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Psychopathic, Not Psychopath: Taxometric Evidence for the Dimensional Structure of Psychopathy

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Although psychopathy is frequently regarded as qualitatively distinct from other conditions, relatively little research has examined whether psychopaths represent a distinct class of individuals. Using a sample of 876 prison inmates and court-ordered substance abuse patients who were administered the Psychopathy Checklist—Revised (R. D. Hare, 2003), the authors examined the latent structure of psychopathy using several taxometric procedures developed by Meehl and colleagues (P. E. Meehl & L. J. Yonce, 1994; N. G. Waller & P. E. Meehl, 1998). The results across these procedures offer no compelling support for the contention that psychopathy is a taxonic construct and contradict previous reports that psychopathy is underpinned by a latent taxon. The authors discuss the theoretical, public policy, and practice-level implications of these findings.

Keywords: psychopathy, PCL–R, taxometrics, discrete class, taxon

Psychopathic personality (psychopathy) is a constellation of relatively distinctive personality traits (e.g., callousness, grandiosity, pathological lying) that may occur in the context of a criminal or socially deviant lifestyle. Although overlapping with the *Diagnostic and Statistical Manual of Mental Disorders* diagnosis of antisocial personality disorder, psychopathy is a separable construct that places a greater emphasis on affective and interpersonal traits. In fact, in prison settings, most individuals who meet the diagnostic criteria for antisocial personality disorder do not meet the standard definition of psychopathy (Hare, 2003; cf. Skilling, Harris, Rice, & Quinsey, 2002). The widespread theoretical and applied interest in the construct of psychopathy is evident in the large number of peer-reviewed journal articles published on the topic over the past few decades (e.g., several recent special issues of journals such as *Behavioral Sciences and the Law* and *Criminal Justice and Behavior* have been devoted specifically to the topic of psychopathy) as well as in the growing use of this disorder to inform legal decision making across the world (for overviews, see Edens & Petrila, 2006; Ogloff & Lyon, 1998).

Psychopathy, typically operationalized by the Psychopathy Checklist—Revised (PCL–R; Hare, 1991, 2003), is associated with a number of real world criterion measures of considerable practical significance, such as increased risk for misconduct while institutionalized (Guy, Edens, Anthony, & Douglas, in press), community violence (Skeem & Mulvey, 2001), and criminal recidivism following release from prison (Hemphill, Hare, & Wong, 1998). Laboratory studies also have suggested that high scorers on the PCL–R and other measures of psychopathy differ in theoretically meaningful ways from other individuals, such as in their lower resting electrodermal activity, aberrant processing of affectively charged linguistic and nonlinguistic stimuli, and lack of responsiveness to punishment cues (see Hare, 2003; Lorber, 2004).

Although the clinical and theoretical importance of psychopathy is well established, there remain a number of unresolved and intensely debated issues regarding the nature of the construct, such as the generalizability of psychopathy across gender and ethnic groups (e.g., Cale & Lilienfeld, 2002; Skeem, Edens, Camp, & Colwell, 2004), the putative existence of variants or subtypes of psychopathy (e.g., Brinkley, Newman, Widiger, & Lynam, 2004;

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Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003), and the nature of the underlying factor structure of the PCL–R (e.g., Cooke & Michie, 2001; Cooke, Michie, Hart, & Clark, 2005; Hare, 2003). Regarding the latter issue, research on psychopathy typically has focused on two moderately correlated factors initially identified by Hare and colleagues (Harpur, Hakstian, & Hare, 1988; Harpur, Hare, & Hakstian, 1989). More recent factor analytic work (discussed below), however, suggests that either a three-factor (Cooke & Michie, 2001; Cooke et al., 2005) or four-factor (Hare, 2003) model may better represent the dimensions tapped by the PCL–R than the traditional two-factor approach.

Another ongoing controversy that continues to attract research attention is the extent to which psychopathic personality is dimensional or categorical in nature. That is, to what extent does the construct of psychopathy identify a fundamentally distinct class of individuals who differ qualitatively from the rest of society? Although this question is by no means specific to psychopathy (e.g., similar questions have been raised in relation to other personality disorders [Haslam, 2003] as well as various Axis I diagnoses), the outcome of this debate may be of considerable practical significance given the increasing role of the highly charged label of *psychopath* in the legal system, where the PCL–R has been used to justify indeterminate commitment, rebut insanity defenses, and bolster support for the death penalty in capital murder trials (Cunningham & Reidy, 2002; DeMatteo & Edens, 2005; Edens, in press; Edens & Petrila, 2006; Edens, Petrila, & Buffington-Vollum, 2001; Ogloff, & Lyon, 1998).

Although some researchers have argued that psychopathy is a constellation or configuration of extreme levels of continuously distributed personality traits (e.g., Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Lilienfeld & Fowler, 2006; Miller, Lynam, Widiger, & Leukefeld, 2001), other researchers have staked out a position that strongly endorses a taxonic structure. For example, Harris, Skilling, and Rice (2001) asserted that psychopaths "comprise a *discrete natural class* [italics added] of individuals" (p. 197) and that there are fundamental, qualitative differences between psychopaths and nonpsychopaths. Moreover, in other contexts, this research team has referred to their alternative measure of the psychopathy construct as the *Child and Adolescent Taxon Scale* (Quinsey, Harris, Rice, & Cormier, 1998), ostensibly reflecting a categorically distinct class of individuals who can be identified at a very early age. Consistent with this argument, Skilling, Quinsey, and Craig (2001) reported evidence of a low base rate taxon underlying antisocial behavior in a community sample of boys. The extent to which these findings identify a latent taxon in reference to psychopathy is unclear, as most of this research (see below) has found support for such a taxon in relation to socially deviant and antisocial behavior rather than the personality traits associated with psychopathy (e.g., Cleckley, 1988) per se.

The dimension versus taxon question is relevant to a number of debates related to psychopathy, such as its etiology. For example, a taxon in some instances can be explained by a specific causal agent (e.g., Huntington's disease results from the action of a single gene). If psychopathy were shown to be dimensional, however, it would be unlikely to result from a dichotomous causal agent, such as the presence–absence of frontal lobe damage, a threshold level of an environmental stressor (e.g., severe child abuse), or a dominant gene (see Meehl & Golden, 1982). From a pragmatic perspective, support for the position that psychopathy is taxonic would render it more difficult to justify research programs that focus on studying psychopathy in samples in which the base rate is likely to be very low, such as college students (Lilienfeld, 1998). In contrast, support for the position that psychopathy is dimensional would provide at least some indirect support for the practice of conducting psychopathy research in college or community samples, although it is worth noting that these samples may be characterized by a paucity of extremely high scorers on psychopathy measures. If certain clinically important behaviors associated with psychopathy (e.g., serious physical aggression, severe illicit substance use) tend to emerge only among individuals with markedly elevated scores, then psychopathy research on college or community samples could yield incomplete or misleading conclusions even if psychopathy were found to be dimensional.

Evidence supporting the taxonic structure of psychopathy derives primarily from an influential study conducted by Harris, Rice, and Quinsey (1994) as well as a follow-up report reexamining many of the same participants (Skilling et al., 2002). Harris et al. (1994) conducted taxometric analyses (Meehl & Golden, 1982) of the PCL–R using data from a large sample of mentally disordered offenders detained in Canadian psychiatric facilities. Harris et al. (1994) asserted unequivocally that their findings demonstrated that a taxon could be identified using PCL–R scores, with a base rate in their sample ranging from .44 to .46 across four sets of analyses. They also claimed that a cut score of 19 or 20 would optimally differentiate between the taxon and complement classes, although these scores are considerably lower than the traditional cut score (total score $\geq$ 30) for "diagnosing" psychopathy using the PCL–R (Hare, 1991, 2003). Although their conclusions applied to psychopathy per se, Harris et al. (1994) found evidence of a taxon only for PCL–R Factor 2, which is composed of items assessing a chronically unstable and socially deviant lifestyle. Their analyses relevant to PCL–R Factor 1, which represents such affective and interpersonal features as grandiosity, callousness, and superficial charm, were inconsistent with a taxonic structure.

Since the publication of this study, several criticisms have been raised regarding Harris et al.'s (1994) methodology and statistical analyses (Lilienfeld, 1998; Marcus, John, & Edens, 2004). For example, the sample was an atypical group of criminal offenders—inmates in a maximum-security psychiatric institution—a large minority of whom had been adjudicated not guilty by reason of insanity (NGRI). Although the authors conducted ancillary analyses in which patients with psychotic diagnoses were removed, examination of the reported sample sizes suggests that a significant number of NGRI acquittees remained in the sample, even though they had not been diagnosed as psychotic. This fact raises the possibility that Harris et al. (1994) could have inadvertently uncovered a taxon for schizotypy (see Lenzenweger & Korfine, 1992, for evidence that schizotypy is taxonic). The content of the PCL–R items showing evidence of a taxon (e.g., reflecting persons who lacked realistic goals, were irresponsible, and had childhood behavior problems) was relatively nonspecific; Cooke & Michie, 1997). The schizotypy hypothesis also is rendered plausible by findings suggesting that at least some psychopathy measures, including a preliminary version of the PCL (the precursor of the PCL–R), may erroneously detect a nontrivial number of patients with schizophrenia (Howard, Bailey, & Newman, 1984; but see Hare & Harpur, 1986, for a critique).

Additionally, the PCL–R scores used in the taxometric analyses were based solely on file review data, with no interview information to supplement the ratings. Although it is acceptable to score the PCL–R for clinical and research purposes solely on the basis of adequate file data, it is unclear whether or how such a strategy might impact taxometric analyses conducted on these ratings. Furthermore, each item of the PCL–R was dichotomized instead of scored using the standard 3-point scoring system—a scoring method that may enhance the probability of detecting a pseudotaxon (Lenzenweger, 2004; Meehl & Yonce, 1996; M. B. Miller, 1996). Moreover, although it is standard practice in taxometric studies to report the validity of the indicators included in the analyses (i.e., the difference in scores on the indicators between members of the putative taxon and the complement in standard deviation units), neither Harris et al. (1994) nor Skilling et al. (2001) reported indicator validity values. Finally, the item-level indicators were more strongly correlated with the PCL–R total score in the Harris et al. (1994) sample than would be expected on the basis of the data reported in the 1991 and 2003 manuals. For example, the correlation between Item 15 and the total score was .70, whereas this correlation among forensic patients in the PCL–R manual (Hare, 2003) is only .41. Similarly, Item 9 was more strongly correlated with the total score in their sample ($r = .63$) than in the manual ($r = .37$). In other words, inmates rated as possessing one attribute of psychopathy were also more likely (than was typically the case in the normative PCL–R data) to be seen as having other attributes of psychopathy. Given the sole reliance on file review, the dichotomous rating scales used, and the higher-than-expected corrected item–total correlations, it seems possible, if not likely, that the taxonic findings obtained for Factor 2 are questionable.

Since the Harris et al. (1994) article, additional taxometric procedures have been developed (Waller & Meehl, 1998), and researchers have become more sensitive to the dangers of producing pseudotaxonic solutions that could result from skewed distributions (e.g., Hankin, Fraley, Lahey, & Waldman, 2005; J. Ruscio, Ruscio, & Keane, 2004). Recent research examining the latent structure of psychopathy has been much less supportive of an underlying taxon, although no published studies explicitly have examined the structure of psychopathy using the PCL–R. Marcus et al. (2004) conducted taxometric analyses on a sample of jail and prison inmates who had been administered the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996), which is a promising self-report measure designed to assess the core personality dimensions of psychopathy. Across several taxometric analyses, there was virtually no evidence supporting a categorical model. These findings are broadly consistent with the Harris et al. (1994) findings for Factor 1 of the PCL–R. Inconsistent with Harris et al.'s (1994) taxonic results for Factor 2, however, are findings suggesting that the more chronically antisocial aspects of psychopathy also are dimensional in nature (Bucholz, Hesselbrock, Heath, Kramer, & Schuckit, 2000; see also Osgood, McMorris, & Potenza, 2002). Nevertheless, because the analyses of Bucholz et al. (2000) and Osgood et al. (2002) were not based on PCL–R Factor 2 data specifically and were performed using statistical procedures other than taxometrics, their relevance to Harris et al.'s (1994) psychopathy findings is indirect at best.

The two published taxometric studies that have directly examined the latent structure of psychopathy have yielded somewhat inconsistent results. Although the methodologies used by Harris et al. (1994) and Marcus et al. (2004) differed in a number of ways (e.g., Harris et al., 1994, used univariate taxometric analyses, whereas Marcus et al., 2004, used multivariate methods; Harris et al., 1994, used a maximum-security forensic sample, many of whom were acquitted on the basis of NGRI, whereas Marcus et al., 2004, used jail and prison inmates, who were far less likely to be severely mentally ill), perhaps the most important difference was that Harris et al. (1994) used the PCL–R to assess psychopathic traits, whereas Marcus et al. used the PPI. Thus, the question of whether psychopathy per se is dimensional or categorical or whether these differing results were artifacts of the instruments used remains unresolved. Although concerns have been expressed about an overreliance on the PCL–R as the exclusive measure of psychopathic traits (e.g., Poythress, Edens, & Lilienfeld, 1998), there is little question that it is the most widely used and extensively validated measure of this construct (Hare, 2003). Moreover, given the potential etiological, pragmatic, and public policy implications of the taxometric status of psychopathy, research addressing this issue specifically in relation to the PCL–R would be informative.

Additionally, Hare (2003) recently has argued for a four-factor solution to the PCL–R in which the original two factors are split into narrower facets. More specifically, Factor 1 is composed of separable interpersonal (e.g., glibness, pathological lying) and affective (callousness, shallow affect) facets (four items each), whereas Factor 2 is composed of separable lifestyle (e.g., impulsivity, parasitic lifestyle) and antisocial (e.g., juvenile delinquency, criminal versatility) facets (five items each). The relevance of this newer model is that it allows taxometric analyses to be conducted at the scale (as opposed to item) level, which was not possible at the time Harris et al. (1994) conducted their analyses. Although debates exist concerning the theoretical relevance of Hare's (2003) fourth facet (Cooke & Michie, 2001; Cooke et al., 2005; Hill, Neumann, & Rogers, 2004), this model does encapsulate the multidimensional nature of the construct of psychopathy as operationalized by the PCL–R, which comprises a mixture of emotional, behavioral, and interpersonal features. In other words, most individuals who commit crimes are not psychopathic, nor are most callous individuals psychopathic: The term typically is reserved for those who evidence psychopathic qualities across these domains. If psychopathy is a taxon, then these four domains should co-occur in a nonrandom manner that would reflect this latent structure.

In the present study, like in Harris et al. (1994), we used the PCL–R to examine the latent structure of psychopathy. However, we attempted to avoid many of the pitfalls of that earlier study. First, by including interviews in addition to file reviews, our administration of the PCL–R was consistent with the method preferred by the instrument's developer (Hare, 2003). Second, we scored the PCL–R using the standard 3-point scoring method and did not artificially dichotomize the data. Third, we did not recruit mentally ill offenders, so if a taxon were to be identified, it would be less likely to be a schizotypy taxon. Fourth, as additional consistency checks, we incorporated multivariate taxometric procedures (e.g., maximum eigenvalue, latent mode factor analysis; described below) that had not been published until Waller and Meehl (1998). Fifth, we took advantage of the new four-facet model of the PCL–R to conduct taxometric analyses of indicators that should be more stable and valid than the individual-item indicators used in the previous PCL–R taxometric studies. Finally, given concerns about (a) the sample, (b) the administration of the PCL–R, (c) the inflated item–total correlations, and (d) the absence of indicator validity data in the Harris et al. (1994) study, we attempted to replicate their findings using the same individual-item indicators that they had used. If Harris et al. (1994) succeeded in detecting a psychopathy taxon, independent investigators should be able to replicate this finding using the same indicators.

Method

Participants

Participants were male offenders who had completed the PCL–R (Hare, 1991, 2003) as part of a larger study funded by the National Institute of Mental Health examining personality features and their relation to social deviance. These individuals were serving sentences in state prisons in Florida, Nevada, Utah, or Oregon, or they were in court-ordered residential drug treatment sites in Florida, Texas, Utah, Nevada, or Oregon. Participation was limited to English-speaking individuals between the ages of 21 and 40 (inclusive) and to European American or African American racial groups. Participants also had to complete a preliminary IQ screen and obtain an estimated IQ ≥ 70. Across all sites, individuals receiving psychotropic medication for active symptoms of psychosis were excluded from participation. Because we conducted item-level analyses, study participants who were missing any of the individual PCL–R items (10%) were dropped from this data set, which resulted in a final sample of 876 for the analyses reported here. Approximately 59% of the sample identified themselves as Caucasian, 31% identified themselves as African American, and 10% identified themselves as Hispanic. The mean age was 31.56 years ($SD = 7.04$).

Measures

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

PCL–R (Hare, 1991, 2003). The PCL–R consists of 20 items reflecting psychopathic personality traits and behaviors that are scored on the basis of a semistructured interview and review of institutional file information. Each item is scored on a 3-point scale, depending on the degree to which each applies to the individual, with a possible total score ranging from 0 to 40. As noted earlier, the reliability and construct validity of the PCL–R have been well established (see Hare, 1991, 2003, for an overview). PCL–R ratings for this study were performed by research assistants who had received extensive training in the administration and scoring of this measure prior to the onset of data collection for the larger National Institute of Mental Health study. Interrater reliability for a total of 51 cases was $ICC_1 = .88$. Cronbach's alpha was .81 for the total score in this sample. Notably, unlike the PCL–R results reported by Harris et al. (1994), our corrected item–total correlations for the PCL–R items of interest were generally very similar to those reported in the most recent manual (Hare, 2003).

In terms of the cut score historically used to identify psychopathic and nonpsychopathic groups, 20.4% of the sample scored ≥ 30 on the PCL–R total score, which is generally consistent with base rates reported in other samples of adult offenders. The mean PCL–R score for the sample was 23.16 ($SD = 7.12$), which is generally consistent with mean scores from other research conducted on similar samples (see Hare, 2003, for an overview of several data sets). There was a small but statistically significant difference in the mean total scores across the prison ($M = 24.22$; $SD = 7.08$; $n = 405$) and drug treatment ($M = 22.25$; $SD = 7.03$; $n = 471$) groups, $t(875) = 4.11$, $p < .01$ (Cohen's $d = 0.28$).

As noted earlier, more recent factor analytic work suggests that a four-factor model (Hare, 2003) may better reflect the dimensions tapped by the PCL–R than the two-factor model historically associated with this measure (Hare, 1991). This approach bifurcates the original Factor 1 items into separable affective and interpersonal facets and parses the remaining items into two narrower facets reflecting impulsive lifestyle and antisocial behavior.

Quick Test (Ammons & Ammons, 1962). The Quick Test is a screening measure of intellectual abilities that can be administered in approximately 10 min. The respondent is shown a card that displays four pictures, and the test administrator reads aloud words that represent items or concepts that are represented in only one of the four pictures. Participants then indicate the picture in which they believe the item or concept is portrayed. Only participants who obtained an estimated IQ ≥ 70 were allowed to continue in the study. The Quick Test is an excellent predictor of IQ scores in the normal range (Traub & Spruill, 1982) and provides a good estimate of the Wechsler Adult Intelligence Scale—Revised (Wechsler, 1981) IQ scores in both genders and in both Caucasians and African Americans (Craig & Olsen, 1988). It has also been shown to satisfactorily estimate intelligence scores in offender populations (DeCato & Husband, 1984; Doss, Head, Blackburn, & Robertson, 1986; Simon, 1995).

Procedure

At each site, potentially eligible participants were randomly selected from lists of individuals who met basic inclusion criteria (i.e., age, race, English fluency). Enrollment interviews were conducted in a private room, and informed consent was obtained using procedures approved by a university institutional review board. After informed consent was obtained, the IQ screening test was administered, followed by the full research protocol. As the entire protocol took on average 4.5 hr to complete, it was administered in at least two, and sometimes three, sessions. Except at one agency that did not permit participant payments, at the end of protocol administration, $20 was deposited into the agency account of all participants for the time they contributed to the study.

Data Analytic Strategy

We analyzed PCL–R scores using a series of taxometric procedures developed by Meehl and his colleagues (Meehl & Yonce, 1994; Waller & Meehl, 1998) using statistical software programs written by J. Ruscio (2004). If psychopathy is taxonic, these procedures can detect a qualitative distinction between psychopaths (taxon) and nonpsychopaths (complement). Dimensional data will lack this qualitative difference. Rather than relying on standard null hypothesis testing, the taxometric method applies various mathematically distinct procedures to several combinations of indicator variables, the results of which will tend to converge on a taxonic or dimensional structure. These procedures also produce one or more estimates of the base rate of the taxon in the sample. If the construct of interest is taxonic, these base rate estimates may be consistent (a) both within and across the various taxometric procedures and (b) with estimates of the base rate in the population being sampled. Wide variation among base rate estimates may be more consistent with a dimensional solution.[1]

In this study, we used four taxometric procedures to examine the latent structure of psychopathy: mean above minus below a cut (MAMBAC;

[1] However, J. Ruscio (2005) recently conducted a Monte Carlo study that found that base rate consistency tests were not a reliable method for inferring latent structure: Under certain circumstances, dimensional data yielded more consistent base rate estimates than taxonic data. Because base rate consistency has become somewhat of a convention in the taxometric literature, we still report these values, but no decisions about latent structure were made solely (or even primarily) on the basis of base rate consistency.

Meehl & Yonce, 1994), maximum covariance (MAXCOV; Meehl & Yonce, 1996) or maximum eigenvalue (MAXEIG; Waller & Meehl, 1998), and latent mode factor analysis (L-Mode; Waller & Meehl, 1998). MAMBAC uses two indicator variables, the input indicator *x* and the output indicator *y*. Cuts are made at regular intervals along the input indicator (50 in the present study); at each cut, the mean score on the output indicator for those cases above the cut and the mean scores for those cases below the cut are computed. The difference between these two means is then graphed on the *y*-axis. Taxonic graphs will evidence a single peak, with the location of the peak reflecting the base rate of the taxon. The further to the left side of the graph the peak is, the greater the base rate. If the construct of interest is continuous, the graph will appear concave rather than peaked. A second MAMBAC analysis is performed with the input and output variables reversed to provide a more extensive test of the latent structure. In this study, we used a modification of MAMBAC developed by J. Ruscio (2004) in which multiple indicators are combined to create a single input variable. Because this multivariate procedure makes use of all of the data in each MAMBAC analysis, it may be more powerful than the traditional approach—assuming that all indicator variables demonstrate adequate validity.

MAXEIG is a multivariate extension of Meehl and Yonce's (1996) MAXCOV procedure that uses all of the indicators simultaneously. The sample is first divided into a succession of overlapping windows along the input indicator. In MAXCOV, the covariance between the two output indicators for the cases in each slice is then computed and plotted on the *y*-axis. In MAXEIG, all of the remaining indicators are factor analyzed, and the eigenvalue of the first principal factor, which represents the multivariate analogue of the covariance, is plotted for each window. If taxonic, the eigenvalue should be maximal in the subsample most evenly divided between members of the taxon and the complement, and the graph should peak at this cut. The graph should appear concave, flat, or irregular if the construct is dimensional. Fifty windows with .90 overlap were used for the analyses in the present study.

Finally, the L-Mode procedure factor analyzes all of the indicators and graphs the distribution of scores on the first principal factor. If the construct of interest is taxonic, the graph will be bimodal, whereas a unimodal graph is consistent with a dimensional interpretation.

Results

We performed two sets of taxometric analyses. When Harris et al. (1994) conducted their study, the PCL–R was considered to have only a two-factor structure, which would preclude using factor scores for most taxometric analyses. However, as noted earlier, Hare (2003) recently has argued for a four-factor solution to the PCL–R in which the original two factors essentially are split into narrower facets, which allow for scale-level analyses. Thus, we conducted one set of taxometric analyses using factor scores as the indicators of psychopathic traits.

Our second set of analyses involved item-level data. Harris et al. (1994) performed their analyses by selecting eight items from the PCL–R that correlated most highly with the PCL–R total score in their data set. We therefore used those same eight indicators in an attempt to replicate their findings. For analyses that involved individual items, we used MAXCOV (rather than MAXEIG) and had two variables serve as the outputs and summed the remaining six items to create the input to most closely follow the approach taken by Harris et al. (1994).

Skewed indicators can yield results that appear taxonic even when the latent structure is dimensional (Hankin et al., 2005; J. Ruscio et al., 2004). Indicators with a negative skew are likely to produce graphs that indicate a high base rate taxon, whereas positively skewed indicators can yield a pseudotaxon with an apparent low base rate. Three of the four PCL–R factors had negative skews that were more than twice the standard error (Affective = −.22, Lifestyle = −.52, Antisocial = −.30; *SE* = .08). Similarly, six of the eight items that Harris et al. (1994) used had substantial negative skew (see Table 1).

Because visual inspection of the taxometric graphs without an accompanying context could produce misleading results, we also used J. Ruscio's (2004) programs to create simulated taxonic and dimensional data sets that matched the parameters of the research data, including the skew of the distribution. This procedure allows for comparisons across graphs produced by the actual and simulated data. Additionally, J. Ruscio's (2004) programs compute goodness-of-fit statistics that quantify whether the graph produced by the research data is closer in appearance to the simulated dimensional or taxonic results (see J. Ruscio et al., 2004). A smaller root-mean-square residual (Fit_{RMSR}) index indicates better fit (see J. Ruscio et al., 2004). Finally, because multiple sets of simulated data can be created, Cohen's *d* is computed using the mean difference between the dimensional and taxonic fit values across the replications. Dimensional fit values are subtracted from taxonic values, so that a positive result suggests a dimensional structure, whereas a negative value suggests a taxonic structure (J. Ruscio, 2004). Ten sets of simulated dimensional data and 10 sets of simulated taxonic data were generated for each of the analyses.

Because our sample consisted of both prison inmates and individuals who were court ordered into residential drug treatment programs, we also repeated these analyses for each of the two subgroups in addition to performing taxometric analyses on the entire sample. The results of these separate analyses were almost uniformly consistent with those from the entire sample, so in the

Table 1
Descriptive Statistics for the Indicators

Variable	Skew[a]	Validity (*SD*)[b]	Validity (*SD*)[c]
Facet scores			
1. Interpersonal	0.088	1.48	1.46
2. Affective	−0.223	1.45	1.60
3. Lifestyle	−0.522	1.09	1.31
4. Antisocial	−0.296	1.18	1.24
Harris et al. (1994) PCL–R items			
3	−0.617	0.72	0.78
5	−0.429	0.86	0.87
8	−0.310	1.17	1.28
9	−0.273	0.65	0.79
12	−0.026	0.82	0.88
13	−0.141	0.76	0.75
14	−0.478	0.56	0.68
15	−1.356	0.48	0.61

Note. PCL–R = Psychopathy Checklist—Revised.
[a] The standard error for these skew coefficients is .083. [b] These validity estimates were calculated on the basis of a cut score of 30 on the PCL–R total score. [c] These validity estimates were calculated on the basis of a cut score of 25 on the PCL–R total score.

interest of parsimony and increased statistical power, only the results from the entire sample are reported below.[2]

Analysis of Factor-Level Indicators

To check for nuisance covariance (i.e., correlations among the indicators within either the taxon or the complement; Meehl & Golden, 1982), we computed correlations among these four factors for those participants who scored 30 or above (the standard cut score for diagnosing psychopathy) on the PCL–R and those who scored below 30. There was little nuisance covariance among these indicators for those participants who met the cut score for psychopathy ($r = -.08$) and an acceptable level of covariance for those in the putative complement ($r = .22$). The average correlation among the four indicators in the entire sample was .36. Using this same cut score, we estimated the average indicator validity to be 1.30, which exceeds the minimally acceptable value of 1.25 recommended by Meehl (1995). Using the slightly less conservative cut score of 25 suggested by Harris et al. (1994), we found that the average indicator validity value was 1.40 (see Table 1).

The four MAMBAC curves are presented in Figure 1. As can be seen, none of these curves evidenced an inverted-U shape that would be consistent with a taxonic fit for the data. On the contrary, all four curves have a concave appearance that is typical of a dimensional latent structure. The average base rate estimate was .59 ($SD = .09$), with a range of .49 to .68.

Figure 2 displays the average of these four MAMBAC graphs juxtaposed with the graphs for the simulated taxonic and dimensional data sets. The average MAMBAC graph produced by our research data looked much more like the graph produced by the simulated dimensional data than like the graph produced by the simulated taxonic data. In fact, the average MAMBAC curve fit the simulated dimensional data ($\text{Fit}_{\text{RMSR}} = .009$) better than the simulated taxonic data ($\text{Fit}_{\text{RMSR}} = .023$). Cohen's d for the 10 replications was 6.10.

Figure 3 presents the four MAXEIG curves, which also show no evidence of a taxonic structure. Instead, these curves are consistent with a dimensional structure, especially one that would be produced by negatively skewed indicators. The average base rate estimate was .78, with a much narrower range of .74 to .82 ($SD = .04$). Although these are relatively stable, they are far higher than would be expected given the MAMBAC average base rate. Figure 4 presents the average MAXEIG curve along with the curves from the simulated data. Once again, the research data were more consistent with the simulated dimensional curve ($\text{Fit}_{\text{RMSR}} = .079$) than with the curve generated by the simulated taxonic data ($\text{Fit}_{\text{RMSR}} = .125$). Cohen's d for the 10 replications was 1.69.[3]

Finally, the L-Mode graph (see Figure 5) bore little resemblance to the bimodal curve one would expect in a taxonic structure. In contrast, the simulated taxonic data produced a clearly bimodal curve. Averaging the base rate estimates, the left and right modes yielded a .48 base rate estimate, and the base rate estimate based on the classification of cases was .49. L-Mode base rate estimates of about .50 are typical for dimensional constructs (A. M. Ruscio, Ruscio, & Keane, 2002; Waller & Meehl, 1998), and both estimates differ from the estimates provided by MAMBAC and MAXEIG. They are also higher than would be expected in this sample if psychopathy were underpinned by a taxon roughly consistent with a PCL–R cut score of 30.

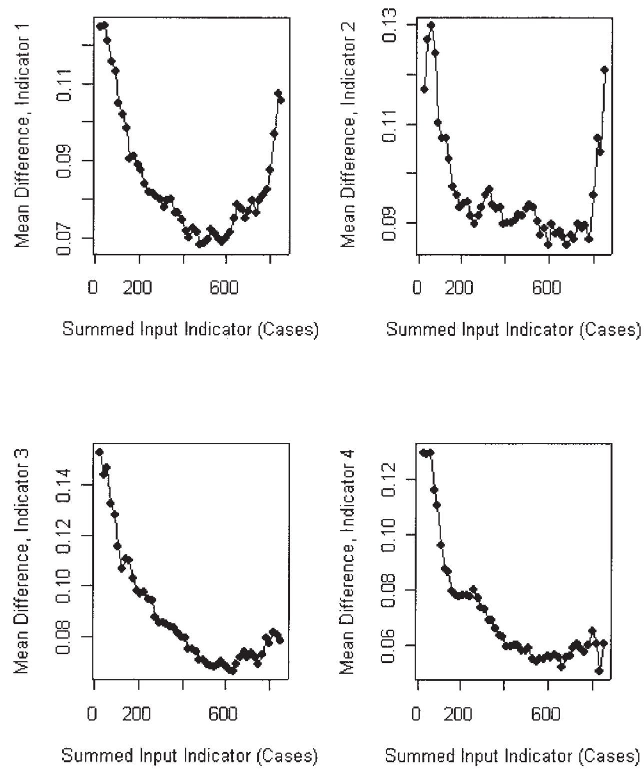

Figure 1. Mean above minus below a cut (MAMBAC) curves for the four Psychopathy Checklist—Revised facet scores: Interpersonal (Indicator 1), Affective (Indicator 2), Lifestyle (Indicator 3), and Antisocial (Indicator 4). The input indicator for each curve (x-axis) was created by combining the three facet scores that were not the output indicator. The data were sorted by the scores on this input indicator, and 50 cuts were made along this input indicator. The y-axis depicts the differences between the mean scores on the output indicator of individuals falling above and below each cut. To stabilize the shape of each curve, we replicated these MAMBAC analyses 10 times by randomly shuffling the cases with equal scores on the input indicator and recalculating the difference scores on the output indicator (J. Ruscio et al., 2004).

Attempted Replication of Harris et al. (1994)

As noted above, for the second set of analyses, we used the same set of eight indicators examined in the Harris et al. (1994) study (PCL–R Items 3, 5, 8, 9, 12, 13, 14, and 15). To check for nuisance covariance (i.e., correlations among the indicators within either the taxon or the complement), we computed correlations among these eight items for those above and below the PCL–R cut score of 30 (Meehl & Golden, 1982). There was little nuisance correlation for

[2] Copies of the set of taxometric graphs produced for each subgroup are available from John F. Edens.

[3] Because there are multiple methods for simulating data, we also used Fraley's program (see Hankin et al., 2005) to simulate data for a set of MAXCOV analyses. These graphs were quite similar to those produced by J. Ruscio's (2004) program. Once again, the results of the dimensional simulation were also much closer to the research data than were the results of the taxonic simulation. Copies of all of these graphs are available from John F. Edens.

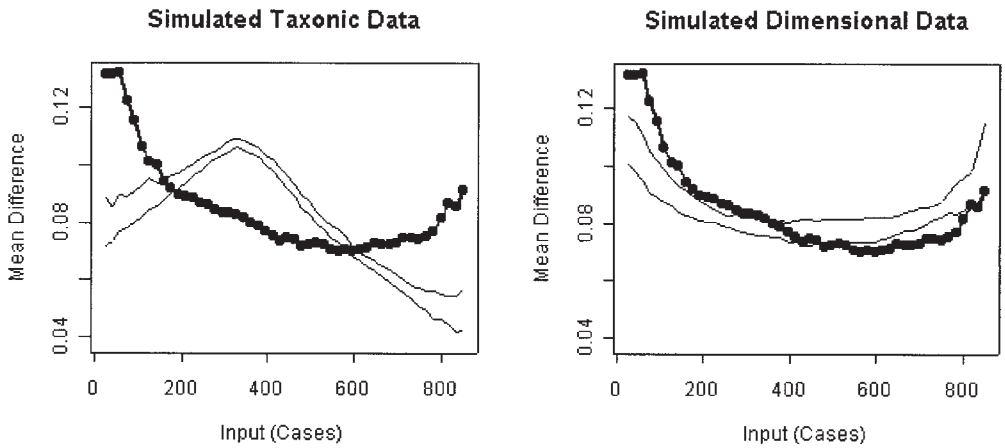

Figure 2. Average mean above minus below a cut curves for the research data, simulated taxonic data, and simulated dimensional data for the four Psychopathy Checklist—Revised facet scores. The graphs for the simulated taxonic and dimensional data were produced by generating 10 data sets for each latent structure. The darker lines represent the actual data, and the lighter lines represent one standard deviation above and below the average for each simulated data set.

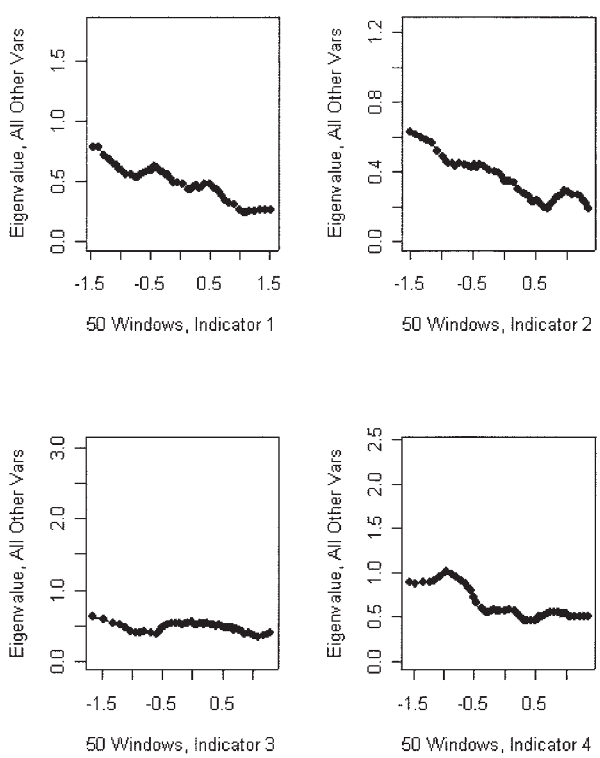

Figure 3. Maximum eigenvalue (MAXEIG) curves for the four Psychopathy Checklist—Revised facet scores: Interpersonal (Indicator 1), Affective (Indicator 2), Lifestyle (Indicator 3), and Antisocial (Indicator 4). The data were sorted along the *x*-axis by the scores on the input indicator and then grouped into 50 subsamples using overlapping windows (.90 overlap). The remaining indicators were factor analyzed, and the eigenvalue of the first principal factor was plotted on the *y*-axis. To stabilize the shape of each curve, we replicated these MAXEIG analyses 10 times by randomly shuffling the cases with equal scores before making the cut on the input indicator and recalculating the eigenvalues on the output indicator (J. Ruscio et al., 2004). Vars = Variables.

those above the cut, with an average correlation among these eight items of .04. The average correlation below the cut was also acceptable ($r = .17$). However, the overall correlation among these eight items in our entire sample was not especially high ($r = .22$). More important, these eight items did not evidence acceptable levels of indicator validity, averaging only 0.75 (see Table 1), which is considerably smaller than the level recommended by Meehl (1995). Given our attempt to replicate the findings of Harris et al. (1994), we conducted taxometric analyses on these items despite the poor indicator validity estimates. Such poor indicator validities call into question the generalizability of Harris et al.'s (1994) findings, especially given that our administration of the PCL–R yielded results that were more consistent with the standardization sample in terms of corrected item-to-total correlations.

The curves resulting from these eight indicators were more consistent with a dimensional than a taxonic structure. Figure 6 provides the average of the eight MAMBAC curves as well as the curves for the simulated taxonic and dimensional data. The average MAMBAC curve for these analyses failed to demonstrate the inverted-U shape that would be expected for taxonic data and was much more similar to the dimensional data ($Fit_{RMSR} = .006$) than to the taxonic data ($Fit_{RMSR} = .016$). Cohen's *d* for the 10 replications was 5.53. Furthermore, the average base rate estimate from these curves was .69 ($SD = .11$), which is much higher than would be expected in this sample and markedly inconsistent with the results of Harris et al. (1994). The estimates ranged from .58 to .94.

Figure 7 presents the average curve generated for the MAXCOV analyses. This curve does not display an inverted-U shape that would be consistent with a taxon. Instead, this curve was more consistent with the simulated dimensional curve ($Fit_{RMSR} = .022$) than with the curve generated by the simulated taxonic data ($Fit_{RMSR} = .088$). Cohen's *d* for the 10 replications was 5.49. Furthermore, at best only 5 of the 28 individual curves appeared as

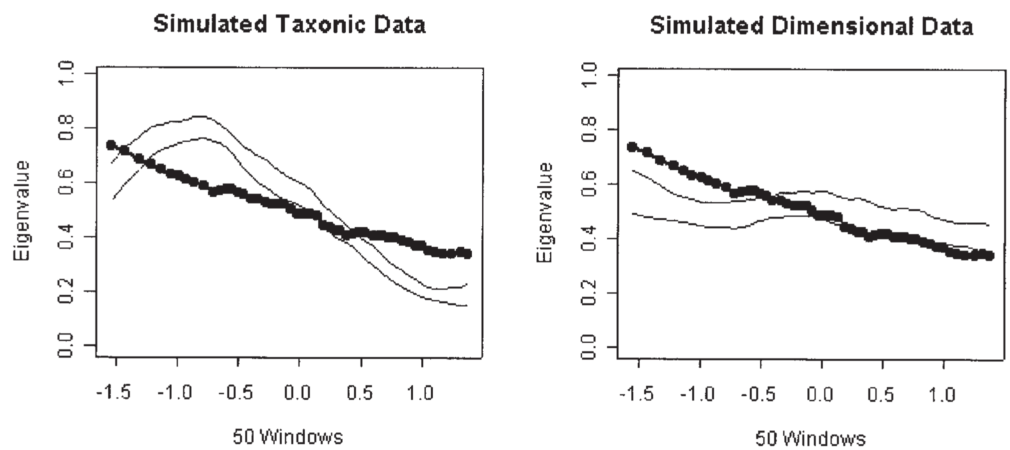

Figure 4. Average maximum eigenvalue curves for the research data, simulated taxonic data, and simulated dimensional data for the four Psychopathy Checklist—Revised facet scores. The graphs for the simulated taxonic and dimensional data were produced by generating 10 data sets for each latent structure. The darker lines represent the actual data, and the lighter lines represent one standard deviation above and below the average for each simulated data set.

if they might be consistent with a taxonic structure.[4] There was also no clear pattern to the taxonic curves that might have suggested that particular indicators were especially valid. The average base rate estimate (.67) was consistent with the average from the MAMBAC analyses, but these estimates were not at all consistent with one another, ranging from .24 to .94 ($SD = .23$). They were also inconsistent, on average, with the base rates reported by Harris et al. (1994).

Finally, unlike the L-Mode graph for the simulated taxonic data, despite a slight hitch, the L-Mode graph for the research data (see Figure 8) did not appear to be bimodal. Averaging the base rate estimates from the left and right modes yielded a .39 base rate estimate, and the base rate estimate based on the classification of cases was .45. Although these two estimates were fairly consistent, they were not at all consistent with the much higher estimates yielded by MAMBAC and MAXCOV.

Overall, despite using the same indicators as Harris et al. (1994), the results of these taxometric procedures seem quite inconsistent with a taxonic structure. Although this finding is to be expected given the very poor indicator validity of these eight items, the point of these analyses is that it was not possible to replicate Harris et al.'s (1994) findings despite using the same indicators and procedures.[5]

Discussion

Across multiple taxometric procedures developed by Meehl and his colleagues (Meehl & Yonce, 1994; Waller & Meehl, 1998)—MAMBAC, MAXEIG (or MAXCOV), and L-mode—our analyses failed to offer support for the contention that psychopathy, as identified by the PCL–R, is underpinned by a latent taxon. None of the taxometric graphs appeared taxonic (i.e., the MAMBAC and MAXEIG curves lacked an inverted-U shape, and the L-Mode curves were unimodal), and the base rate estimates derived from our analyses generally were inconsistent within and across procedures, further suggesting that a taxonic solution is implausible. Additionally, although it is difficult to know what base rate one would have anticipated in our sample if psychopathy were taxonic, the average base rates reported here seem to be remarkably high, especially for the MAXCOV analyses. Such results are difficult to reconcile with the prevailing notion that primary or "Cleckley" psychopaths (Cleckley, 1988), who are ostensibly assessed by the PCL–R, represent a narrow subset of offenders nested with the heterogeneous category of persons with significant histories of antisocial conduct (Hare, 2003; Lykken, 1995). Finally, our analyses did not reveal taxonicity for the PCL–R antisocial items identified by Harris et al. (1994) as taxonic.

Harris et al.'s (1994) article declaring psychopathy to be taxonic has been highly influential (a Web of Science search revealed that this article has been cited over 140 times). Nevertheless, our findings, in conjunction with those for a self-report measure of psychopathy (the PPI) that correlates moderately with the PCL–R (Marcus et al., 2004), cast serious doubts on their conclusions. Although the reasons for the striking discrepancy between our findings and those of Harris et al. (1994) are unclear, their reliance on file data alone to score the PCL–R, unusually high item-to-total score correlations, dichotomous approach to scoring the PCL–R, and inclusion of a large number of offenders who had been adjudicated NGRI are likely candidates. The lattermost point raises the possibility that Harris et al. (1994) may have inadvertently detected a taxon for schizotypy. Schizotypy has been found to be

[4] Copies of all 8 MAMBAC curves and all 28 MAXCOV curves for these analyses are available from John F. Edens.

[5] In addition to eight Harris et al. (1994) items, we also performed a set of analyses using the eight items that correlated most highly with the PCL–R total score in our sample (Items 1, 2, 4, 5, 6, 7, 8, and 10). Although these indicators yielded slightly better validity estimates overall, they still were unacceptably low (1.02), calling into question the appropriateness of using individual PCL–R items for taxometric analyses. Similar to our analyses of the items used by Harris et al. (1994), the analyses were inconsistent with a taxonic latent structure. Copies of these graphs and results are available from John F. Edens.

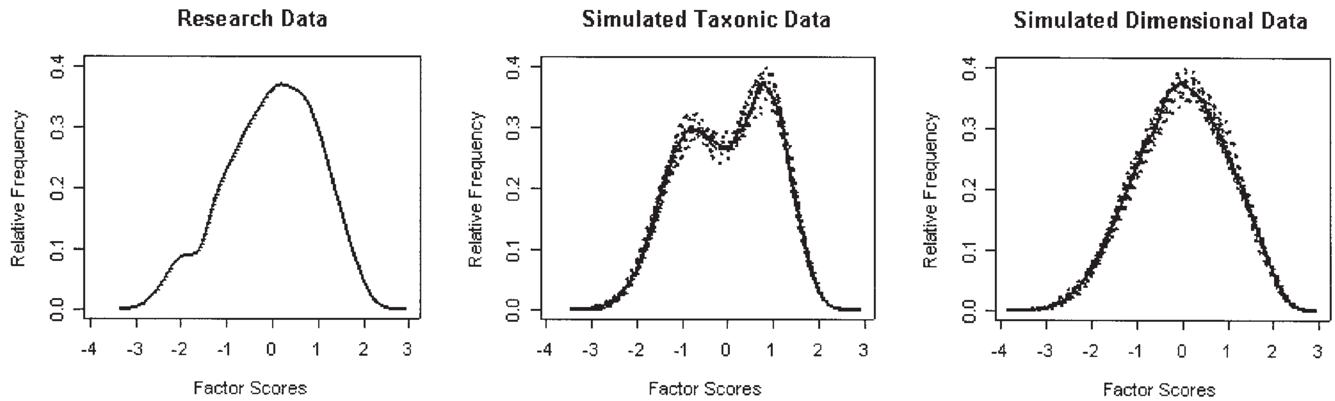

Figure 5. Latent mode factor analysis curves for the four Psychopathy Checklist—Revised facet scores and for the simulated taxonic and dimensional data sets. Each graph displays the frequency distribution of scores on the first factor of a factor analysis of the indicator set. The graphs for the simulated taxonic and dimensional data were produced by generating 10 data sets for each latent structure (dotted lines). The solid lines indicate the average of these data sets.

categorical in numerous studies using taxometric procedures (e.g., Lenzenweger & Korfine, 1992; see Haslam, 2003, for a review), and, as noted earlier, psychopathy measures may misclassify at least some individuals with schizophrenia spectrum disorders as psychopathic (Howard et al., 1984). Although Harris et al. (1994) found evidence for taxonicity even after excluding all participants with a *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; American Psychiatric Association, 1980) diagnosis of psychotic disorder in subsidiary analyses, it is possible that a substantial number of individuals with schizophrenia spectrum disorders (e.g., schizotypal personality disorder, paranoid personality disorder) remained in their sample even following this exclusion (e.g., over 100 participants who were adjudicated NGRI were included in the analyses that excluded psychotic inmates).

Furthermore, evidence suggests that taxometric analyses of rating scale data can be influenced substantially by the perceptions of the persons conducting the ratings (Beauchaine & Waters, 2003). Along these lines, it is possible that the raters in our study differed in some important respect from the raters who coded the archival file data in the Harris et al. (1994) study. One possibility is that the

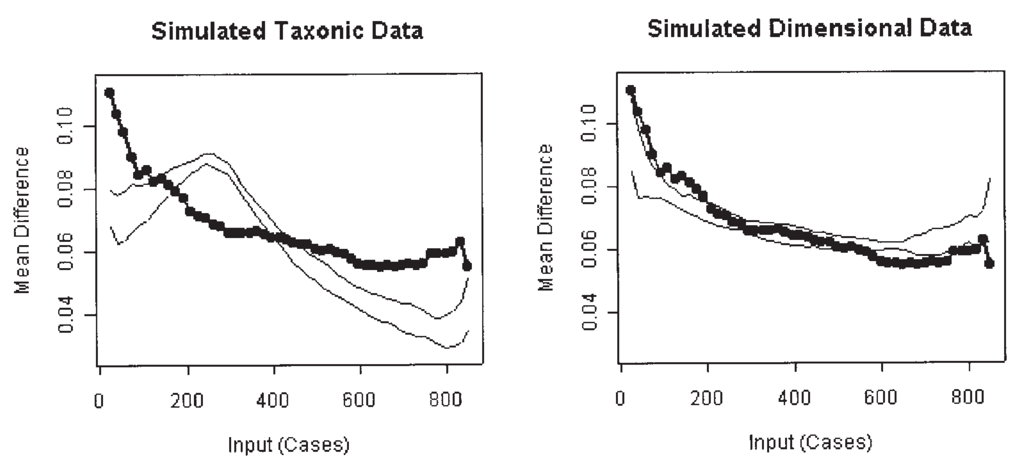

Figure 6. Average mean above minus below a cut (MAMBAC) curves for the research data, simulated taxonic data, and simulated dimensional data for the eight Psychopathy Checklist—Revised items used as indicators by Harris et al. (1994). The input indicator for each curve (x-axis) was created by combining the seven items that were not the output indicator. The data were sorted by the scores on this input indicator, and 50 cuts were made along this input indicator. The y-axis depicts the differences between the mean scores on the output indicator of individuals falling above and below each cut. To stabilize the shape of each curve, we replicated these MAMBAC analyses 10 times by randomly shuffling the cases with equal scores on the input indicator and recalculating the difference scores on the output indicator (J. Ruscio et al., 2004). The graphs for the simulated taxonic and dimensional data were produced by generating 10 data sets for each latent structure. The darker lines represent the actual data, and the lighter lines represent one standard deviation above and below the average for each simulated data set.

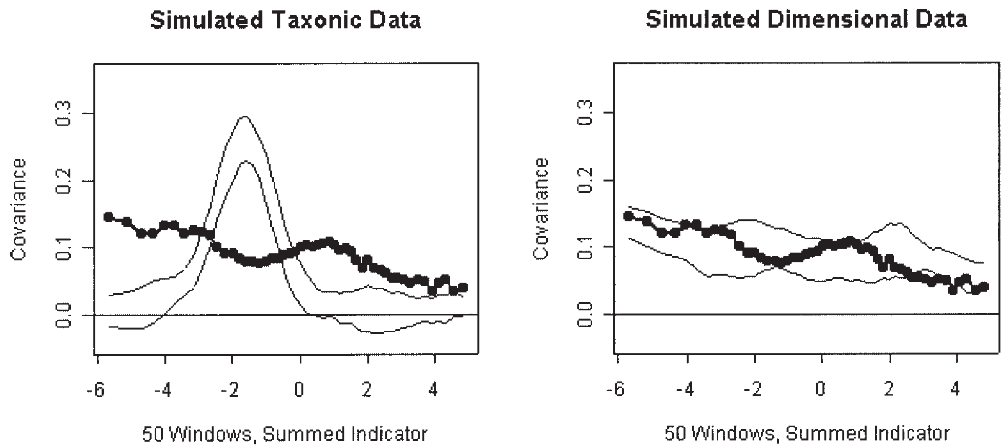

Figure 7. Average maximum covariance (MAXCOV) curves for the research data, simulated taxonic data, and simulated dimensional data for the eight Harris et al. (1994) indicators. The data were sorted along the *x*-axis by summing six of the indicators (i.e., the input indicator) and then grouped into 50 subsamples using overlapping windows (.90 overlap). The covariance between the two other indicators was then was plotted on the *y*-axis. This procedure produced 28 graphs that were averaged to create this graph. To stabilize the shape of each of the 28 curves, we replicated these MAXCOV analyses 10 times by randomly shuffling the cases with equal scores before making the cut on the input indicator and recalculating the covariance on the output indicator (J. Ruscio et al., 2004). The graphs for the simulated taxonic and dimensional data were produced by generating 10 data sets for each latent structure. The darker lines represent the actual data, and the lighter lines represent one standard deviation above and below the average for each simulated data set.

interview information obtained from participants in our study led to more nuanced ratings of the underlying dimensional construct assessed by the PCL–R. Alternatively, one could argue that such information might blur an underlying categorical distinction that is more evident when relying exclusively on file data. The fact that Harris et al. (1994) obtained item-to-total correlations that were unusually high (relative to our data and the data reported in the PCL–R manual) would seem more consistent with the former interpretation rather than the latter, although this is by no means conclusive evidence. To our knowledge, no one has experimentally manipulated a priori beliefs of PCL–R examiners to test systematically whether or how their ratings could affect taxometric analyses, although this would be a fruitful area of future investigation.

Our failure to identify a latent taxon has important implications for the etiology and assessment of this disorder, particularly as other investigators also find evidence of a dimensional latent structure using the PCL–R (see, e.g., Guay, Ruscio, Hare, & Knight, 2005; Looman & Abracen, 2005). If psychopathy were dimensional at a latent level, it would imply that researchers should direct more of their investigative efforts toward etiological agents that are themselves dimensional, such as fearlessness

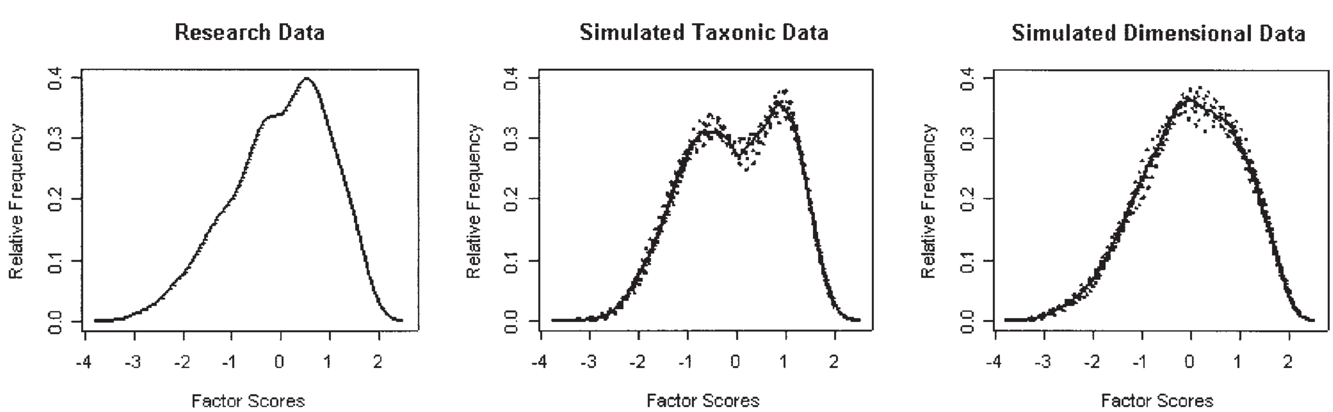

Figure 8. Latent mode factor analysis curves for the eight Harris et al. (1994) indicators and for the simulated taxonic and dimensional data sets. Each graph displays the frequency distribution of scores on the first factor of a factor analysis of the indicator set. The graphs for the simulated taxonic and dimensional data were produced by generating 10 data sets for each latent structure (dotted lines). The solid lines indicate the average of these data sets.

(Lykken, 1995), role-taking deficiency (Gough, 1960), graded deficits in response modulation (Patterson & Newman, 1993), or other continuously distributed source traits (Cattell, 1951) that could give rise to the manifold surface traits of psychopathy. Our findings are also potentially consistent with models positing that psychopathy is a constellation or configuration of extreme scores on several continuously distributed personality dimensions, such as low conscientiousness and low agreeableness (Benning et al., 2005; J. D. Miller et al., 2001), although they do not provide support for any particular dimensional model of psychopathy, such as the five-factor model (Lynam & Widiger, 2001). Conversely, our findings run counter to the position that psychopathy is underpinned by a dichotomous causal agent (Meehl & Golden, 1982) and offer little support for developmental models (e.g., Quinsey, Skilling, Lalumiere, & Craig, 2004) that posit the existence of a qualitatively distinct or discrete natural class of psychopathic youths. We should note that, although our results are generally inconsistent with etiological models based on dichotomous causal factors, they do not address whether the etiology of psychopathy is similar to or appreciably different from other externalizing disorders.

Our results also dovetail with structural equation models suggesting that various phenotypically diverse psychological conditions and characteristics, including constructs closely related to PCL–R Factor 2, are underpinned by a latent externalizing dimension (e.g., Krueger et al., 2002; Krueger, Markon, Patrick, & Iacono, 2005; see also Gorenstein & Newman, 1980). Moreover, the latent structure models used by Krueger and colleagues address the continuous versus categorical question in a manner quite distinct from the taxometric procedures of Meehl (Meehl & Yonce, 1994; Waller & Meehl, 1998), in that they "involve fitting explicit mathematical models to sample data by use of well-characterized estimators of population parameters (e.g., maximum likelihood) and evaluating the fit of these models by use of quantitative indices of fit" (Krueger et al., 2005, p. 540). Future research applying these methods to the psychopathic traits assessed by the PCL–R would be informative because (a) replication of the dimensional structure of psychopathy via separate statistical procedures would bolster the present findings, (b) there is a need to integrate taxometrics with other latent-variable techniques (Waldman & Lilienfeld, 2001), and (c) these latent structure models can address issues of comorbidity with other forms of externalizing psychopathology.

On the assessment front, our findings call into question the widespread practice of dichotomizing or trichotomizing total scores on the PCL–R to establish discrete groups (Lilienfeld, 1994). If psychopathy were dimensional at a latent level, this practice would yield scientifically arbitrary groupings of individuals who differ along one or more continuous dimensions. Moreover, this practice would result in eliminating potentially useful assessment information regarding individuals' standing on these dimensions. From the perspective of Gangestad and Snyder (1985), the class of psychopaths may be a *phenetic* category, that is, an arbitrarily formed grouping that possesses no intrinsic meaning (e.g., professional basketball player may be a category, but there is no natural relationship between height and athletic ability). Of course, our findings do not imply that distinctions between psychopaths and nonpsychopaths cannot be made for practical purposes, such as risk management or violence prediction. If other research teams replicate our findings, however, this would imply that such distinctions are purely pragmatic and do not "carve nature at its joints," because in the case of psychopathy, there are no discrete joints to carve.

It is perhaps worth noting, however, that even if psychopathy were shown to be taxonic, this would not necessarily lend support to the current PCL–R cutoff of 30, as this score may not correspond to the maximally predictive threshold (Meehl & Golden, 1982) for distinguishing the psychopathy taxon from the nonpsychopathy complement group (cf. Gacono, Loving, & Bodholdt, 2001). Moreover, dichotomizing continuous distributions typically results in a loss in statistical power (Cohen, 1983; McCallum, Zhang, Preacher, & Rucker, 2002; but see Farrington & Loeber, 2000, for exceptions), unless there are clear-cut violations of normality in the data. Given that there does not appear to be a clear breaking point in the observed distributions of psychopathy scores, such dichotomization is difficult to justify on strictly scientific grounds, although it may often simplify the presentation of research findings (Farrington & Loeber, 2000).

From a policy perspective, there is ample evidence that the legal system is keenly interested in the identification of psychopaths, based on the amount of legislation in North America and Europe that uses this or related phrases such as *dangerous and severe personality disorders* (see Edens & Petrila, 2006, for an overview). Similarly, references to psychopathy in case law often involve debates as to whether a defendant is in fact "a psychopath" (i.e., PCL–R $\geq$ 30). Such views appear to assume a categorical perspective, in that the intent is to identify a class of individuals deemed appropriate for some form of legal sanction because of their mental disorder or behavioral abnormality (Edens, in press). To the extent that our results undermine the implicit or explicit legal presumption that psychopaths are a discrete category of criminals, they suggest that it is largely arbitrary to draw precise categorical boundaries between psychopathic and nonpsychopathic offenders. Although decision makers can and do use PCL–R scores to inform legal decisions that are by definition categorical (e.g., presence or absence of a behavioral abnormality, indeterminate commitment), there is no clear scientific evidence for a natural breaking point at which such categories should be defined regarding psychopathy. That said, our results by no means argue against the possibility of using particular cut scores that may prove valuable for any number of pragmatic decisions (e.g., optimal cut scores to reduce community violence among released offenders), which may not necessarily correspond to any putative diagnostic thresholds (see, e.g., Skeem & Mulvey, 2001).

One limitation of our study, like that of Harris et al. (1994), Skilling et al. (2001), and the vast majority of taxometric studies across constructs, is mono-operation bias (Shadish, Cook, & Campbell, 2002). As Meehl and Golden (1982) noted, taxometric analyses are ideally performed on indicators derived from maximally independent domains. In contrast, all of our indicators derived from one well-validated measure, namely, the PCL–R. It is conceivable that alternative indicators that tap processes that may be etiologically relevant to psychopathy, such as poor passive-avoidance learning (Lykken, 1957; Schacter & Latane, 1964), poor response modulation (Patterson & Newman, 1993), weak fear potentiated startle (Patrick, Bradley, & Lang, 1993), or diminished electrodermal classical conditioning to aversive stimuli (Lykken, 1957), could yield evidence for the taxonicity of psychopathy. Such presumed "endophenotypic markers" (Gottesman & Gould,

2003) should be incorporated in future investigations of the taxonicity of psychopathy, in part because they may reflect certain processes that are more closely tied etiologically to psychopathy and in part because they should help to obviate the problem of method covariance introduced by an exclusive reliance on a single measure (e.g., the PCL–R, the PPI). Nevertheless, one should not necessarily assume that biological or laboratory markers are necessarily endophenotypic as opposed to exophenotypic (hence, our use of the term *presumed* in the previous sentence), as they could reflect the action of processes (e.g., attentional deficits) that lie causally downstream from psychopathy. Moreover, at least some of these markers appear to be relatively nonspecific; for example, deficits in passive-avoidance learning have been reported not only in psychopathic individuals but also in individuals with borderline personality disorder (Hochhausen, Lorenz, & Newman, 2002).

In summary, our findings contrast sharply with those of Harris et al. (1994) and fail to offer support for the view that psychopathy assessed by the PCL–R is underpinned by a discrete taxon. Our results are consistent with recent calls for closer research linkages between the often-disconnected domains of personality and psychopathology (e.g., Benning et al., 2005; Harkness & Lilienfeld, 1997; Krueger & Tackett, 2003; Lynam & Widiger, 2001). Moreover, they are encouraging in that they suggest that researchers ultimately may be able to draw from the large body of research on the assessment and causes of continuously distributed personality traits to better inform their understanding of psychopathy among criminal populations.

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