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Paralimbic Biomarkers in Taxometric Analyses of Psychopathy: Does Changing the Indicators Change the Conclusion?

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Abstract

Although psychopathy has consistently been shown to distribute as a dimension, all prior studies have examined behavioral indicators that may be phenotypically distant from core biological correlates of the syndrome. The current studies attempted to determine whether biomarkers from a high-resolution structural magnetic resonance imaging (MRI) scan of selected limbic and paralimbic structures identified the latent structure of psychopathy as continuous. Participants were 254 adult male medium/maximum security inmates (Study 1) and 191 adolescent male maximum security inmates (Study 2) who volunteered to undergo research MRI scans. Indicators of gray matter concentration (GMC) in the adult sample and of gray matter volume (GMV) in the adolescent sample were subjected to taxometric analysis using three non-redundant taxometric procedures: mean above minus below a cut (MAMBAC), maximum covariance (MAXCOV), and latent-mode factor analysis (L-Mode). Evidence of continuous latent structure was found across samples (adults, adolescents), measures (GMV, GMC, Psychopathy Checklist-Revised [PCL-R], Psychopathy Checklist: Youth Version [PCL: YV]) and procedures (MAMBAC, MAXCOV, L-Mode). Continuous latent structure was also noted when biomarker (GMV, GMC) and behavioral (PCL) indicators were included in the same analysis. The current results support the view that psychopathy is a quantitative construct on which people differ in degree (“more of” or “less of”) rather than a qualitative construct that assigns people to distinct categories (“either or”). Continued development of the psychopathy construct may depend on our ability to identify, understand, and make effective use of its apparent continuous latent structure.

Keywords

taxometric; psychopathy; paralimbic; biomarkers

Psychopathy is an important theoretical construct characterized by interpersonal, affective, behavioral, and neurodevelopmental elements that can be assessed with the Psychopathy Checklist-Revised (PCL-R: Hare, 2003) and its derivatives, the Psychopathy Checklist: Screening Version (PCL: SV: Hart, Cox, & Hare, 1995) and the Psychopathy Checklist: Youth Version (PCL: YV: Forth, Kosson, & Hare, 2003). In discovering how these elements interrelate to form the foundation for what will eventually become psychopathic personality disorder, it is vital that we identify the construct's underlying latent structure for this provides a blueprint of how the construct is organized.

By clarifying how a construct is organized, latent structure holds important implications for theory, research, and practice (Ruscio, Haslam, & Ruscio, 2006). In a theoretical context latent structure tells us whether a construct assigns cases to categories or places them along a continuum. In a research context latent structure informs us as to whether or not studies on subthreshold cases can provide useful information. In a clinical context latent structure provides practical guidelines for classification, assessment, and treatment. Meehl's (1995, 2004) taxometric method is one way of differentiating between continuous latent structure in constructs like intelligence (excluding non-familial mental retardation) and categorical latent structure in constructs like sex and the neurodegenerative genetic disorder, Huntington's disease (Ruscio et al., 2006). In standard taxometric practice, results from three quasi-independent and non-redundant procedures—mean above minus below a cut (MAMBAC), maximum covariance (MAXCOV), and latent-mode factor analysis (L-Mode)—are compared to determine whether the results are consistent with categorical latent structure, continuous latent structure, or neither (Meehl, 2004). Ruscio, Ruscio, and Meron (2007) improved on Meehl's method by using simulated data to create categorical and continuous models that are compared to the actual data curve by means of the comparison curve fit index (CCFI). The results of several Monte Carlo studies document the accuracy of Ruscio et al.'s comparison curve approach to taxometric research (McGrath & Walters, 2012; Ruscio & Marcus, 2007; Ruscio, Walters, Marcus, & Kaczetow, 2010; Walters, McGrath, & Knight, 2010; Walters & Ruscio, 2009, 2010).

Harris, Rice, and Quinsey (1994) performed what is generally considered the inaugural taxometric study on psychopathy. Subjecting dichotomized items from the PCL-R to MAXCOV analysis in a sample of 653 mentally disordered offenders, Harris et al. (1994) found evidence of categorical latent structure in the PCL-R total and Factor 2 (impulsive and antisocial features) scores but not in the Factor 1 (interpersonal and affective features) score. On the basis of these results Harris et al. (1994) concluded that a psychopathy taxon exists and that psychopaths are categorically distinct from non-psychopaths. This conclusion needs to be tempered, however, by the fact that this study suffered from several serious methodological flaws. First, Harris et al. (1994) used only one of the three available taxometric procedures (MAXCOV), thus limiting their opportunities for consistency testing. Second, the study made use of dichotomous indicators. Subsequent research has shown that indicators composed of fewer than four ordered categories produce unreliable and largely inaccurate results (Walters & Ruscio, 2009). Third, Harris et al. (1994) evaluated latent structure type using criteria (e.g., base rate consistency, Bayesian estimates of taxon membership) that were subsequently discredited (Ruscio, 2007). Another procedure, Ruscio

et al.'s (2007) comparative curve fit approach, which was not available at the time Harris et al. conducted their research, has been found to be more accurate than the procedures Harris et al. (1994) used.

Since the original Harris et al. (1994) investigation, there have been several taxometric studies performed in an attempt to gain a better understanding of the latent structure of psychopathy. Most of these studies have used Ruscio's comparative fit approach and a variety of quasi-continuous indicators composed of four or more ordered categories. These indicators include the four facet scores from the PCL-R (Edens, Marcus, Lilienfeld, & Poythress, 2006; Guay, Ruscio, Knight, & Hare, 2007; Walters, Duncan, & Mitchell-Perez, 2007; Walters, Marcus, Edens, Knight, & Sanford, 2011), PCL:SV (Walters, Gray, Jackson, Sewell, Rogers, Taylor, & Snowden, 2007), and PCL:YV (Murrie, Marcus, Douglas, Lee, Salekin, & Vincent, 2007) as well as individual scales from popular self-report measures of psychopathy (Edens, Marcus, & Vaughn, 2011; Guay & Knight, 2004; Marcus, John, & Edens, 2004; Murrie et al., 2007; Walters, Brinkley, Magaletta, & Diamond, 2008). In each of these studies consistent evidence for continuous latent structure was found. Although several taxometric investigations have uncovered evidence of a psychopathy taxon (Coid & Yang, 2008; Harris, Rice, Hilton, Lalumière, & Quinsey, 2007; Skilling, Quinsey, & Craig, 2001; Vasey, Kotov, Frick, & Loney, 2005), each of these studies suffered from one or more of the methodological shortcomings mentioned previously with respect to the Harris et al. (1994) study: (a) use of a single taxometric procedure; (b) use of severely limited (dichotomous, trichotomous, or redundant) indicators; and (c) use of non-evidence based criteria for latent structure, such as base rate consistency and a Goodness of Fit Index (GFI) > .90 between the observed and predicted covariance matrices.

Quantitative reviews indicate that 80% of the taxometric studies on externalizing behavior in general (Haslam, Holland, & Kuppens, 2012) and 75% of the taxometric studies on psychopathy in particular (Haslam, 2011) support the conclusion of continuous latent structure. None of these studies, however, included biological markers of externalizing behavior or psychopathy. To the authors' knowledge only two taxometric studies have used physical or biological indicators (Ingram & Kattan, 2010; Rhudy, Green, Arnau, & France, 2008) and neither was on psychopathy. This is surprising because Meehl developed the taxometric method as a reliable means of evaluating his biologically and genetically based theory of schizotypy (Golden & Meehl, 1979). Moreover, if psychopathy is a primary brain disorder, as has been asserted and for which there is now evidence (Decety, Skelle, & Kiehl, 2013; Gregory et al., 2012), then biological markers should be considered independent of and in conjunction with the behavioral and self-report indicators that have thus far been used to assess the latent structure of psychopathy. With this in mind we sought in the current study to investigate the latent structure of psychopathy using biological markers as indicators.

Psychopathy has been found to covary with a number of subtle neuroanatomical abnormalities (Raine & Yang, 2006). In a review of the literature on the behavioral sequelae of paralimbic system damage, Kiehl (2006) concluded that paralimbic dysfunction or hypofunction may be responsible for the language, orienting, and affective processing deficits observed in psychopathy. The neural structures encompassed by the paralimbic

system, including the amygdala, hippocampus, insula, orbital frontal cortex, anterior superior temporal gyrus, and the rostral, caudal, and posterior cingulate, may accordingly play a key role in the development of psychopathic personality disorder. Assessing these areas with high-resolution structural magnetic resonance imaging (MRI) scans of 254 male medium and maximum security inmates, Kiehl and colleagues determined that reduced gray matter volumes (GMV) and concentrations (GMC) in paralimbic regions correlated significantly with the total PCL-R score (Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012). When these same procedures were used with samples of male and female adolescents incarcerated in maximum-security juvenile facilities, reduced GMVs in similar areas of the paralimbic system were found to correlate with psychopathy as measured by the PCL:YV (Cope, Ermer, Nyalakanti, Calhoun, & Kiehl, 2014; Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2013). If these paralimbic and limbic structural deficits act as endophenotypes of psychopathy (Gottesman & Gould, 2003), then their use as indicators in a taxometric analysis may further our understanding of the latent structure of this disorder.

The purpose of this study was to determine whether the latent structure of psychopathy—which most studies suggest is continuous—remains continuous or becomes categorical when paralimbic biomarkers replace the rating scales and self-report inventories that have been used as indicators in all of the taxometric research thus far conducted on psychopathic personality disorder. If psychopathy is truly continuous, then biomarkers of paralimbic dysfunction should produce results similar to those obtained with the PCL family of measures or with the self-report measures that have been examined. If prior research supporting the continuous nature of psychopathy is an artifact of the indicators used, research employing paralimbic indicators may show something other than the consistent evidence for continuous latent structure that has been observed in previous research. Data for the current investigation came from the original Ermer et al. (2012, 2013) studies, with indicators selected to test theoretical models of psychopathy (Kiehl, 2006). The hypothesis tested in both studies held that the latent structure of psychopathy, as measured by indicators of paralimbic system dysfunction or hypofunction, would be continuous rather than categorical in nature.

Study 1

Method

Participants—The original sample for this study consisted of 296 male medium- and maximum-security inmates from several New Mexico prisons evaluated between May 2007 and June 2010 (Ermer et al., 2012). After excluding individuals with traumatic brain injury, mental health disorders and missing data, the sample was reduced to 254 individuals. These 254 inmates had an average age of 33.63 years ($SD = 9.10$, range = 18–60), an average full scale IQ of 96.28 ($SD = 13.78$, range = 66–137), and an average total PCL-R score of 21.27 ($SD = 7.00$, range = 4–38). More than four-fifths of the sample was classified as either Hispanic/Latino (52%) or White/Caucasian (31%).

Measures—The PCL-R is a 20-item rating scale designed to assess the theoretical construct of psychopathy. Each item is rated on a three-point scale (0 = does not apply, 1 = applies somewhat, 2 = definitely applies) to create a total score with a range of 0 to 40, a

Factor 1 (interpersonal and affective features) score that ranges from 0 to 16, and a Factor 2 (impulsive and antisocial features) score that ranges from 0 to 20. Factor 1 can be broken down further into Facets 1 (Interpersonal) and 2 (Affective), and Factor 2 can be broken down further into Facets 3 (Impulsive Lifestyle) and 4 (Antisocial Behavior). Clinical cutoffs of one standard deviation above the mean (total PCL-R = 30; Hare, 2003) for psychopathy and one and one-half standard deviations above the mean (total PCL-R = 34) for severe psychopathy have been proposed for the PCL-R. Approximately 10% of the interviews for the current sample were videotaped and double-rated for a total PCL-R one-way random single measure intraclass correlation coefficient (ICC, 1, 1) of .96.

High-resolution T1-weighted structural MRI scans were performed at the correctional institution where the participant was housed. Gray matter volumes (GMV) and gray matter concentrations (GMC) were estimated using voxel-based morphometry (VBM). Only the GMC scores were used because they correlated higher with the PCL-R and because they provided enhanced opportunities for consistency testing in that only GMV scores were available for the second (adolescent) sample. Fifteen regions of interest (ROIs) in the paralimbic system were pre-selected based on theory (Kiehl, 2006) and identified using anatomical image masks. See Ermer et al. (2012) for full scanning parameters and analytical procedures.

The 15 ROIs were divided into two groups for the purpose of this study. The amygdala-hippocampus-parahippocampal (AHP) group consisted of the mean ROI from the right amygdala, left amygdala, right hippocampus, left hippocampus, right parahippocampal cortex, and left parahippocampal cortex. The temporal pole-orbitofrontal-cingulate-insula (TOCI) group consisted of the mean ROI from the right temporal pole, left temporal pole, right orbitofrontal cortex (OFC), left OFC, medial OFC, anterior cingulate cortex (ACC), posterior cingulate cortex (PCC), right insula, and left insula. Each group was divided further into left and right side ROIs. All six AHP sites and three of the TOCI sites (right temporal pole, left temporal pole, left OFC) achieved significant correlations with the total PCL-R score. One additional site (right OFC), which achieved a borderline significant correlation with the PCL-R ($p = .07$), was added to the TOCI group so that there would be two sites covered by the TOCI right subgroup. The rationale for dividing the ROIs into AHP and TOCI was that AHP covers the core limbic areas and a principal component analysis (PCA) with Varimax rotation of the 15 ROIs was consistent with this breakdown (Factor Loadings = .681–.935 for AHP and .536–.802 for TOCI).

Procedure—Participants were recruited from medium- and maximum-security prisons in New Mexico and provided their informed consent to take part in the original study. They were paid for their participation at a rate commensurate with institutional wages (\$1/hour). The original research was approved by the University of New Mexico Health Sciences Center Institutional Review Board, and the current secondary analysis of these data was approved by the Kutztown University Institutional Review Board. Over a period of several weeks participants completed a battery of tests, spoke to interviewers, and received MRI scans. AHP right, AHP left, TOCI right, and TOCI left served as indicators in the GMC analyses, the four PCL-R facets served as indicators in the PCL analyses, and the total AHP

and TOCI scores along with the PCL-R Factor 1 and 2 scores served as indicators in the GMC-PCL analyses. There were no missing data for any of the indicators.

Taxometric analyses were performed using Ruscio's (2011) computerized taxometric program for R language (R Development Core Team, 2005). Analyses were conducted with three non-redundant and quasi-independent taxometric procedures: mean above minus below a cut (MAMBAC: Meehl & Yonce, 1994), maximum covariance (MAXCOV: Meehl & Yonce, 1996), and latent mode factor analysis (L-Mode: Waller & Meehl, 1998). Before a taxometric analysis can be performed certain requirements must be satisfied. First, all indicators must be quasi-continuous, which means that they should be composed of at least four ordered categories (Walters & Ruscio, 2009). Second, Meehl (1995) held that the mean inter-indicator correlations within the putative taxon and complement groups (nuisance covariance) should fall below .30 and the mean inter-indicator correlation for the entire sample should be at least .30 for the data to be amenable to taxometric analysis. For these analyses, putative taxon and complement groups were created from the average base rate estimates obtained from preliminary MAMBAC and MAXCOV analyses. Third, Meehl (1995) held that each indicator should differentiate between the putative taxon and complement groups at $d = 1.25$.

With MAMBAC, the input indicator scores are sorted from lowest to highest along the x -axis of a graph and the differences between mean scores on the output indicator are then plotted on the y -axis of the graph at regular intervals of the x -axis. A peak on the MAMBAC curve signifies that there is an optimal cutting score that divides the sample into two or more distinct groups, a sign of categorical latent structure. If there is no optimal cutting score, the MAMBAC curve will not peak but will assume a concave or dish-shaped appearance, indicative of continuous latent structure. In the current study, MAMBAC was calculated with 50 equally spaced cuts along a single input indicator and input indicators were summed based on research showing that summed input MAMBAC may be slightly more accurate than standard input MAMBAC (Walters & Ruscio, 2009). In addition, tied scores were resorted 10 times, cases were assigned to the putative taxon and complement groups by means of the base rate classification procedure, and curves were averaged before the fit index was calculated.

MAXCOV is conducted by first arranging the input indicator scores from lowest to highest along the x -axis and then grouping these scores into intervals (non-overlapping subsamples) or windows (overlapping subsamples). A peak will register on the MAXCOV curve if the covariance between two output indicators is higher in one subsample than it is in neighboring subsamples. This peak suggests the presence of a taxonic boundary or categorical construct based on the logic that heterogeneous groups (subsamples composed of an equal number of taxon and complement members) produce higher correlations than homogeneous groups (subsamples composed primarily of either taxon or complement members). A continuous construct, by comparison, will produce a non-peaked MAXCOV curve. In this study MAXCOV was calculated with 25 sliding windows (subsamples) that overlapped 90% with each other based on research showing that sliding windows tend to produce more accurate results than non-overlapping intervals (Walters & Ruscio, 2010). Tied scores on the input indicator were resorted ten times, cases were assigned to the

putative taxon and complement groups using the base rate classification procedure, and the individual MAXCOV curves were averaged before being analyzed.

MAMBAC and MAXCOV originate from a procedure Meehl (2001) referred to as coherent cut kinetics, which means studying the effect of moving a cut over the length of an input indicator. L-Mode, a procedure introduced several years after MAMBAC and MAXCOV by Waller and Meehl (1998), is, as a point of contrast, grounded in factor analysis. Indicator scores in L-Mode, rather than being grouped or correlated, are factor analyzed. Once this is accomplished, the first (and largest) principal factor is extracted and the participant's scores on this one latent factor are plotted using Bartlett's (1937) method of factor score estimation. An L-Mode graph that features two prominent peaks is indicative of categorical latent structure, whereas an L-Mode graph marked by a single bump suggests the presence of continuous latent structure. Because the base rate estimates produced by L-Mode tend to be inflated and unreliable (Walters et al., 2010), base rate estimates used in conducting analyses for all three procedures were calculated by averaging the more reliable estimates produced by MAMBAC and MAXCOV.

Given the fact that taxometric curves tend to vary as a function of certain data characteristics (e.g., skew, kurtosis, putative base rate, indicator correlations), the MAMBAC, MAXCOV, and L-Mode curves were compared to categorical and continuous models using Ruscio et al.'s (2007) simulated data approach and relative fit was computed by means of the comparison curve fit index (CCFI). The CCFI compares the root mean square residual (RMSR) of fit between the data curve and each of the comparison curves. Maximum support for the categorical model is achieved when the CCFI reaches 1.00, and maximum support for the continuous model is achieved when the CCFI falls to .00. Equal support for the categorical and continuous models is suggested by a CCFI of .50. Monte Carlo analyses have demonstrated that the CCFI yields highly accurate results, particularly when values around .50 are classified as ambiguous or uninterpretable (Ruscio et al., 2010). A dual-threshold criterion was used in the current study, meaning that CCFI values of .55 and higher were classified as consistent with categorical latent structure, CCFI values of .45 and lower were classified as consistent with continuous latent structure, and CCFI values between .451 and .549 were classified as indeterminate.

Results

The six GMC paralimbic biomarkers that served as indicators in this study all correlated significantly ($p < .05$) with the PCL-R: $-.23$ (AHP), $-.18$ (TOCI), $-.21$ (AHP right), $-.20$ (AHP left), $-.16$ (TOCI right), and $-.18$ (TOCI left), for a mean correlation of $-.19$.

GMC Analyses—Pre-taxometric analysis of the four GMC indicators revealed that these indicators correlated more than .30 in the full sample and less than .30 in the putative taxon and complement groups, and that they effectively distinguished between the putative taxon and complement groups at or above d 1.25 (see upper portion of Table 1). Visual inspection of the data curve relative to the categorical and continuous comparison curves indicates that the data curve more closely approximated the continuous comparison curve than it did the categorical comparison curve (see Figure 1). These visual comparisons were corroborated

by statistical results from the CCFI (.45) for all three procedures. The mean CCFI, which achieved 98% accuracy in a Monte Carlo analysis (99.4% when scores > .45 and < .55 were treated as indeterminate: Ruscio et al., 2010), supported a conclusion of continuous latent structure (see Table 2).

PCL-R Analyses—A pre-taxometric analysis of the four PCL-R facet scores showed that these indicators correlated above .30 with each other in the full sample but below .30 in the putative taxon and complement groups and achieved separation between the putative taxon and complement groups at or above d 1.25 (see middle portion of Table 1). As indicated by the data summarized in Table 2, a taxometric analysis of the four PCL-R facet scores revealed consistent evidence of continuous latent structure for MAMBAC, MAXCOV, L-Mode, and the mean CCFI.

GMC-PCL Analyses—Indicators for the GMC-PCL analyses were the two AHP and TOCI paralimbic biomarkers and two PCL-R factors scores. All four indicators distinguished between the putative taxon and complement at or above d 1.25 (see lower portion of Table 1). Although there was no evidence of nuisance covariance, indicators correlated only .29 in the full sample, which is slightly below the .30 threshold set by Meehl (1995). Cross-domain correlations ranged from .12 to .20 with a mean value of .18. The taxometric analyses nonetheless consistently supported continuous latent structure (see Table 2).

Discussion

Although these results support the hypothesis that replacing the exophenotypic indicators normally used in taxometric research on psychopathy with endophenotypic indicators did not alter the conclusion that the latent structure of psychopathy is continuous, the six paralimbic biomarkers employed as indicators in this study correlated only .19 with the PCL-R and the mean inter-indicator correlation for the cross-domain analysis was just .29. A second study was therefore conducted on a group of adolescent males in an effort to cross-validate these results.

Study 2

Method

Participants—A group of 191 male juvenile offenders from a maximum security youth detention facility in New Mexico served as participants in this second study. All data were collected between June 2007 and March 2011. Out of the 218 male juvenile offenders who received brain scans, 18 were dropped from the study because they displayed excessive movement during the scan, and nine were removed due to a history of psychosis, seizures, traumatic brain injury, or inability to read above a fourth grade level. Participants ranged in age from 13 to 19 years ($M = 17.32$, $SD = 1.18$), had an average full scale IQ of 92.80 ($SD = 12.06$, range = 63–140), and an average total PCL:YV score of 23.58 ($SD = 6.19$, range = 2–35). The most common racial/ethnic statuses were Hispanic (56.6%), Caucasian (14.8%), and Native American (11.7%).

Measures—The PCL:YV is a 20-item rating scale designed to measure psychopathy in youth. Each item is rated on a three-point scale (0 = does not apply, 1 = applies somewhat, 2 = definitely applies) and scores are combined to form a total score, two factor scores (core personality features of psychopathy; behavioral deviance), and four facet scores (interpersonal, affective, impulsive lifestyle, antisocial behavior). Interviews were conducted by trained raters and videotaped. Approximately 12% of the protocols were double-rated and yielded an ICC (1, 1) of .90 for the total PCL:YV score.

High-resolution T1-weighted structural MRI scans were performed and gray matter volumes (GMV) were estimated using voxel-based morphometry (VBM). As was already mentioned, gray matter concentrations (GMC) were unavailable for the adolescent sample. The only ROI excluded from this second study was the ACC because of its weak correlations with both the PCL:YV and the other ROIs. The remaining 14 ROIs were used to construct indicators for the GMV and GMV-PCL analyses. See Ermer et al. (2013) for full scanning parameters and analytical procedures.

Procedure—Participants were recruited from a maximum-security youth detention facility in New Mexico, and they and their parent/guardian provided informed consent for participation. Each participant was paid commensurate with the standard institutional pay rate. IRBs at the University of New Mexico Health Sciences Center and Kutztown University approved this study. A PCA with varimax rotation of the paralimbic data produced results similar to that observed in the PCA conducted on the adult sample except that the two temporal pole ROIs loaded better onto the AHP component than they did onto the TOCI component. Because taxometric analyses conducted on both sets of indicators achieved comparable results only the analyses performed on the original AHP and TOCI components are reported here.

Three analyses were performed, each with its own set of indicators. Indicators for the first set of analyses were right AHP (mean GMV for right amygdala, right hippocampus, and right parahippocampal cortex), left AHP (mean GMV for left amygdala, left hippocampus, and left parahippocampal cortex), right TOCI (mean GMV for right temporal pole, right OFC, medial OFC, and right insula), and left TOCI (mean GMV for left temporal pole, left OFC, PCC, and left insula). The two medial structures (medial OFC, PCC) were randomly assigned to either the left or right TOCI. Indicators for the second set of analyses were the four PCL:YV facet scores. Indicators for the third set of analyses were the mean AHP and TOCI scores and PCL:YV factor scores. All analyses were performed with (summed input) MAMBAC, MAXCOV, and L-Mode.

Results

The six GMV paralimbic biomarkers that served as indicators in this study all correlated significantly ($p < .05$) with the PCL:YV: $-.25$ (AHP), $-.45$ (TOCI), $-.24$ (AHP right), $-.20$ (AHP left), $-.35$ (TOCI right), and $-.42$ (TOCI left), for a mean correlation of $-.32$.

GMV Analyses—All GMV indicators correlated above .30 in the full sample (good model covariance), below .30 in the putative taxon and complement samples (low nuisance covariance), and discriminated between the putative taxon and complement groups at a level

greater than $d = 1.25$ (see upper portion of Table 3). The results of the GMV taxometric analyses are listed in Table 4 and clearly favor the continuous model, with all three CCFIs falling below .450. In addition, the MAMBAC, MAXCOV, and L-Mode data curves visually matched the dimensional comparison curves better than they did the categorical comparison curves (see Figure 2).

PCL: YV Analyses—As summarized in Table 3, the PCL:YV facet scores satisfied all three pre-criteria for taxometric analysis (good model covariance, low nuisance covariance, and good indicator validity). Taxometric analysis of the four PCL:YV facet scores produced mixed results, as indicated in Table 4. Whereas the CCFI from the MAMBAC analysis favored the categorical model, the CCFIs from the MAXCOV and L-Mode analyses favored the dimensional model. In addition, when the PCL:YV facet scores were subjected to standard MAMBAC analysis the results were consistent with dimensional latent structure (CCFI = .370). The single most accurate estimate in a taxometric analysis is the mean CCFI and this fell slightly below the cutoff for continuous latent structure even with the categorical summed input MAMBAC result (CCFI = .450).

GMV-PCL Analyses—Adequate model covariance, minimal nuisance covariance, and good indicator validity were all substantiated in pre-taxometric analyses of the four GMV-PCL indicators (see lower portion of Table 3). Cross-domain correlations ranged from .20 to .42 ($M = .30$). Taxometric analysis using the four GMV-PCL indicators revealed consistent support for dimensional latent structure as denoted by a relatively better fit between the data curve and dimensional model, CCFIs below .450 for MAMBAC, MAXCOV and L-Mode, and a mean CCFI of .276.

A second GMV-PCL analysis was conducted to evaluate the original Harris et al. (1994) conclusion that Factor 2 may be categorical. The two PCL:YV facets that form Factor 2 (i.e., Facets 3 and 4) were paired with the AHP and TOCI biomarker indicators in a supplemental taxometric analysis in which the full sample inter-indicator correlation reached .37 and the cross-domain correlations ranged from .16 to .40 ($M = .28$). The results of this analysis also supported dimensional latent structure: MAMBAC (CCFI = .374), MAXCOV (CCFI = .398), L-Mode (CCFI = .400), and mean CCFI = .391. This analysis could not be conducted for the sample of adult males in Study 1 because the two biomarkers correlated too weakly (.04–.10) with Facet 4.

Discussion

Congruent with the results of the first study, taxometric analyses performed on a group of adolescent male offenders, in which the paralimbic biomarkers correlated .32 (on average) with the PCL:YV, displayed consistent evidence of continuous latent structure. Like the first study, however, the sample size ($N = 191$) fell below Meehl's (1995) 300-participant threshold.

General Discussion

The question this study sought to answer was whether changing indicators from rating scales and self-report inventories to paralimbic biomarkers altered the conclusion that the latent

structure of psychopathy is continuous. The answer to this question, at least with respect to the paralimbic biomarkers examined in the current set of studies, is no—the latent structure of psychopathy is still continuous, even when paralimbic ROIs replaced the rating scales and self-report inventories from previous research or whether the full Psychopathy Checklist or just Factor 2 was analyzed. Congruent with the results of the majority of taxometric studies conducted on psychopathy (Edens et al., 2006; Edens et al., 2011; Guay et al., 2007; Marcus et al., 2004; Murrie et al., 2007; Walters, Duncan et al., 2007; Walters, Gray et al., 2007; Walters et al., 2008; Walters et al., 2011), the conclusion that psychopathy has a continuous latent structure does not appear to change when indicators tapping biological as opposed to behavioral/phenomenological sources are used to represent psychopathic personality disorder. It should be noted, however, that taxometric analysis does not assess the latent structure of a test, method, or procedure, but rather, the latent structure of the construct the test, method, or procedure is designed to measure. If the PCL-R, self-report measures of psychopathy, and paralimbic biomarkers of psychopathy are assessing the same underlying construct, they should produce similar taxometric results, which is exactly what happened in the current series of studies.

According to the results of these studies and the bulk of previous taxometric research, there is no clear dividing line between psychopathy and non-psychopathy. It would seem that individual differences in psychopathy are quantitative (“more of” or “less of”) rather than qualitative (“either or”) in nature. As such, everyone can be plotted along a psychopathic personality disorder continuum. Whether or not people move up and down this continuum in response to developmental changes or situational events is an open question that is clearly within the realm of possibilities. What is not an open question is that the continuous nature of psychopathy has important implications for classification, assessment, and treatment. A critical empirical question for classification that must be addressed is whether the psychopathy continuum comprises a single dimension or multiple dimensions. In the event psychopathy comprises multiple dimensions, it will be important to define and clarify these dimensions. Assessment is also shaped by a construct’s latent structure (Ruscio et al., 2006). If, as current research suggests, the latent structure of psychopathy is continuous, assessment needs to be directed at the entire spectrum using a relatively large number of items to plot a person’s position on the continuum rather than identifying a handful of items that cluster around the taxonic boundary and maximally differentiate between individuals classified as psychopaths and non-psychopaths. Treatment is also influenced by a construct’s latent structure. Labeling offenders “psychopaths” can limit their opportunities for treatment and increase the likelihood of more serious sanctions such as preventive detention (Edens et al., 2006; Edens & Petrila, 2006).

Although the taxometric method is incapable of informing causal content (e.g., biological versus environmental etiology), it can be of great assistance in clarifying causal process (i.e., contrasting specific etiology, threshold effects, nonlinear interaction, and developmental bifurcation, on the one hand, with additive etiology, on the other hand: Meehl, 1992; Walters, 2012). Environmental mold taxa may be just as prevalent as genetic mold taxa, and dimensions can assume the form of either situational continua or biological spectra (Waller & Meehl, 1998). Etiological process, on the other hand, is more accessible to the results of taxometric research. In situations where categorical latent structure is indicated the causal

process often assumes one of four patterns: specific etiology, a threshold effect, a nonlinear interaction, or developmental bifurcation. In situations where continuous latent structure is indicated the causal process is normally characterized as additive. This would suggest that the paralimbic biomarkers included in the two studies reported in this paper exert their effect by means of an additive process, in which different abnormal ROIs in the paralimbic system accumulate to affect behavior. With an additive model it is the number of relevant risk factors or etiological agents rather than any specific combination of risk factors or etiological agents that determines the level of risk or the severity of disorder. Investigations on the paralimbic correlates of psychopathy might therefore benefit from continued research directed at identifying the ROIs that are most closely associated with psychopathy and the process by which these ROI effects accumulate.

It could be argued that because the biomarker indicators correlated only modestly with the PCL-R in Study 1, they were not measuring the same region of the psychopathy criterion space as the PCL-R. In responding to this criticism there are three points that need to be emphasized. First, the six paralimbic biomarkers correlated $-.19$ (range = $-.16$ to $-.23$) with the PCL-R in Study 1 and $-.32$ (range = $-.20$ to $-.45$) with the PCL:YV in Study 2, signifying small-to-medium and medium size effects, respectively (Cohen, 1988). Second, the paralimbic anomalies found in psychopathy are subtle and the size of an effect for any given voxel is small. It was the overall pattern, or cluster, that yielded significant associations in Ermer et al. (2012). This cluster inference is well-established in structural brain imaging studies, where the effects of interest are small and distributed, rather than large and focal. Whereas optimally it is the totality of brain structure that should be considered, for this study we had to settle for voxel level data in selecting indicators, because such global measures did not fit well into a taxometric analysis. Third, for taxometric analysis the key is not how indicators correlate with outside criteria but how they correlate with one another. In the current study the mean full sample inter-indicator biomarker correlations were all above $.30$, the mean taxon and complement inter-indicator correlations were all below $.30$, and the between-group separation (d) exceeded 1.25 .

Although the current studies used the largest samples published in the brain imaging literature on psychopathy to date, they were both relatively small for a taxometric study. One of Meehl's (1995, 2004) rules of thumb is that a minimum sample size of 300 subjects is required for stable taxometric results. Given that the size of both samples ($N = 254$ in Study 1 and $N = 191$ in Study 2) fell below this threshold, it could be argued that a categorical effect did not surface because the sample sizes were too small. Two issues precluded combining the adult and juvenile samples to create a larger sample—(a) GMV values for adults were significantly higher than for juveniles, due to both developmental and scaling differences; and (b) potential psychometric differences between the PCL-R and PCL:YV may confound the results. Such a combination would likely yield a pseudotaxon for age. Results from a large-scale Monte Carlo study by Ruscio et al. (2010) indicate that sample size limitations cannot account for the current results. In testing the limits of some of Meehl's rules of thumb, Ruscio et al. discovered that in the case of a categorical construct, sample sizes as low as 100–200 provided accurate CCFI estimates. Although there was a significant drop in CCFI accuracy for continuous constructs measured with samples sizes

below 300, this was due primarily to an increase in indeterminate results rather than to an increase in false positives (i.e., continuous samples misclassified as categorical). Hence, if the latent structure of psychopathy, as measured by paralimbic biomarker indicators, is indeed categorical, the number of cases was sufficient to allow identification of a taxonic boundary. Conversely, if the latent structure of psychopathy is continuous, as suggested by the current results, then none of the taxometric analyses on the paralimbic biomarkers produced categorical or even indeterminate results. In either case there is minimal support for a categorical interpretation of the latent structure of biologically measured psychopathy.

Another of Meehl's (1995, 2004) rules of thumb postulates that for a taxon to be reliably identified using the taxometric method it should have a base rate of at least 10%. The base rate for psychopathy in the current studies ranged from 42% to 56% when estimates obtained in preliminary MAMBAC and MAXCOV analyses were averaged. Given the relatively small size of the samples used in the current series of studies, however, it could be argued that a psychopathy taxon exists but that the number of cases in the taxon was too small to be detected using the taxometric method. If a small undetected taxon did, in fact, exist it would have had to have been well below Hare's (2003) prison estimate of 20%–25% psychopathy given that all of the participants in both studies were incarcerated offenders. This possibility could only be ruled out by conducting another taxometric analysis on a much larger sample of prisoners. The current study was the first time, to the authors' knowledge, that data from MRI brain scans were used as indicators in a taxometric analysis. Future researchers might want to extend this methodology to other psychiatric conditions, such as schizotypy, where controversy continues to rage over whether the latent structure of the disorder is continuous or categorical (see Beauchaine, Lenzenweger, & Waller, 2008; Rawlings, Williams, Haslam, & Claridge, 2008).

The samples for the current studies consisted of adult and adolescent male incarcerated offenders, over half of whom were Hispanic. In addition, all inmates were sampled from medium- and maximum-security institutions (Study 1) or a single maximum-security detention facility (Study 2) in a single southwestern state. This clearly raises questions about the generalizability of the current results. Significant racial (Skeem, Edens, Camp, & Colwell, 2004) and gender (Verona & Vitale, 2006) effects have been observed in psychopathy research. This just said, race and gender have not been found to moderate the taxometric results of studies on adult psychopathy (Guay et al., 2007; Walters, Gray et al., 2007; Walters et al., 2008). Even so, the need for more research is obvious. A study with a larger sample size and greater diversity of participant age, gender, race, and geographic location would allow for more conclusive statements about the latent structure of psychopathy. Moreover, studies employing indicators other than the rating scales, self-report inventories, and paralimbic biomarkers that have been used thus far are necessary. Behavioral performance indicators designed to assess the putative attentional, language, and passive avoidance deficits in psychopathy (Hiatt & Newman, 2006) or brain activity during an impulse control task (Aharoni et al., 2013) would appear to be the next logical candidates for taxonomic investigation. Although the current results suggest that conclusions about the continuous latent structure of psychopathy are not an artifact of rating scale and self-report inventory indicators, additional research is required to determine the generalizability and robustness of these preliminary findings.

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Figure 1.

Results from Study 1 of a taxometric analysis of four indicators representing gray matter concentration (GMC) scores in adult males, with the base rate set at .560 (average of the MAMBAC and MAXCOV base rate estimates). The categorical, dimensional, and data curves are shown for summed input MAMBAC (mean above minus below a cut; top panel), MAXCOV (maximum covariance; middle panel), and L-Mode (latent mode; bottom panel). In each graph, the dark line represents the data curve, lighter lines connect the minimum and maximum values at each data point, and the gray area signifies the middle 50% of values.

Figure 2.

Results from Study 2 of a taxometric analysis of four indicators representing gray matter volume (GMV) scores in adolescent males, with the base rate set at .545 (average of the MAMBAC and MAXCOV base rate estimates). The categorical, dimensional, and data curves are shown for summed input MAMBAC (mean above minus below a cut; top panel), MAXCOV (maximum covariance; middle panel), and L-Mode (latent mode; bottom panel). In each graph, the dark line represents the data curve, lighter lines connect the minimum and maximum values at each data point, and the gray area signifies the middle 50% of values.

Table 1
Descriptive Statistics, Indicator Validities, and Mean Inter-Indicator Correlations for Indicators in the GMC, PCL-R, and GMC-PCL Analyses: Adult Sample

Indicators	Range	M	SD	Skew	Kurtosis	d	r(tot)	r(tax)	r(comp)
GMC Analyses									
AHP right	.328-.544	0.428	0.044	0.178	-0.438	1.92	1.64	.51	.19
AHP left	.374-.673	0.510	0.047	0.053	0.262	1.53			
TOCright	.372-.606	0.502	0.036	-0.356	0.798	1.49			
TOCleft	.429-.624	0.514	0.035	0.041	-0.177	1.62			
PCL-R Analyses									
Facet 1	0-8	2.339	1.995	0.780	0.012	1.50	1.45	.37	.03
Facet 2	0-8	3.913	2.088	-0.125	-0.907	1.57			
Facet 3	0-10	5.791	2.232	-0.253	-0.681	1.47			
Facet 4	0-10	7.047	2.355	-0.784	0.118	1.27			
GMC-PCL Analyses									
GMC AHP	.368-.605	0.469	0.040	0.130	0.004	1.45	1.33	.29	-.03
GMC TOCI	.417-.599	0.508	0.033	-0.131	-0.011	1.26			
PCL Factor 1	0-15	6.256	3.395	0.397	-0.499	1.30			
PCL Factor 2	1-20	12.831	3.896	-0.575	-0.073	1.29			

Note. Putative taxon and complement groups formed by dividing the groups at the base rate suggested by preliminary MAMBAC and MAXCOV analyses (see Table 2); Range = range of scores in the current sample; M= mean; SD= standard deviation; d = Cohen's d as a measure of indicator validity; r(tot) = mean inter-indicator correlation for total sample; r(tax) = mean inter-indicator correlation for the putative taxon; r(comp) = mean inter-indicator correlation for the putative complement; GMC = gray matter concentration; PCL-R= Psychopathy Checklist -Revised; AHP = amygdala-hippocampus-parahippocampal; TOCI= temporal pole-orbitofrontal-cingulate-insula.

Table 2

Results of the GMC, PCL-R, and GMC-PCL Analyses: Adult Sample

	Taxon Base Rate Estimate			Comparison Curve Fit Index (CCFI)		
	MAMBAC	MAXCOV	Mean	MAMBAC	MAXCOV	L-Mode Mean
GMC	.538	.581	.560	.338	.425	.223 .329
PCL-R	.522	.433	.478	.261	.280	.283 .275
GMC-PCL	.422	.420	.421	.187	.264	.305 .252

Note. MAMBAC = mean above minus below a cut; MAXCOV = maximum covariance; L-Mode = latent mode factor analysis; Mean (under Taxon Base Rate Estimate) = mean of the MAMBAC and MAXCOV base rate estimates; Mean (under Comparison Curve Fit Index) = mean of MAMBAC, MAXCOV, and L-Mode CCFI values; GMC = gray matter concentration; PCL-R = Psychopathy Checklist-Revised.

Table 3

Descriptive Statistics, Indicator Validities, and Mean Inter-Indicator Correlations for Indicators in the GMV, PCL:YV, and GMV-PCL Analyses: Adolescent Sample

Indicators	Range	M	SD	Skew	Kurtosis	d	r(tot)	r(tax)	r(comp)
GMV Analyses									
AHP right	.054–.073	0.064	0.003	0.024	–0.147	1.78			
AHP left	.052–.068	0.058	0.003	0.386	–0.206	1.67			
TOCI right	.054–.068	0.060	0.003	0.051	–0.258	1.40			
TOCI left	.045–.061	0.052	0.003	0.302	0.182	1.58			
PCL:YV Analyses									
Facet 1	0–8	2.194	1.938	0.902	–0.167	1.26	.42	.01	.07
Facet 2	0–8	4.461	1.761	–0.229	0.729	1.79			
Facet 3	0–10	6.438	1.999	–0.578	0.017	1.69			
Facet 4	0–10	8.223	1.701	–1.370	3.102	1.63			
GMV-PCL Analyses									
GMV AHP	.055–.070	0.061	0.003	0.109	–0.475	1.26	.37	.01	.07
GMV TOCI	.050–.063	0.056	0.002	0.115	–0.179	1.60			
PCL Factor 1	0–15	6.658	3.142	0.391	–0.428	1.40			
PCL Factor 2	1–20	14.660	3.270	–1.125	2.085	1.51			

Note. Putative taxon and complement groups formed by dividing the groups at the base rate suggested by the preliminary MAMBAC and MAXCOV analyses (see Table 4); Range = range of scores in the current sample; M= mean; SD= standard deviation; d = Cohen's d as a measure of indicator validity; r(tot) = mean inter-indicator correlation for total sample; r(tax) = mean inter-indicator correlation for the putative taxon; r(comp) = mean inter-indicator correlation for the putative complement; GMV = gray matter volume; PCL:YV = Psychopathy Checklist: Youth Version; AHP = amygdala-hippocampus-parahippocampal; TOCI = temporal pole-orbitofrontal-cingulate-insula.

Table 4

Results of the GMV, PCL:YV, and GMV-PCL Analyses: Adolescent Sample

	Taxon Base Rate Estimate			Comparison Curve Fit Index (CCFI)		
	MAMBAC	MAXCOV	Mean	MAMBAC	MAXCOV	L-Mode Mean
GMV	.484	.607	.545	.399	.299	.280 .326
PCL:YV	.526	.520	.523	.581	.336	.412 .443
GMV-PCL	.500	.510	.505	.292	.388	.148 .276

Note. MAMBAC = mean above minus below a cut; MAXCOV = maximum covariance; L-Mode = latent mode factor analysis; Mean (under Taxon Base Rate Estimate) = mean of the MAMBAC and MAXCOV base rate estimates; Mean (under Comparison Curve Fit Index) = mean of MAMBAC, MAXCOV, and L-Mode CCFI values; GMV = gray matter volume; PCL:YV = Psychopathy Checklist: Youth Version.