designs, such as the quantitative, latent trait model-based approach proposed by Krueger, Markon, Patrick, and Iacono (2005), in explicating the comorbidity among externalizing disorders. The dimensionalization of psychopathy is also consistent with the recent increase in research on subclinical manifestations of psychopathy (Hall & Benning, 2006) and suggests the importance of such research for unraveling etiological factors of the components of psychopathy. In this regard, it is interesting to note that the factors of psychopathy have recently been identified as critical in predicting sexual coercion against women (Knight & Guay, 2006). The predictive potency of these factors is similar in criminal, noncriminal, juvenile, and adult samples (Knight & Sims-Knight, 2003, 2004), suggesting a gradual rather than a step function in the contribution of psychopathy to rape.

Certainly, the strong evidence for the dimensional latent structure of psychopathy should affect how this construct is conceptualized and used in criminal justice proceedings. The PCL–R is the most commonly cited measure in such proceedings (Edens & Petrila, 2006; Hare, 1996) and is a part of some actuarials that have been constructed to predict both violence (e.g., *Violence Risk Appraisal Guide*; Harris, Rice, & Quinsey, 1993) and sexual violence (e.g., *Sex Offender Risk Appraisal Guide*; Quinsey et al., 1998). In civil commitment procedures for sexual predators, it is sometimes a critical issue to determine whether a defendant is or is not "a psychopath" (Edens & Petrila, 2006). The present research suggests that criminal justice language and conceptualization should be modified and should stop talking about individuals as being "psychopaths." Rather, lawyers should refer to defendants as being "high on measures of psychopathy." Although this appears to be a subtle difference, it may have important consequences in reducing juries' perceptions of particular defendants as different in kind. The suggestion of Edens and Petrila (2006) to have confidence intervals as well as discrete scores reported in court cases is also apropos. The data supporting the dimensionalization of psychopathy also suggest that actuarial measures used in the criminal justice system might profit from the use of the full PCL–R score rather than by reifying an arbitrary dichotomization or by weighting scores to give more leverage to a purported taxon.

PCL–R as a questionable intervening measure. The specificity of both the PCL–R and self-report measures of psychopathy,
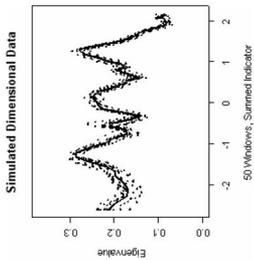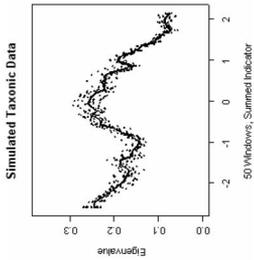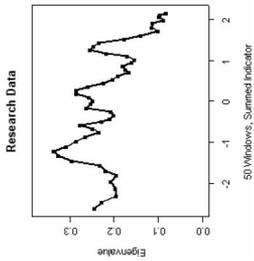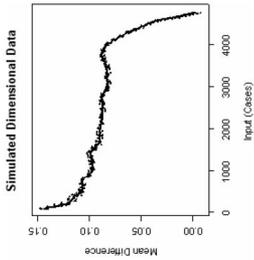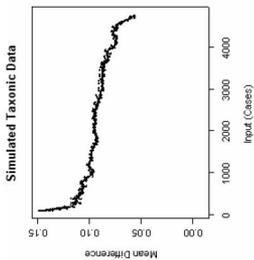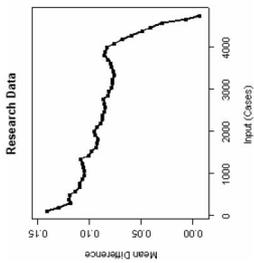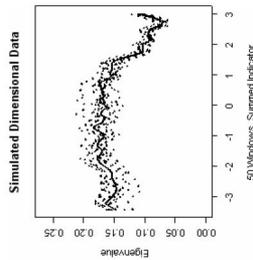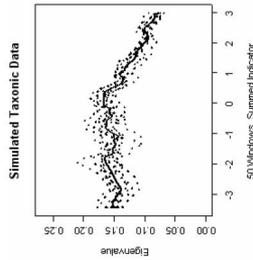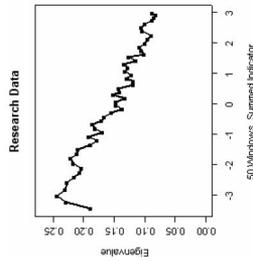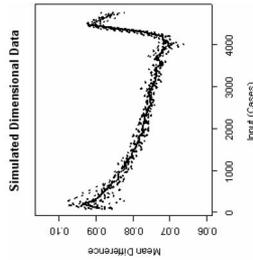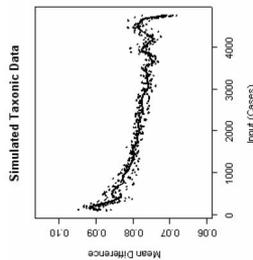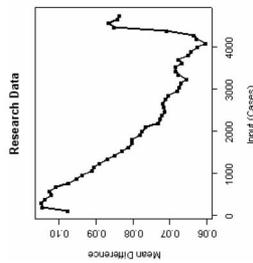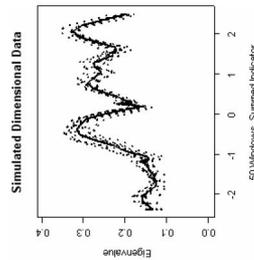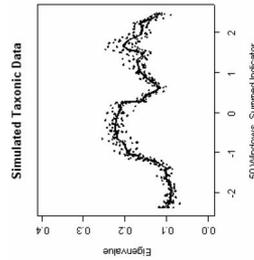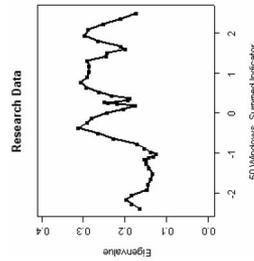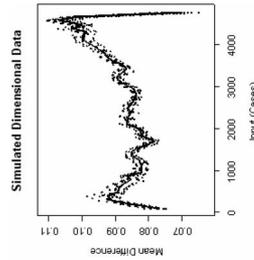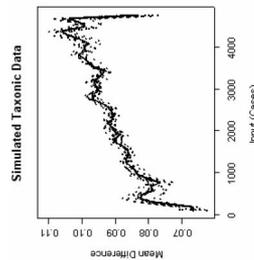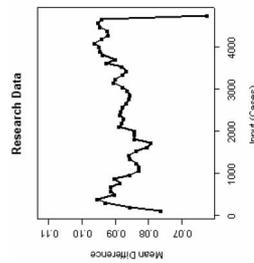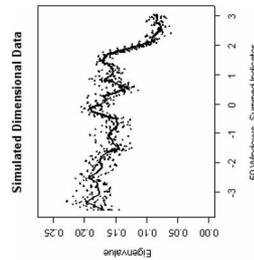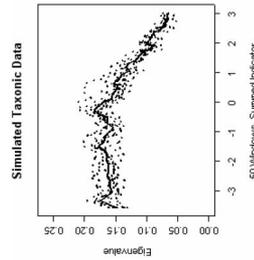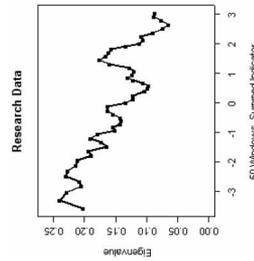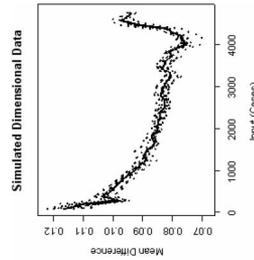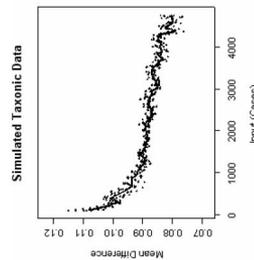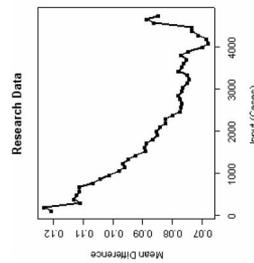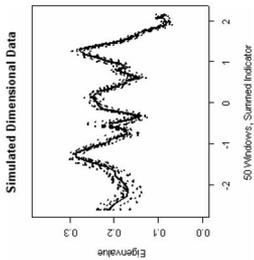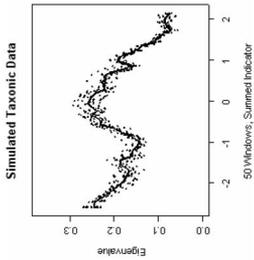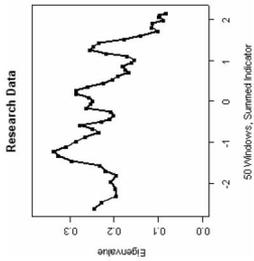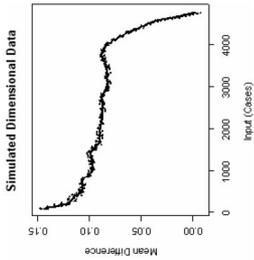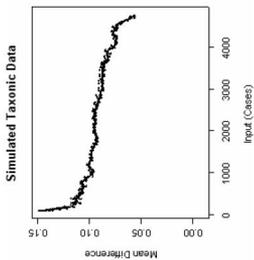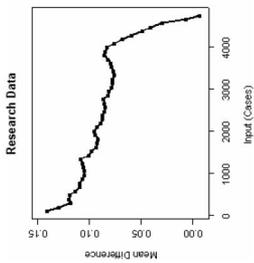
like the MASA and PPI, may not be sufficient to identify genotypic and biological causation. It is possible that such assessment instruments, which predominantly measure interpersonal behavior in a social environment, may be too distant phenotypically from the biological substrate to covary with a hypothetical taxon. Endophenotypic (Gottesman & Shields, 1972) indicators should be explored. One approach might be to use as dependent variables scores on the cognitive (e.g., Kosson, 1996, 1998; Newman, 1987; Newman et al., 1987; Schmauk, 1970) and affective (e.g., Levenston, Patrick, Bradley, & Lang, 2000; Patrick, Bradley, & Lang, 1993) measures that have been found to covary with psychopathy and might better tap processes more closely related to underlying mechanisms. Although some biological markers may be nonspecific (e.g., reduced P300 latency) or causally downstream from symptomatic measures of psychopathology, less molar, less "psychological," or less "social" kinds of indicators are likely to be connected to any putative underlying disposition by shorter causal chains and hence may involve fewer attenuating factors. Such measures may have a higher likelihood of being taxometrically strong indicators of psychopathy, if indeed it constitutes a taxon.

Heterogeneity in psychopathy and the obscuring role of subtypes. The typological purity of psychopathy has often been challenged. It has most commonly been proposed that two distinct subtypes of psychopaths can be identified, often referred to as *primary* and *secondary psychopathy* (Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003). Frick, Lilienfeld, Ellis, Loney, and Silverthorn (1999) have argued that "cooperative suppressor" effects of two correlated psychopathic types might mask important differences between the types. It might also be argued that the presence of these correlated subtypes might mask taxonic differences as well.

Using Fraley's (1998) model-based clustering, which in contrast to other clustering techniques provides a fit index that allows one to assess the best fitting model, Hicks, Markon, Patrick, Krueger, and Newman (2004) have provided the most compelling demonstration of these subtypes. They cluster analyzed a group of high PCL–R psychopaths with the scales from the Multidimensional Personality Questionnaire—Brief Form (MPQ–BF; Patrick, Curtin, & Tellegen, 2002) as dependent measures. The best model yielded two clear clusters. The first cluster, comparable to the primary psychopath and labeled the *stable psychopath*, was low in Stress Reaction, Social Closeness, and Harm Avoidance, and high in Agentic Positive Emotionality. The authors interpreted this type as approximating the classical psychopath, who is immune to stress, who is socially dominant, but who is unattached to others, and who is prone to take risks. The second subtype, called the *aggressive psychopath*, scored high on Negative Emotionality and low on Constraint and was characterized by high Aggression. This type appears to capture undercontrolled, externalizing individuals

(e.g., Krueger et al., 2002), whom the authors compared with Moffitt's (1993) life-course persistent offenders.

Even though these markedly different personality types apparently emphasize specific characteristics of the PCL–R superordinate factors, these types are nonetheless incongruent with these factors, and their presence might mask the manifestation of underlying taxa. One strategy to resolve this issue would be to use measures independent of the PCL–R to remove one or the other of these subtypes and to conduct the taxometrics on the remaining offenders. For instance, one could follow Newman's strategy (e.g., Newman & Schmitt, 1998; Newman et al., 1997) and use the Welsh Anxiety Scale (Welsh, 1956) to remove alternatively high- and low-anxious psychopaths from the analyses. The results of Hicks et al. (2004) could also be used to identify MPQ–BF profiles for the two subtypes, and each subtype could be removed in alternate analyses leaving the other to determine whether a taxon solution emerges in the absence of either.

Problems of mapping from the phenotypic to underlying causes. A final possible interpretation is illustrated in the neurobehavioral model of personality traits proposed by Depue and Lenzenweger (2001). They conceive personality disorders as functions of the variations or interactions of the most extreme values that contribute to high-order traits: Constraint, Affiliation, Positive–Negative Emotionality (PEM–NEM), and Fear. The authors proposed that higher order heterogeneous phenotypes result from the combination of a heterogeneous set of lower order traits that have different sources of genetic variation. The task of identifying neurobiological foundations is rendered particularly difficult, considering that some higher order traits are typically associated with two or more behavioral systems and neurobehavioral networks (Depue & Lenzenweger, 2001). Consequently, it would be particularly difficult to observe taxonic results.

As Gottesman (1997) illustrated in the domain of intelligence assessment, phenotypical continuity can represent the true manifestation of distinct underlying genotypic distributions. Genetic and biological causation and phenotypic discontinuity are not synonymous. Depue and Lenzenweger (2001) suggested that at least four different neurobehavioral systems may be necessary to account for impulsivity—(a) positive incentive motivation, (b) fear, (c) aggression, and (d) low levels of a nonaffective form of impulsivity (which results in disinhibition of the above neurobehavioral systems). For impulsivity to emerge, at least four to five independent neurobehavioral systems may have to be elicited (not to mention an increased complexity when possible interaction with other higher order traits is considered). Ultimately, the determination of the number of core processes that are necessary and sufficient to explain and predict psychopathy will depend on the isolation of the basic processes involved (Depue & Lenzenweger, 2001; Tellegen & Waller, in press). Specific etiologies, including particular genetic origins, prenatal, perinatal, and post-

Figure 2 (opposite). Mean above minus below a cut (MAMBAC) and maximum eigenvalue (MAXEIG) results for indicator sets representing four theoretical facets of psychopathy. Aff. = affective; Anti. = antisocial; Inter. = interpersonal; Life. = lifestyle. Within each three-panel graph, the first panel shows the averaged curve for the research data and the second and third panels show the averaged curves for the simulated taxonic and dimensional comparison data. For comparison data, each sample's results are represented by a dotted line, with the average across all samples plotted as a solid line.

PCL–R Factor 1 – MAMBAC
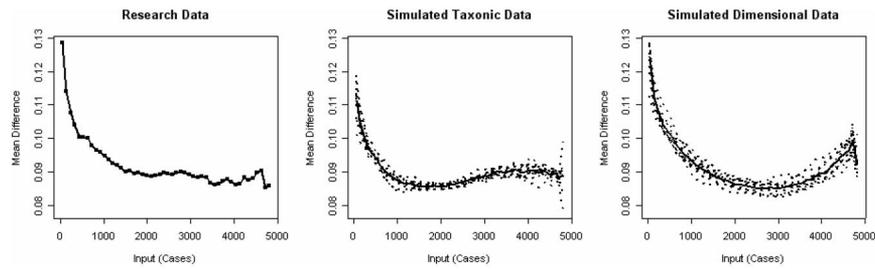
PCL–R Factor 1 – MAXEIG
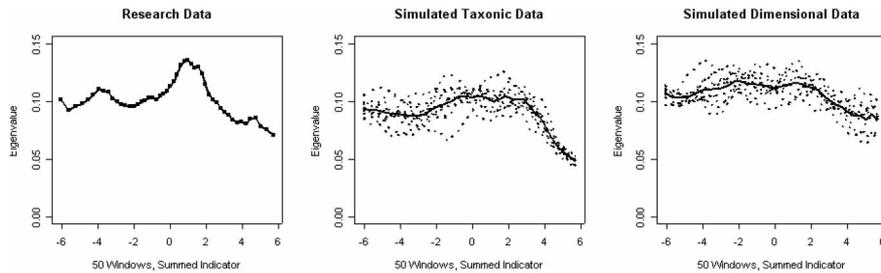
PCL–R Factor 2 – MAMBAC
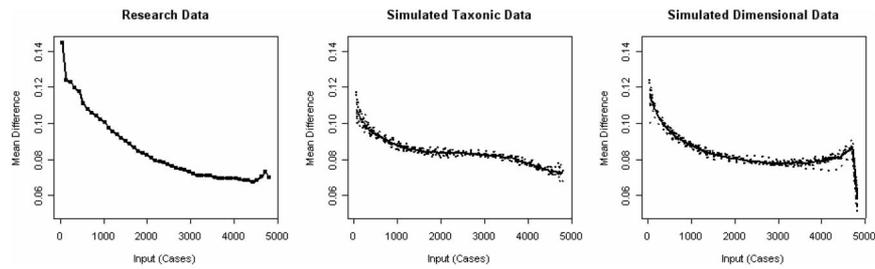
PCL–R Factor 2 – MAXEIG
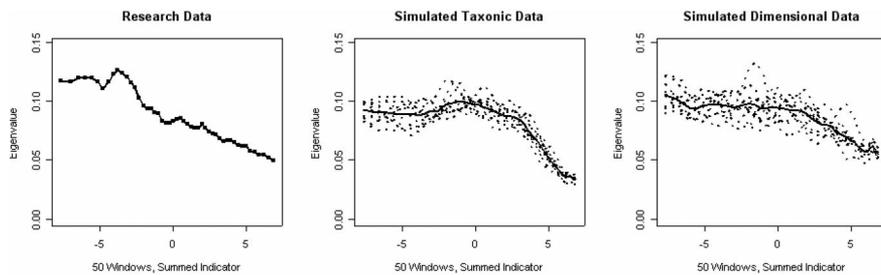
Figure 3. Mean above minus below a cut (MAMBAC) and maximum eigenvalue (MAXEIG) results for indicator sets representing Psychopathy Checklist—Revised (PCL–R) Factors 1 and 2. Within each three-panel graph, the first panel shows the averaged curve for the research data and the second and third panels show the averaged curves for the simulated taxonic and dimensional comparison data. For comparison data, each sample's results are represented by a dotted line, with the average across all samples plotted as a solid line.

natal biological determinants, and specific life experiences impacting on causal neurobiological influences within personality structure will determine the relevant processes contributing to psychopathy. As in the hunt for the "quantitative trait loci" of

intelligence (Gottesman, 1997), such complexity does not easily yield its underlying causal mechanisms to empirical scrutiny, and investment in a long-term search is necessary. The advantage of being able to identify a taxon in the phenotypical distributions of

PCL–R or its subcomponents would have been the location of a beacon guiding us to a specific etiology (Meehl, 1977). The absence of such a beacon may mean that no phenotypic measures, even cognitive or affective assessments that may be closer to the underlying causal mechanisms, will provide such guidance, and longitudinal research strategies aimed at identifying and tracking multiple underlying processes and environmental contingencies will be necessary.

Conclusions

The analyses in the present article clearly indicate that the disorder defined by high scores on the PCL–R and the correlated factors that the PCL–R comprises are distributed dimensionally. Although such results strongly disconfirm the hypothesis that the distribution of PCL–R scores can be accounted for by a latent taxon, questions about the specificity of the measure in covarying with underlying processes, the possibility of covarying but etiologically distinct subtypes of psychopathy, and the possibility that the manifest behavior of psychopathy is the interaction of multiple independent neurobiological systems leave open the possibility that other measures might yet uncover a taxon. The support for a dimensional model of psychopathy buttresses research strategies that examine the correlates of psychopathy self-report scales in both criminal and noncriminal samples (Benning, Patrick, Salekin, & Leistico, 2005; Brook, Kosson, Walsh, & Robins, 2005; Knight & Sims-Knight, 2003) encourages structural equation modeling approaches to conceptualizing and testing etiological hypotheses about psychopathy (Krueger et al., 2002) and argues for the creation of scales that are equally reliable and discriminating across the full spectrum of the scale. Although we should not yet forego the more traditional group comparison approaches, such data should be analyzed and interpreted with dimensional precautions in mind. We have not resolved the issue of taxonicity sufficiently to leave any strategy behind in the attempts to unravel the Gordian knot of psychopathy.

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