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Identifying Subtypes Among Offenders With Antisocial Personality Disorder: A Cluster-Analytic Study

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The question of whether antisocial personality disorder (ASPD) and psychopathy are largely similar or fundamentally different constructs remains unresolved. In the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994), many of the personality features of psychopathy are cast as associated features of ASPD, although the *DSM–IV* offers no guidance as to how, or the extent to which, these features relate to ASPD. In a sample of 691 offenders who met *DSM–IV* criteria for ASPD, we used model-based clustering to identify subgroups of individuals with relatively homogeneous profiles on measures of associated features (psychopathic personality traits) and other constructs with potential etiological significance for subtypes of ASPD. Two emergent groups displayed profiles that conformed broadly to theoretical descriptions of primary psychopathy and Karpman's (1941) variant of secondary psychopathy. As expected, a third group (nonpsychopathic ASPD) lacked substantial associated features. A fourth group exhibited elevated psychopathic features as well as a highly fearful temperament, a profile not clearly predicted by extant models. Planned comparisons revealed theoretically informative differences between primary and secondary groups in multiple domains, including self-report measures, passive avoidance learning, clinical ratings, and official records. Our results inform ongoing debates about the overlap between psychopathy and ASPD and raise questions about the wisdom of placing most individuals who habitually violate social norms and laws into a single diagnostic category.

Keywords: antisocial personality, psychopathy, criminal offenders

The relation between antisocial personality disorder (ASPD) and the personality construct of psychopathy is controversial (Lilienfeld, 1994). Widiger (2000) identified Cleckley's (1941/1982) classic work as providing one of the early influential formulations of ASPD, although the work of Robins (1966) has influenced more recent formulations. Cleckley used the term *psychopathy* to identify a syndrome that included substantial interpersonal (e.g., untruthfulness, insincerity), affective (e.g., lack of remorse or

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shame), and behavioral (e.g., inadequately motivated antisocial behavior, failure to learn from punishment) symptoms that were masked by superficial features of positive adjustment (e.g., superficial charm, appearance of good intelligence, absence of delusions or psychoneurotic features). Cleckley's conceptualization was featured prominently in the *Diagnostic and Statistical Manual of Mental Disorders* (2nd ed.; *DSM–II*; American Psychiatric Association, 1968).

However, in part because many core interpersonal and affective features of psychopathy were considered difficult to assess reliably, the next revision of the *DSM*—the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM–III*; American Psychiatric Association, 1980)—largely excluded such features in favor of more explicitly behavioral indicators of antisocial conduct that presumably could be assessed more reliably. These competing approaches—one more personality-based (Cleckley's, 1941/1982, formulation) and one more behavior-based (the *DSM–III* ASPD)—remain in use today, although the question of whether they identify substantially different individuals continues to inspire vigorous debate (Lilienfeld, 1998).

At about the same time that the *DSM* was moving to a more behavior-based approach to the assessment of ASPD, psychopathy as a clinical construct was gaining ascendancy, largely through the work of Robert Hare. Hare's Psychopathy Checklist (PCL; 1980) and Psychopathy Checklist–Revised (PCL-R; 1991/2003) were based in part on Cleckley's (1941/1982) work and included items that assessed interpersonal and affective features as well as a socially deviant lifestyle and explicitly criminal behavior. Researchers using the PCL/PCL-R in correctional settings have revealed that as many as 70%–80% of prisoners met the *DSM* criteria for ASPD, whereas only about a third of those individuals met the recommended PCL/PCL-R cutoff for a diagnosis of psychopathy (Widiger & Corbitt, 1995).

For many, this differential epidemiology of ASPD versus psychopathy in prisoner populations signaled that the constructs were markedly different (e.g., Hare, 1991/2003; Lykken, 1995). Henry and Moffitt (1998) noted that the different measurement approaches "do not produce identical subject groups . . . we may be comparing apples and oranges" (p. 284). Similarly, Hare (1998) asserted: "[T]he listed criteria for APD [antisocial personality disorder] actually identify individuals who are persistently antisocial, most of whom are not psychopaths," (p. 191) and "[S]ome clinicians and investigators use the [psychopathy and APD] labels as if the constructs they measure were interchangeable. They are not" (p. 193).

Others, however, view ASPD and psychopathy as essentially the same disorder, most likely reflecting only minor variants of each other. Some do so implicitly by citing research findings based on ASPD assessments to draw inferences about psychopathy (e.g., Paris, 1998). For others, the comparison is more explicit. Citing high correlations between dimensional measures of the two constructs and similar associations for the *DSM* and PCL-R criterion sets with external measures in the field trials of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994; see Widiger et al., 1996), Widiger (2006) concluded,

> In sum, the *DSM–IV* APD and PCL-R psychopathy criterion sets do not appear to be identifying different disorders. The two respective criterion sets are instead quite similar albeit alternative efforts at identifying the same personality disorder. (p. 158)

The *DSM–IV* text also supports this view, noting that ASPD "has also been referred to as psychopathy, sociopathy, or dissocial personality disorder" (American Psychiatric Association, 1994, p. 645).

An apparent effort to reconcile these views can be gleaned from the observation that the personality features of psychopathy, not referenced in the *DSM–III* or the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM–III–R*; American Psychiatric Association, 1987), were added as "associated features" in the text of the *DSM–IV*. Widiger and Corbitt (1995) characterized this addition as a "remedy" for prior omissions (p. 121), and in the following they asserted that these features may have particular importance in offenders:

> Failure to conform to social norms with respect to lawful behavior . . . will be relatively nonspecific within a prison setting. In this setting, such traits of psychopathy as lack of empathy, glib charm, and arrogant self-appraisal may then be more diagnostic of ASPD. (Widiger & Corbitt, 1997, p. 76)

Although acknowledging the relevance of psychopathic traits as associated features alongside the formal ASPD diagnostic criteria *seems* to close the schism between the personality-based and behavior-based approaches, this closure may be more apparent than real. Reflecting on this change in the *DSM–IV*, Hare (1998) noted that the *DSM–IV* provided no explicit instruction regarding the integration of the associated features into diagnostic considerations: "Curiously, clinicians are left entirely on their own when it comes to whether and how to assess these traits" (p. 191).

Integrating the Personality-Based and Behavior-Based Approaches to ASPD

In a thoughtful commentary on these issues, Pilkonis and Klein (1998) proposed an empirical strategy for integrating these different approaches to the assessment of ASPD/psychopathy. They recommended that researchers define

> multidimensional trait models . . . to try to identify clusterings of features that define groups that appear to be relatively homogeneous within themselves and different from other groups. One task for future research is to provide evidence regarding the construct validity of such groups . . . . the best categories are likely to grow out of the best profiling that we can do across relevant traits. (p. 111)

Specifically, researchers could use the *DSM* criteria to identify a large sample of individuals who meet the ASPD diagnosis, and then they could attempt to disaggregate that sample into relatively homogeneous clusters, using as clustering variables valid dimensional measures of the associated features. However, variations in the associated features alone would likely not be adequate to establish an optimal typology. Pilkonis and Klein (1998) further noted that

> to establish a maximally useful way of conceptualizing and assessing antisocial behavior and personality, a variety of factors related to dimensions of the individual's functioning and interaction with the environment must be taken into account. These . . . variables are both

> internal and external to the individual and . . . influence behavior as well as personality. (p. 109)

Fortunately, a rich clinical and theoretical literature provides guidance on the selection of internal and external variables that bear implications for the etiology of potential variants of ASPD. Regarding internal variables, constructs relating to fear sensitivity and reward sensitivity have been implicated in theories of psychopathy that include constructs from Gray's (1982) reinforcement sensitivity theory (RST). Lykken (1995) suggested that an innately fearless temperament is the basis for Cleckley's (1941/1982) *primary* psychopath; low fear sensitivity renders these individuals less responsive than others to the kinds of threats that parents or other caregivers apply in trying to shape their behavior and instill prosocial attitudes and internal constraints (i.e., conscience). Lykken also described primary psychopathic individuals as likely to exhibit relatively few signs of anxiety. Lykken's model of primary psychopathy is consistent with that of other RST investigators except for the role of reward sensitivity. Whereas Lykken postulated normal reward sensitivity in primary psychopaths, Blackburn (2006) suggested that a relatively fearless temperament would be accompanied by unusually high reward sensitivity. In terms of manifest psychopathic features, Lykken (p. 132) hypothesized that RST primary psychopaths should exhibit higher elevations on PCL-R Factor 1, which includes the interpersonal (Facet 1) and affective (Facet 2) features.

Lykken (1995) hypothesized that an alternative influence on the development of psychopathic features is elevated reward sensitivity. Individuals with presumably normal fear sensitivity might find that their internal restraints often fail due to powerful appetitive urges that overwhelm normal inhibitions, resulting in behavior that violates laws or other social norms. Lykken also hypothesized that this *secondary* psychopath, unlike the primary psychopath, would be vulnerable to anxiety and other negative emotions. Lykken's model of secondary psychopathy is consistent with that of other RST investigators except for the role of fear sensitivity. Whereas Lykken postulated normal fear sensitivity in secondary psychopaths, Blackburn (2006) suggested that both reward sensitivity and fear sensitivity would be elevated in this group.

Regarding external factors, Karpman (1941) and Porter (1996), among others, emphasized the role that environmental factors may play in the development of psychopathic features. Karpman described secondary psychopaths as individuals whose psychopathic features are a consequence of unresolved emotional conflicts resulting from parental overindulgence or rejection. As did Lykken (1995), Karpman viewed secondary psychopaths as more vulnerable to negative emotions (i.e., anxiety, guilt) and as more impulsive in their actions (Karpman, 1948), thereby implicating relatively higher scores on PCL-R Facet 3. Like Karpman (1941, 1948), Porter hypothesized that disrupted parental relationships, specifically the occurrence of early abuse or abandonment by parents, might result in secondary psychopathy. However, Porter hypothesized that such adverse experiences significantly influence the child's capacity for social attachment and emotional responding, thereby implicating enhanced interpersonal (Facet 1) and affective (Facet 2) features of PCL-R psychopathy. Also, unlike Karpman's model, Porter's model does not predict high levels of anxiety in secondary psychopaths.

Overview of the Present Study

Following the strategy proposed by Pilkonis and Klein (1998), we used the technique of model-based cluster analysis, which arbitrates among alternative cluster solutions using goodness-of-fit criteria, to disaggregate a large sample of offenders who met ASPD diagnostic criteria. We used as clustering variables the scores on Facets 1–3 of the PCL-R, along with a measure of anxiety, to index the associated features of ASPD. Our cluster variable set also included measures of the aforementioned internal and external variables that relate to putative etiologies of antisocial behavior and psychopathic traits.

This set of theoretical ASPD/psychopathic subtypes is not exhaustive. Lykken (1995) described other potential variants, and Millon and Davis (1998) described 10 theoretical subtypes that differ mainly in psychodynamic features. Mealey (1995) discussed an array of family (e.g., single parent, large family size) and social (e.g., lower socioeconomic status) problems that may place individuals at risk for antisocial traits and tendencies. The possibility of other theoretical subtypes leaves open the possibility that one or more clusters might emerge whose profile is not consistent with our predicted patterns. In addition, the researchers referenced earlier have demonstrated that many offenders who meet ASPD diagnostic criteria will not manifest substantial levels of associated features (i.e., psychopathic personality traits). Thus, we anticipated that at least one such group would be identified through cluster analysis. However, the aforementioned variants are those most extensively discussed in the research and clinical literatures and are therefore most important to consider in investigations of ASPD subtypes. Importantly, they provide a basis for a priori hypotheses about the profile patterns.

Hypotheses

Anticipating that the hypothesized variants of ASPD identified on the basis of the previously mentioned literature might emerge from our cluster analysis, we articulated a series of hypotheses as to how primary and secondary variants should differ on varying external criteria. Planned comparisons on these variables provided preliminary evidence regarding the validity of the emergent clusters.

Hypothesis 1. The internalizing dimension of psychopathology relates to anxiety and other negative moods and emotions. Karpman (1948) characterized the secondary psychopath as "pervaded with states of anxiety, depression and guilt" (p. 526). Similarly, Lykken (1995) described the secondary psychopath as "stress-reactive, worried, irritable, dissatisfied with his life and with himself" (p. 37). In contrast, primary psychopaths are thought to be largely immune to anxiety and other negative emotions (Karpman, 1949). Thus, we predicted higher mean scores for a secondary group on a measure of internalizing psychopathology.

Hypothesis 2. The externalizing dimension of psychopathology captures behaviors and traits such as aggression, antisocial actions, and substance use. Perhaps in part because of their greater susceptibility to internalizing problems, individuals with secondary psychopathy are thought to express more reactive aggression (Karpman, 1948), and prior studies have demonstrated stronger associations with substance use problems for those with features of secondary psychopathy than for those with features of primary

psychopathy (Smith & Newman, 1990). Thus, although all individuals meeting ASPD criteria would be expected to manifest some externalizing behavior, we hypothesized that externalizing problems would be stronger in a secondary psychopathic group.

Hypothesis 3. Clinically, Karpman (1948) described secondary psychopaths as hot-headed and impulsive but primary psychopaths as comparatively cool and deliberate. Thus, we predicted higher impulsivity scores for a secondary group.

Hypothesis 4. Blackburn (1987) emphasized the role of interpersonal style in distinguishing primary and secondary psychopathy, describing the primary psychopath as extroverted, confident, and dominant but the secondary psychopath as more socially withdrawn and inhibited. Thus, we predicted higher scores on an index of interpersonal dominance for a primary group.

Hypothesis 5. Cleckley (1941/1982) described the psychopath as failing to learn from prior experience, more specifically, failing to inhibit behaviors that result in punishment. Lykken (1995) described how either a fearless temperament (primary psychopath) or an elevated reward sensitivity (RST secondary psychopath) could result in poor passive avoidance learning. Thus, we predicted that emergent groups representing RST models of primary and secondary psychopathy would make more passive avoidance (commission) errors on a passive avoidance learning task than would a group representing Karpman's (1941, 1948) or Porter's (1996) secondary subtype.

Hypothesis 6. Karpman (1946) suggested that psychopathy resulting from adverse environmental experiences might be more responsive to treatment than psychopathy associated with constitutional deficits. Thus, we predicted that an emergent group representing Karpman's (1948) or Porter's (1996) secondary psychopath would show more positive scores on indices of treatment engagement, participation, and outcomes than would primary or RST secondary groups.

Hypothesis 7. Lykken (1995) hypothesized that the secondary psychopath "would be likely to make a poor adjustment to the stresses of prison life" (p. 122), and Blackburn (1987) cited two findings that secondary psychopaths displayed more aggressive and disruptive behavior in an institutional setting. Thus, we predicted that secondary clusters would exhibit higher rates of institutional infractions.

Hypothesis 8. Meta-analyses (e.g., Walters, 2003) have reported weaker associations with recidivism for the traditional Factor 1 of the PCL-R (which includes Facets 1 and 2) than for Factor 2 (which includes Facet 3). Thus, we predicted that secondary ASPD clusters would display higher rates of recidivism upon release into the community.

Method

Participants

We recruited 1,413 male offenders who had been court-ordered to residential drug treatment programs ($n = 660$; 47%) or were serving prison sentences ($n = 753$; 53%) at multiple sites in Oregon, Utah, Nevada, Florida, and Texas. An additional 222 individuals declined to participate. Because we were interested in comparing emergent ASPD clusters on both prison adjustment during the first year of incarceration and recidivism upon release into the community, we recruited inmates who had either been newly admitted to prison (new admission inmates), for whom we could later retrieve records of disciplinary reports, or who were nearing release (near release inmates), for whom we could obtain follow-up arrest records after release from prison.

English-speaking African American and Caucasian men were enrolled. The mean age of the sample was 30.87 years ($SD = 6.63$). An attempt was made to recruit participants between the ages of 21 and 40, although when offenders in this age range were not available, we recruited offenders outside this range (younger: $n = 28$; 2%; older: $n = 50$; 3.5%). To minimize the potential influence of psychiatric symptoms on responding, we did not recruit from prison mental health treatment units, and at all sites research assistants (RAs) screened out individuals currently taking psychotropic medications for active symptoms of psychosis (assessed by self-report and chart review). We also excluded individuals with a chart diagnosis of mental retardation and those with an estimated IQ < 70 on the basis of a standardized assessment (Ammons & Ammons, 1962) administered at the time of enrollment. We endeavored to recruit equal numbers of African American and Caucasian participants. However, African Americans were better represented in the prison sample (46.2%) than in the drug treatment sample (24.4%), $\chi^2(1) = 44.88$, $p < .001$. The percentage of the sample at each level of self-reported educational attainment was as follows: high school uncompleted, 28.9%; general equivalency diploma (GED), 22.1%; high school diploma, 20.7%; some college, 24.9%; college diploma, 2.6%; any graduate/professional school, 0.5%.

Of the 1,413 men enrolled in the study, data were excluded for 30 participants whose scores on the Personality Assessment Inventory (PAI; see Measures, Measures of Clustering Variables, Measures of Psychopathic Features subsection) were indicative of invalid responding.[1] An additional 175 participants were excluded due to missing data: 80 participants failed to complete the ASPD diagnostic interview; 73 did not complete the PAI, and an additional 22 cases had missing data on other clustering variables. Of the remaining 1,208 cases, analyses were conducted on data from the 691 individuals who met *DSM–IV* criteria for an ASPD diagnosis. Mean age (30.03 years) and racial distribution (34% African American) were approximately the same as in the full recruitment sample.

Measures

Assessment of ASPD. The ASPD module of the Structured Clinical Interview for *DSM–IV* Axis II Personality Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1996) was used to identify participants who met diagnostic criteria for ASPD. The prevalence of ASPD was virtually identical across site (58.2% prison; 57.7% drug treatment) and ethnicity (57.9% of both Caucasian and African American participants). In the current sample, interrater reliability was good for ASPD diagnoses ($\kappa = .74$; $n =$

[1] Two validity scales from Morey's (1991/2007) PAI were used to judge for potentially invalid responding. The Infrequency scale contains items that are rarely endorsed, whereas the Inconsistency scale indexes pairs of items of similar content that were not answered in a consistent fashion. In correction samples, T scores > 79 on these scales suggest possibly invalid responding (Edens & Ruiz, 2005).

50) and total symptom count (*intraclass correlations* [ICC_1] = .86; n = 46). Internal consistency (alpha) was .83.

Measures of clustering variables

Measures of psychopathic features. Hare's (1991/2003) PCL-R was used to assess the associated features of ASPD (i.e., psychopathic personality traits). The PCL-R involves a lengthy (about 1.5 hr) clinical interview and review of agency/institutional records. These sources of information provide the basis for clinical ratings (0 to 2) on each of 20 items, resulting in scores that range from 0 to 40.

We used as clustering variables factor scores from the first three facets of Hare's (1991/2003) four-facet model. These facets capture the core interpersonal (four items; e.g., glibness/superficial charm, conning/manipulative) and affective (four items; e.g., lack of remorse or guilt, callous/lack of empathy) features as well as indicators of an impulsive lifestyle (five items; e.g., need for stimulation/proneness to boredom). We excluded the fourth (antisocial) facet, which comprises mainly explicitly criminological items (e.g., juvenile delinquency; criminal versatility); these items are more similar to the *DSM–IV* criteria for ASPD than to the more personality-based associated features of ASPD. Internal consistency (alpha) for the PCL-R total score was .81; for Facets 1–3, alphas were .70, .74, and .57, respectively. On the basis of 51 cases, interrater reliability (ICC_1) for the PCL-R total scores was .88.

Some authors (e.g., Karpman, 1948, 1949) have asserted that anxiety or neuroticism is a significant trait for distinguishing primary (low anxiety) from secondary (high anxiety) psychopaths. To assess trait anxiety we used the Anxiety (ANX) scale from Morey's (1991/2007) PAI. The PAI is a 344-item self-report inventory that includes 4 validity scales, 11 clinical scales, 5 treatment scales, and 2 scales that assess interpersonal style. The PAI scales have demonstrated satisfactory psychometric properties across large samples of student, community, clinical, and corrections samples. The ANX scale includes a variety of cognitive, physiological, and affective symptoms associated with anxiety. Internal consistency (alpha) was .91.

Measures of putative etiological factors. We administered the Harm Avoidance (HA) scale from the Multidimensional Personality Questionnaire (MPQ; Tellegen, in press) as a (reversed) index of fearless temperament. The HA scale consists of 28 forced-choice items; each item juxtaposes a risky or potentially fear-inducing activity with another that is less so. A high score on the HA indicates a preference for avoiding potentially harmful situations, whereas a low score suggests a more fearless temperament. In this sample, internal consistency (alpha) for the HA was .86.

Sensitivity to reward stimuli was assessed with Carver and White's (1994) three self-report behavioral activation system (BAS) scales, which were designed to index different aspects of Gray's (1982) BAS construct. The Reward Responsiveness scale (RR) taps the individual's affective motivation to pursue appetitive or other positive rewards (e.g., "When I get something I want, I feel excited and energized"). The Drive scale (DR) is a measure of the intensity or tenacity with which one pursues positive rewards (e.g., "When I want something, I usually go all-out to get it"). Finally, Fun Seeking (FS) is akin to the construct of stimulation seeking and indexes the tendency to seek out novel and stimulating situations (e.g., "I crave excitement and new sensations"). In this sample, internal consistencies (alphas) were .82, .85, and .77 for the RR, DR, and FS, respectively.

To identify individuals whose prominent psychopathic features may have emerged as a consequence of early abuse, we had participants complete the Child Abuse and Trauma Scale (CATS; Sanders & Giolas, 1991). This 38-item self-report measure indexes participants' recall of the frequency with which they experienced negative events in the home environment, including physical abuse, sexual abuse, psychological abuse, and neglect. Internal consistency (alpha) of the CATS was .95.

Measures of cluster validation variables

Internalizing problems. To test Hypothesis 1, we computed an index of internalizing psychopathology identified by Ruiz and Edens (2008) by using scales from the PAI. This index is computed as the average of scores from scales that assess symptoms associated with *DSM* disorders including somatization (α = .88 in the present sample), anxiety-related disorders (α = .81), schizophrenia (α = .85), and depression (α = .89), as well as suicidal ideation (α = .88).

Externalizing problems. To test Hypothesis 2, we computed an index of externalizing psychopathology identified by Ruiz and Edens (2008) by using scales from the PAI. This index is computed as the average of scores from scales that assess symptoms associated with *DSM* disorders including alcohol problems (α = .94), drug problems (α = .89), borderline personality (α = .88), mania (α = .81), paranoia (α = .84), and antisocial features (α = .85), as well as symptoms of aggression (α = .91).

Impulsivity. To test Hypothesis 3, we administered the Barratt Impulsivity Scale–Version 11 (BIS-11; Stanford & Barratt, 1995). This 30-item self-report measure assesses various aspects of impulsivity, including attentional deficits, motor restlessness, and nonplanning. In the present sample, internal consistency (alpha) was .86.

Dominance. To test Hypothesis 4, we administered the Dominance scale from the PAI. This scale indexes the extent to which an individual is independent, controlling, and autonomous in interpersonal situations versus being more submissive. Internal consistency (alpha) was .78.

Passive-avoidance learning. To test Hypothesis 5, we assessed passive–avoidance learning with a computerized successive go–no-go[2] discrimination task (Newman & Kosson, 1986) in which respondents must learn, during an initial block of 40 trials, to press a button in response to the presentation of certain two-digit stimuli (S+) to earn a monetary award (e.g., 10 cents) and to suppress responding to other two-digit stimuli (S–) to avoid losing the monetary reward. In our study, the dependent measure was the number of errors of commission made during the second block of 40 trials. Rather than competing for the actual cumulative 10-cent rewards, participants competed for a reward of $25 to be given to the individual with the highest cumulative 10-cent reward total.

Treatment participation and progress. To test Hypothesis 6, we obtained information regarding treatment behavior, motivation, and progress for 193 drug treatment participants. Sources of information included a review of records and postdischarge inter-

[2] Our go–no-go task was downloaded from the website of Joseph Newman at the Department of Psychology, University of Wisconsin: http://psych.wisc.edu/newman/Pages/Tasks2.html

views with participants' primary counselors. Using a 4-point scale ranging from 1 (*Never*) to 4 (*Often*), counselors rated (a) the frequency with which the individual had unexcused absences from treatment sessions and (b) the frequency with which the counselor had to confront the individual for disruptive behavior in group therapy. Counselors also completed ratings on a 7-point scale to describe the individual's level of motivation for treatment and progress in mastering skills needed to reduce substance abuse. The PAI Treatment Rejection scale ($\alpha = .69$) was used as a supplementary indicator of treatment motivation. Finally, therapists provided subjective ratings of participants' end of treatment status, which were coded as 0 (failed treatment or made minimal gains) or 1 (achieved substantial gains or succeeded in treatment). Records were used to code objective end of treatment status. Each treatment program involved a multitiered system through which clients progressed over the course of treatment. We extracted from agency records information about whether participants had achieved the program's highest level at the time of discharge.

Institutional misconduct and criminal recidivism. To test Hypotheses 7 and 8, we obtained counts of disciplinary reports for offenders in prisons as well as rearrest counts for participants released into the community. For participants recruited in the prisons as new admissions, we obtained disciplinary reports for a 1-year period following recruitment into the study. Following the procedure described by Edens, Poythress, and Lilienfeld (1999), we compared disciplinary infraction policies obtained from the various states' Departments of Corrections and developed a common scheme for coding infractions as nonaggression (e.g., "possession of contraband"), verbal aggression/acts of defiance (e.g., "spoken or written threats"), or physical aggression (e.g., "assault or battery with a deadly weapon"). Because acts of physical aggression were exceedingly rare over the follow-up period, we collapsed this category with verbal aggression/acts of defiance to create a broadband aggression outcome measure. Dichotomous (yes/no) variables indicated whether each participant had incurred a disciplinary report for any kind of infraction or for only aggression during the 1-year follow-up. Base rates for these two outcome categories were 42.5% (any) and 27.4% (aggression).

We obtained arrest records of participants who were released into the community after protocol completion. Identifying information for all participants from drug treatment programs and for those near-release prison inmates recruited into the study within 6 months of their sentence completion was used to search arrest records, both state and federal, archived by the Federal Bureau of Investigation. Dichotomous (yes/no) variables indicated whether each participant had been arrested for any kind of offense or for only a violent offense within a 1-year period following enrollment (drug treatment program participants) or following release from prison into the community (near-release prisoners). Our definition of *violent offense* included any explicitly assaultive act against another person (e.g., murder, manslaughter, assault), robbery, and rape or other sexual assault.

Procedure

RAs were advanced graduate students in clinical psychology or social work. They were trained on informed consent and data collection procedures, including extensive training on the PCL-R and the ASPD module of the SCID-II. RAs were trained to reliability on the PCL-R ($ICC_1 \geq .80$) prior to commencing data collection and were observed by Kevin Douglas on two cases every 6 months throughout the course of the study, receiving feedback and supervision to prevent rater drift. These observations also provided interrater reliability data. At each site, participants were randomly recruited from lists of individuals who met basic eligibility criteria. Enrollment interviews were conducted in a private room, and informed consent was obtained, using procedures approved by university institutional review boards. After participants gave informed consent, they took the IQ screen test.

Participants completed the PAI using paper-and-pencil and then the remaining self-report measures and the go–no-go task using a laptop computer. The reading ability of participants who (a) did not have either a GED or a 10th-grade education or (b) could not easily read the first few items of the PAI was assessed with the Basic Reading Inventory (Johns, 1997). The RA read aloud items from self-report measures to participants who did not demonstrate a 9th-grade reading level on the inventory. Except at one agency that prohibited payment, participants received $20 for study participation.

Data Analysis

Cluster analysis was conducted with model-based clustering (Mclust; Fraley & Raftery, 2003) using both R (R Development Core Team, 2009) and S-PLUS 6.2 (Insightful, 2001) software. Clustering variables were *z* scores for the interpersonal, affective, and impulsive lifestyle features (Facets 1–3) of Hare's (1991/2003) PCL-R; the ANX scale from Morey's (1991/2007) PAI; Tellegen's (in press) HA scale; Carver and White's (1994) reward sensitivity scales (the RR, DR, and FS); and Sanders and Giolas's (1991) CATS.

Under the assumption that the data represent an unknown number of different subpopulations, we used a statistical fit index with Mclust to attempt to fit multiple mixture Gaussian models and to evaluate the goodness of fit of multiple solutions within each model and across models. On the basis of various parameterizations of the covariance matrices of the Gaussian distributions, 10 models that differ in terms of their geometric assumptions relating to the volume, shape, and orientation of the clusters were tested. For example, the well-known Ward's model specifies that all clusters be spherical in shape (thus no orientation with respect to the coordinate axes) and of equal volume. Other models involve different assumptions. In contrast to standard clustering techniques, model-based cluster analysis will not automatically yield multiple clusters if the data do not provide evidence for them.

Using the EMclust command, we fit 10 different agglomeration models. The default output is for one to nine clusters for each model, so 90 different cluster solutions were examined. The goodness-of-fit index in Mclust is the Bayesian information criterion (BIC), which relates to the conditional probability that individual cases have been assigned to correct groups given the various model parameters. A higher BIC value indicates a better fit, and differences among BIC values for competing models indicate the relative strength of evidence favoring the model with the larger BIC. Guidelines from Raftery (1995, p. 139) describe the evidence as "weak" for models whose BIC values differ by only 0–2, but when BIC values differ by 10, the posterior odds favoring the model with the larger BIC are about 150:1 ("very strong" evidence).

Results

Model Fitting

The best Mclust solution was a model with five clusters in which the groups are of approximately equal volume and have elliptical shape, with the elliptoids' axes having equal orientation. For this model, the BIC value was –17,031.97. For the next best fitting model (which yielded seven clusters) the BIC value was –17,040.40, and the difference between these BIC values (8.43) is interpreted as providing "strong evidence" (Raftery, 1995, p. 139) in favor of the better fitting five-group solution.

Group 4 appeared to be a small ($n = 12$) outlier group distinguished by extremely low scores on the RR scale (mean z score = –3.79). This finding raised concerns about the reliability of this group's responses; thus, we examined mean scores on the PAI Negative Impression scale. This scale contains items that "present an exaggerated unfavorable impression or represent extremely bizarre and unlikely symptoms" (Morey, 1991/2007, p. 12). Only the mean score for Group 4 (73.25) was above the PAI cutoff ($T > 72$), suggesting possible deliberate distortion of responses. In light of concerns about invalid responding, we excluded this group from the analyses in the next section.

Description of Clusters

Z score profiles on clustering variables for Groups 1, 2, 3, and 5 are shown in Figure 1. Raw score means and standard deviations are shown in Table 1.[3] Multivariate analysis of variance and Wilk's Lambda = .069, $F(3, 27) = 108.201$, $p < .001$, followed by individual analyses of variance (ANOVAs) for each variable, revealed significant differences on all variables (all ps $< .001$) except RR ($p = .02$). Effect sizes (η_p^2) were largest for the CATS (.636), PCL-R Facet 2 (affective features, .542), and HA (.389); effect sizes were smallest for the RR (.014), ANX (.038), and PCL-R Facet 1 (interpersonal features, .052).

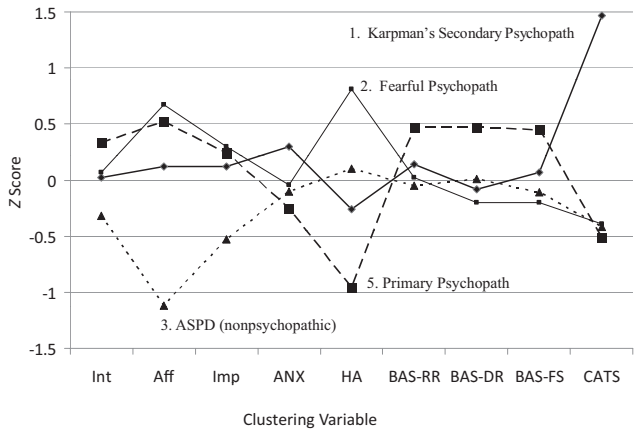

Figure 1. Cluster variable z score profiles for four emergent clusters (1, 2, 3, and 5). ASPD = antisocial personality disorder; Int = interpersonal facet from the Psychopathy Checklist–Revised (PCL-R); Aff = affective facet from the PCL-R; Imp = impulsive lifestyle facet from the PCL-R; ANX = Anxiety scale from the Personality Assessment Inventory; HA = Harm Avoidance scale from the Multidimensional Personality Questionnaire; BAS-RR = Reward Responsiveness scale from the behavioral activation system scales; BAS-DR = Drive scale of the BAS scales; BAS-FS = Fun Seeking scale of the BAS scales; CATS = Child Abuse and Trauma Scale.

Three of the cluster profiles were readily interpretable. Group 1 ($n = 153$) exhibited by far the highest score on the CATS but only modest scores on measures of internal variables (HA, RR, DR, and FS) having potential etiological significance for the development of antisocial traits. Substantial associated features of ASPD are also evident, with a slightly higher elevation on PCL-R Facet 3 relative to Facets 1 and 2. This configuration, along with having the highest score of all groups on ANX, suggests that Group 1 is consistent with Karpman's (1941, 1948) model of secondary psychopathy.

Bypassing Group 2 for the moment, we found that Group 3 participants ($n = 195$) had very low scores on PCL-R Facets 1–3. Thus, this cluster represents an ASPD group without significant associated features of the disorder. We labeled this cluster *the nonpsychopathic ASPD group.*

Group 5 ($n = 141$) manifested a pattern of associated features consistent with the theoretical primary psychopathy profile, including somewhat higher scores on Facets 1 and 2 than on Facet 3 and the lowest score on both the HA scale and the ANX scale. The elevated scores on the scales measuring reward sensitivity (the RR, DR, and FS) indicate a profile consistent with Blackburn's (2006) model of primary psychopathy.

As shown in Figure 1, Cluster 2 ($n = 190$) has a profile that does not conform to any expected subtype. This group has substantial associated features of ASPD, particularly the affective (Facet 2) and impulsive lifestyle (Facet 3) traits. Cluster 2 is particularly distinguished by its prominent elevation on the HA. We provisionally labeled this unpredicted cluster *the fearful group.*

Table 2 provides social and demographic information for the clusters. The mean age of participants in the clusters did not differ significantly, $F(3, 675) = 1.71$, $p = .16$. The groups did differ significantly in terms of mean IQ scores, $F(3, 675) = 4.39$, $p < .005$; racial distribution, $\chi^2(3) = 30.81$, $p < .001$; and proportion of members in prison versus residential drug treatment programs, $\chi^2(3) = 27.73$, $p < .001$.

Results of Planned Comparisons on Measures of Cluster Validation Variables

Our analyses focused on the planned comparisons between the primary and secondary ASPD/psychopathic clusters.[4] Table 3 presents mean scores and standard deviations for self-report criterion variables relevant to Hypotheses 1–4. Results were consistent with predictions for Hypotheses 1–3. Significantly higher mean scores were observed in the secondary group for measures of internalizing symptomatology, $t(292) = 5.21$, $p < .001$, $d = 0.61$, exter-

[3] The groups' raw score means on PCL-R Facet 4 were as follows: Group 1, $M = 7.58$ ($SD = 1.80$); Group 2, $M = 7.45$ ($SD = 1.90$); Group 3, $M = 6.35$ ($SD = 2.22$); Group 5, $M = 7.26$ ($SD = 1.94$). In a one-way ANOVA, these means differed significantly ($p < .001$). A post hoc comparison (least significant difference) indicated that the means for Groups 1, 2, and 5 did not differ, but each of these differed significantly from the mean of Group 3.

[4] Due to space limitations, we do not present the results of exploratory analyses involving differences among all four clusters. These results may be obtained from Norman G. Poythress.

Table 1
Cluster Variable Raw Score Means and Standard Deviations for ASPD Clusters 1, 2, 3, and 5

	1. Secondary		2. Fearful		3. Nonpsychopathic ASPD		5. Primary	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
PCL-R f1	4.48	2.20	4.57	2.19	3.74	2.01	5.13	2.09
PCL-R f2	5.34	1.91	6.53	1.30	2.68	1.38	6.21	1.22
PCL-R f3	7.50	1.57	7.81	1.42	6.39	1.79	7.71	1.56
ANX	59.94	12.20	55.90	11.01	55.28	10.67	53.47	11.01
HA	13.89	5.27	20.32	4.19	16.04	5.53	9.65	3.51
RR	18.30	1.87	18.01	2.09	17.86	2.07	18.48	1.79
DR	12.22	2.72	11.91	2.66	12.45	2.45	13.69	2.20
FS	12.67	2.43	12.01	2.41	12.23	2.08	13.58	2.27
CATS	131.75	16.92	79.39	16.01	78.41	18.10	75.96	16.94

Note. ASPD = antisocial personality disorder; PCL-R f1, f2, f3 = Facets 1–3 (interpersonal, affective, impulsive lifestyle features, respectively) from the Psychopathy Checklist–Revised; ANX = Anxiety scale from the Personality Assessment Inventory; HA = Harm Avoidance scale from the Multidimensional Personality Questionnaire; RR, DR, FS = Reward Responsiveness, Drive, and Fun Seeking scales, respectively, from the Behavioral Activation System scales; CATS = Child Abuse and Trauma Scale.

nalizing symptomatology, $t(292) = 3.75$, $p < .001$, $d = 0.44$, and impulsivity, $t(292) = 1.92$, $p < .05$, $d = 0.22$. However, for Hypothesis 4, interpersonal dominance failed to differentiate significantly between the two groups, $t(292) = 1.25$, $p = ns$, $d = 0.15$, although the difference was in the predicted direction.

Hypothesis 5. For the go–no-go passive avoidance learning task, the dependent variable was the number of errors of commission (pressing the button in response to S–) during the second block of 40 trials. Prior to data analysis, we excluded 37 cases with scores less than 2 standard deviations below the mean to control for potential careless responding or lack of effort. As predicted, the primary group committed a significantly higher number of commission errors than did the secondary group, $t(277) = -2.30$, $p < .05$, $d = 0.27$.

Hypothesis 6. We predicted that the secondary group would manifest less disruptive behavior in treatment as well as better treatment engagement and motivation. As predicted, the secondary group manifested fewer unexcused absences ($d = -0.56$) and higher treatment motivation, as measured by two indices (counselor-rated motivation $d = 0.56$; PAI Treatment Rejection $d = 0.50$) than did the primary group. However, contrary to the hypotheses, the two groups did not differ significantly in counselor-rated levels of disruptive behavior or skill mastery, although differences were in the predicted direction (see Table 4).

Also contrary to the hypotheses, chi-square analyses revealed no significant differences between the percentages of individuals in these groups deemed "treatment successes" on the basis of either subjective (clinician ratings) or objective (program level achieved) indices. Regarding posttreatment recidivism, the percentage of each cluster that was arrested within 1 year at risk is presented at the bottom of Table 4. Chi-square analyses revealed no significant differences between the primary and secondary groups. To assess the association between treatment involvement and arrests, we performed a logistic regression to determine whether primary versus secondary group membership moderated the effect of treatment on the likelihood of arrest during the year after enrollment. The dependent variable was any arrest during the first year at risk for arrest. The first independent variables entered were treatment agency (to control for site differences in effectiveness), days in treatment (the index of treatment involvement), and cluster membership; the second independent variables entered were the key variable of interest and the interaction between cluster membership and days in treatment. Neither the group membership term nor its interaction with treatment involvement was significant.

Hypothesis 7. The upper half of Table 5 summarizes group rates for institutional infraction variables for those cluster members who were new admission inmates. Because new admission inmates represent only a small portion of our clustering sample, the group sizes are much smaller than they are for the remaining criterion measures (combined $n = 153$), which precluded the use of logistic regression analyses controlling for site differences.

Table 2
Social-Demographic Features of ASPD Clusters 1, 2, 3, and 5

Variable	1. Secondary	2. Fearful	3. Nonpsychopathic ASPD	5. Primary
Age in years: *M* (*SD*)	30.19 (6.44)	30.29 (6.00)	30.30 (6.06)	28.97 (5.73)
IQ: *M* (*SD*)	96.22 (8.39)	92.95 (8.97)	95.59 (9.59)	94.72 (9.22)
Race (%)				
Caucasian	75.3	49.5	71.4	68.3
African American	23.8	48.9	28.2	31.2
In prison (%)	47.1	64.7	40.0	58.9

Note. ASPD = antisocial personality disorder.

Table 3
Means (and Standard Deviations) for ASPD Clusters 1, 2, 3, and 5 on Criterion Personality Scales

Scale	1. Secondary	2. Fearful	3. Nonpsychopathic ASPD	5. Primary
PAI internalizing problems	60.97 (11.66)	56.32 (9.46)	54.65 (9.12)	54.55 (8.72)
PAI externalizing problems	72.94 (9.14)	67.79 (8.70)	67.46 (7.77)	69.05 (8.60)
BIS-11	75.99 (12.15)	73.52 (12.21)	72.98 (11.03)	73.29 (11.79)
PAI: Dominance	56.69 (10.88)	54.07 (10.29)	53.78 (10.37)	58.16 (9.02)

Note. ASPD = antisocial personality disorder; PAI = Personality Assessment Inventory; BIS-11 = Barratt Impulsivity Scale–Version 11.

Consistent with predictions, planned comparisons revealed a significant difference in the proportion of primary versus secondary group members who were cited for general infractions, $\chi^2(n = 67) = 3.23$, $p < .05$ (one-tailed). There was also a modest difference supporting the hypothesis that the secondary group would be more prone to aggressive misconduct while incarcerated, $\chi^2(n = 67) = 3.24$, $p < .05$ (one-tailed).

Hypothesis 8. The lower half of Table 5 summarizes findings regarding general and violent recidivism over the year following either release into the community (for all prisoners and those substance abusers in the closed-unit Texas treatment program) or recruitment into the research project (for substance abuse patients receiving outpatient treatment). Despite comparable follow-up periods, rates of recidivism varied widely across sites. Therefore, multiple logistic regression analyses were performed in which site was entered on the first step of the analyses to control for differences in recidivism accounted for by this variable.

Next we performed planned comparisons by entering the cluster membership variable (primary or secondary). Results for Step 2 of the logistic regression indicated no differences in general recidivism between the primary and secondary clusters, $\Delta\chi^2(n = 211) = 0.37$, *ns* (one-tailed). For violent recidivism, only a trend in the predicted direction was noted in this (low base rate) category, $\Delta\chi^2(n = 211) = 2.39$, $p = .06$.

Post hoc analyses. Cluster 2 was distinguished by substantial psychopathic features in addition to a substantial elevation on the HA, suggesting a fearful temperament. This profile did not conform to any a priori expectations. However, some of the findings in Table 2 suggest that this fearful group may be representative of Mealey's (1995) disadvantaged sociopath (secondary psychopath), that is, Mealey identified lower intelligence and lower socioeconomic status group as risk factors for secondary psychopathy. She also identified other social and contextual risk factors, including being raised in a disrupted or nontraditional family structure (i.e., single parent) or a family with a large number of siblings. To investigate these issues, we coded participants' responses to questions from the PCL-R concerning family size and structure. Regarding family disruption, except for the secondary group (28.3%), a smaller proportion of the fearless group (33.3%) reported being raised in an intact, two-parent family than did any other group (nonpsychopathic ASPD group, 39.1%; primary group, 41.3%), a finding in the expected direction but not statistically significant, $\chi^2 (n = 464) = 5.62$, $p = ns$. A greater proportion of the fearful group (62.4%) reported being raised in a large family, defined as having four or more siblings in the home, than did the secondary group (49%), the nonpsychopathic ASPD group (49.7%), or the primary group (51.8%), $\chi^2(n = 646) = 7.98$, $p < .05$. These additional findings must be considered cautiously because they were not planned and because substantial data were missing. However, along with the sociodemographic differences observed in Table 2, they are suggestive that the fearful group may map onto Mealey's secondary subtype.

Discussion

The role of the personality features of psychopathy identified by Cleckley (1941/1982) in the conceptualization and operationalization of ASPD and cognate disorders has varied substantially across editions of the *DSM*. They were featured prominently in the *DSM–II*, were conspicuously absent in the *DSM–III* and the *DSM–*

Table 4
ASPD Cluster Differences on Treatment Participation and Outcomes Variables

Variable	1. Secondary (*ns* = 44–49)	2. Fearful (*ns* = 38–43)	3. Nonpsychopathic ASPD (*ns* = 65–69)	5. Primary (*ns* = 30–32)
Clinician ratings of treatment participation: *M* (*SD*)				
Unexcused absences	1.93 (0.84)	2.09 (1.04)	1.94 (0.83)	2.45 (0.99)
Disruptive in group	2.00 (0.93)	2.09 (0.94)	1.71 (0.82)	2.31 (0.86)
Motivation	4.59 (1.51)	4.46 (1.65)	4.69 (1.65)	3.78 (1.33)
Skill mastery	3.72 (1.65)	3.98 (1.51)	4.17 (1.59)	3.28 (1.61)
Self-reported treatment motivation: *M* (*SD*)				
PAI: RXR scale	32.46 (6.38)	34.35 (8.48)	34.06 (7.31)	35.55 (7.80)
Treatment outcomes (% successful)				
Subjective (clinician rating)	44	56	59	41
Objective (program steps completed)	55	71	63	60
Rearrested within 1 year (%)	45	37	44	55

Note. ASPD = antisocial personality disorder; PAI: RXR = Treatment Rejection scale of the Personality Assessment Inventory.

Table 5
ASPD Cluster Differences in Rates of Institutional Infractions and Recidivism

Variable	1. Secondary	2. Fearful	3. Nonpsychopathic ASPD	5. Primary
Institutional infraction rates for new admission prison inmates	$n = 31$	$n = 57$	$n = 29$	$n = 36$
General (%)	58	46	41	36
Aggressive (%)	36	33	31	14
Recidivism type	$n = 111$	$n = 111$	$n = 155$	$n = 100$
General (%)	41	45	36	45
Violent (%)	10	10	1	5

Note. ASPD = antisocial personality disorder.

III–R, and were partially restored as associated features of ASPD in the *DSM–IV*.

Following the strategy proposed by Pilkonis and Klein (1998), we used model-based cluster analysis to disaggregate a large sample of offenders who met diagnostic criteria for *DSM–IV* ASPD, using as clustering variables the best validated measure of the associated features of ASPD, along with measures of internal and external constructs bearing theoretical implications for the etiology of psychopathic features and antisocial behavior. Our findings do not conclusively resolve the debate concerning whether ASPD and psychopathy are the same or different constructs. However, they do provide perhaps the most compelling empirical support to date for the contention that ASPD is heterogeneous and not isomorphic with primary psychopathy.

Most notably, we identified one cluster group (Group 3) that met *DSM–IV* criteria for ASPD but that did not exhibit markedly elevated scores on any aspect of the associated features indexed by the PCL-R, including the core interpersonal and affective features widely regarded as indexing primary psychopathy. Two additional clusters were observed to exhibit profiles that were highly similar to theoretically predicted profiles of primary and secondary psychopathy. Further, planned comparisons between these latter two groups yielded predicted differences on most of the external criterion measures that we selected to examine construct validity.

Specifically, compared with the primary group, the secondary group had higher mean scores on self-report measures of internalizing and externalizing psychopathology and impulsivity, fewer errors on a passive avoidance learning task, a higher incidence of both general and aggressive disciplinary problems in prison, and a trend ($p = .06$) toward a higher incidence of violent recidivism. Also as predicted, those secondary participants recruited in substance abuse treatment programs displayed less clinician-rated disruptive behavior and higher treatment motivation. Contrary to predictions, the secondary group did not score lower on a self-report measure of dominant interpersonal style, did not have a higher incidence of general recidivism, and did not have better drug abuse treatment outcomes than did the primary group.

The differences between the primary and secondary clusters were more consistent with theoretical predictions for measures of personality correlates but less consistent for socially relevant outcome measures. However, some of the latter analyses may have been underpowered due to small samples (i.e., the number of participants followed with respect to treatment outcomes), and low base rates of some behaviors (e.g., violent recidivism) may have made it difficult to detect significant differences. The failure to find differences in treatment outcomes may also have been affected by the limited scope of services provided to offenders in the drug treatment programs. The more extensive internalizing psychopathology and trauma histories reported by the secondary group suggest the need for treatment that focuses on issues (e.g., possible posttraumatic stress disorder) beyond the substance use problems that are the primary focus in these treatment programs. To the extent that successful treatment of drug problems may be conditioned in part on the diagnosis and treatment of these additional problems, then differences in drug treatment outcomes between the primary and secondary groups would be diminished.

Overall, the present results provide evidence that subgroups of offenders who meet ASPD criteria differ significantly in their patterns of associated features of the disorder and that some of the empirically derived ASPD clusters exhibit profiles that match theoretical subtypes described in the literature that dates back to Karpman (1941). However, caveats are in order regarding the emergent clusters. First, the cross-sectional design of the study and reliance on self-report measures to index key internal variables—particularly fearless temperament—precludes a demonstration that these features were present early in development, when their presence would be expected to interfere with the socialization processes designed to instill prosocial attitudes and the development of conscience. Second, the observed groups may not correspond precisely with the theoretical subtypes represented by the labels we assigned to them. As one thoughtful reviewer noted, some individuals with a predisposition to a fearless temperament may also have been victims of childhood abuse that resulted, in part, because they were difficult to rear. Such individuals might have qualified for either the primary or secondary designation and, depending on their endorsements on measures of clustering variables, could have been assigned to either.

Second, our investigation was not intended to exhaustively test for all potential subtypes of ASPD. We intentionally focused on a small number of specific variants that had been best described in the literature. Additional variants may be ascertained in future investigations that employ an expanded array of clustering variables that represent additional internal and external influences on the development of personality and antisocial behavior. In particular, future investigations will hopefully clarify the nature and validity of the unexpected fearful type that emerged in our study. Our tentative hypothesis that this group represents Mealey's (1995) secondary types suggests that additional clustering variables that assess aspects of social disadvantage should be considered. An alternative hypothesis that awaits further investigation is

that the elevated HA score of our fearful group may be a measurement artifact related to previously reported race differences in the endorsement of items related to sensation seeking (Kurtz & Zuckerman, 1978), a construct associated with (reversed) HA.

Although we found that prisoners with ASPD can be parsed into relatively homogeneous clusters, we do not maintain that these groups constitute true natural categorical subtypes (i.e., taxa). Cloninger, Bayon, and Przybeck (1998) asserted that "personality disorders are not discrete diseases but are clinically distinct and relatively stable configurations of multiple quantitative traits" (pp. 19–20), a position also endorsed by Pilkonis and Klein (1998). Pending further investigations that may provide further insights into the nature of empirically derived variants of ASPD, we adopt this position as well.

Additional limitations of our study include the use of self-report scales to measure some important variables. Because dishonesty and deception are characteristic of many individuals with ASPD, responses to these measures may not always be veridical. However, on the PAI Negative Impression scale, which measures the tendency to create negative impression, none of the clusters scored in the range suggestive of possible deceptive responding (except for Cluster 4, $n = 12$, which was excluded from analyses). In addition to concerns about deception by prisoners with ASPD, use of a retrospective measure of abuse and trauma poses potential problems related to long-term memory for childhood events. Future investigations should consider alternative measures (e.g., official records) to assess abuse or neglect history. In addition, future studies should incorporate psychophysiological indicators (e.g., cortisol levels) and differences in brain structure (i.e., brain imaging) that may bear implications for a better understanding of this disorder.

This study also had several strengths, including a large sample and multisite recruitment of offenders from both prisons and community-corrections programs, its reliance on theory to select clustering variables, a priori specification of the clustering profiles expected to emerge, and the broad array of external validating variables representing diverse measurement domains (self-report, laboratory task, clinician ratings, official records). The use of model-based clustering is also a strength. Traditional clustering approaches often employ a single agglomeration model (e.g., Ward's) to generate clusters and permit considerable investigator discretion in the determination of the optimal number of clusters to analyze or interpret. In contrast, model-based clustering evaluates 10 different agglomeration models (Ward's and nine others) and employs a statistical fit index to identify the best fitting model. Model-based clustering can, and will, designate a one-cluster solution if the BIC evidence indicates that there is no better fitting multicluster solution. Thus, this approach limits the likelihood that investigators will select an a priori preferred solution in the face of statistical evidence favoring other solutions.

Our findings inform ongoing debates about the widely discussed but poorly researched heterogeneity of ASPD and the atheoretical orientation of the *DSM* with respect to ASPD (Lilienfeld, 1994; Lykken, 1995). The American Psychiatric Association is in the process of developing recommendations for possible changes to the *DSM*. The ADHD and Disruptive Behaviors Disorder Workgroup is considering, on the basis of reviews of existing research and secondary data analyses, a recommendation regarding a "specifier" to the diagnosis of conduct disorder (Frick & Moffitt, 2009). The proposed specifier would distinguish among youths who meet full criteria for a conduct disorder diagnosis in terms of the presence or absence of significant callous and unemotional traits. Pending further investigations of differences among adults who meet criteria for ASPD, our study provides analogous evidence that potentially meaningful ASPD subgroups of adults may be distinguished on a similar basis, namely, the presence or absence of specific patterns of associated features, and thus may inform deliberations about possible changes to this diagnosis.

References

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Ammons, R. B., & Ammons, C. H. (1962). The Quick Test (QT): Provisional manual. *Psychological Reports, 11,* 111–161.

Blackburn, R. (1987). Two scales for the assessment of personality disorder in antisocial populations. *Personality and Individual Differences, 8,* 81–93.

Blackburn, R. (2006). Other theoretical models of psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 35–57). New York, NY: Guilford Press.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67,* 319–333.

Cleckley, H. (1982). *The mask of sanity.* St. Louis, MO: Mosby. (Original work published 1941)

Cloninger, C. R., Bayon, C., & Przybeck, T. R. (1998). Epidemiology and Axis I comorbidity of antisocial personality. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 12–21). New York, NY: Wiley.

Edens, J. F., Poythress, N. G., & Lilienfeld, S. O. (1999). Identifying inmates at risk for disciplinary infractions: A comparison of two measures of psychopathy. *Behavioral Sciences and the Law, 17,* 435–443.

Edens, J. F., & Ruiz, M. A. (2005). *PAI interpretive report for correctional settings (PAI-CS).* Lutz, FL: Psychological Assessment Resources.

First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. (1996). *User's guide for the Structured Clinical Interview for DSM–IV Axis II Personality Disorders (SCID-II, version 2.0, July 1996 final version).* New York, NY: Biometrics Research Department, New York State Psychiatric Institute.

Fraley, C., & Raftery, A. E. (2003). Enhanced model-based clustering, density estimation, and discriminant analysis software: Mclust. *Journal of Classification, 20,* 263–286.

Frick, P. J., & Moffitt, T. E. (2009). *A proposal to the DSM–V Childhood Disorders and the ADHD and Disruptive Behavior Disorders Work Groups to include a specifier to the diagnosis of conduct disorder based on the presence of callous-unemotional traits.* Unpublished report.

Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system.* Oxford, England: Oxford University Press.

Hare, R. D. (1980). A research scale for the assessment of psychopathy in criminal populations. *Personality and Individual Differences, 1,* 111–119.

Hare, R. D. (1998). Psychopaths and their nature: Implications for the mental health and criminal justice systems. In T. Millon, E. Simonsen, M. Birket-Smith, & R. D. Davis (Eds.), *Psychopathy: Antisocial, crim-*

inal and violent behavior (pp. 188–212). New York, NY: Guilford Press.

Hare, R. D. (2003). *The Hare Psychopathy Checklist–Revised.* Toronto, Ontario, Canada: Multi-Health Systems. (Original work published 1991)

Henry, B., & Moffitt, T. E. (1998). Neuropsychological and neuroimaging studies of juvenile delinquency and adult criminal behavior. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 280–288). New York, NY: Wiley.

Insightful. (2001). *S-PLUS 6.2 for Windows: Programmer's guide.* Seattle, WA: Author.

Johns, J. (1997). *The basic reading inventory* (7th ed.). Dubuque, IA: Kendall/Hunt.

Karpman, B. (1941). On the need of separating psychopathy into two distinct clinical types: The symptomatic and the idiopathic. *Journal of Criminal Psychopathology, 3,* 112–137.

Karpman, B. (1946). Psychopathy in the scheme of human typology. *Journal of Nervous & Mental Disease, 103,* 276–288.

Karpman, B. (1948). The myth of the psychopathic personality. *American Journal of Psychiatry, 104,* 523–534.

Karpman, B. (1949). Psychopathy as a form of social parasitism: A comparative biological study. *Journal of Clinical Psychopathology, 10,* 160–194.

Kurtz, J. P., & Zuckerman, M. (1978). Race and sex differences on the Sensation Seeking Scales. *Psychological Reports, 43,* 529–530.

Lilienfeld, S. (1994). Conceptual problems in the assessment of psychopathy. *Clinical Psychology Review, 14,* 17–38.

Lilienfeld, S. O. (1998). Methodological advances and developments in the assessment of psychopathy. *Behaviour Research and Therapy, 36,* 99–125.

Lykken, D. (1995). *The antisocial personalities.* Hillsdale, NJ: Erlbaum.

Mealey, L. (1995). The sociobiology of sociopathy: An integrated evolutionary model. *Behavioral and Brain Sciences, 18,* 523–599.

Millon, T., & Davis, R. D. (1998). Ten subtypes of psychopathy. In T. Millon, E. Simonsen, M. Birket-Smit, & R. D. Davis, *Psychopathy: Antisocial, criminal, and violent behavior* (pp. 161–171). New York, NY: Guilford Press.

Morey, L. (2007). *Personality Assessment Inventory: Professional Manual.* Odessa, FL: Psychological Assessment Resources. (Original work published 1991)

Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology, 95,* 252–256.

Paris, J. (1998). A biopsychosocial model of psychopathy. In T. Millon, E. Simonsen, M. Birket-Smith, & R. D. Davis (Eds.), *Psychopathy: Antisocial, criminal and violent behavior* (pp. 277–287). New York, NY: Guilford Press.

Pilkonis, P. A., & Klein, K. R. (1998). Commentary on the assessment and diagnosis of antisocial behavior and personality. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 109–112). New York, NY: Wiley.

Porter, S. (1996). Without conscience or without active conscience? The etiology of psychopathy revisited. *Aggression and Violent Behavior, 1,* 179–189.

Raftery, A. (1995). Bayesian model selection in social research. *Sociological Methodology, 25,* 111–163.

R Development Core Team. (2009). *Manual for R: A language and environment for statistical computing.* Vienna, Austria: R Foundation for Statistical Computing.

Robins, L. N. (1966). *Deviant children grown up.* Baltimore, MD: Williams & Wilkins.

Ruiz, M. A., & Edens, J. F. (2008). Recovery and replication of internalizing and externalizing dimensions within the Personality Assessment Inventory. *Journal of Personality Assessment, 90,* 585–592.

Sanders, B., & Giolas, M. H. (1991). Dissociation and childhood trauma in psychologically disturbed adolescents. *American Journal of Psychiatry, 148,* 50–54.

Smith, S. S., & Newman, J. P. (1990). Alcohol and drug abuse/dependence disorders in psychopathic and nonpsychopathic criminal offenders. *Journal of Abnormal Psychology, 99,* 430–439.

Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology, 51,* 768–774.

Tellegen, A. (in press). *Manual for the Multidimensional Personality Questionnaire.* Minneapolis: University of Minnesota Press.

Walters, G. (2003). Predicting institutional adjustment and recidivism with the Psychopathy Checklist factor scores: A meta-analysis. *Law and Human Behavior, 27,* 541–558.

Widiger, T. A. (2000). Antisocial personality disorder. In A. E. Kazden (Ed.), *Encyclopedia of psychology* (Vol. 1, pp. 207–209). Washington, DC: American Psychological Association.

Widiger, T. A. (2006). Psychopathy and *DSM–IV* psychopathology. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 156–171). New York, NY: Guilford Press.

Widiger, T. A., Cadoret, R., Hare, R., Robins, L., Rutherford, M., Zanarini, M., . . . Frances, A. (1996). *DSM–IV* antisocial personality disorder field trial. *Journal of Abnormal Psychology, 105,* 3–16.

Widiger, T. A., & Corbitt, E. M. (1995). Antisocial personality disorder. In J. Livesley (Ed.), *The DSM-IV personality disorders* (pp. 103–126). New York, NY: Guilford Press.

Widiger, T. A., & Corbitt, E. M. (1997). Comorbidity of antisocial personality disorder with other personality disorders. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 75–82). New York, NY: Wiley.

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