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## The Construct Validity of Passive-Aggressive Personality Disorder

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### Abstract

Although Passive Aggressive personality disorder (PAPD) plays an important role in many theories of personality pathology, it was consigned to the appendix of the fourth edition of the DSM. The scientific basis of this decision has been questioned, but several controversies persist regarding PAPD, including its structure, content validity, overlap with other PDs, and relations to validating variables such as personality traits, childhood experiences, and clinically relevant correlates. This study examined these facets of PAPD's construct validity in a large clinical sample. Results suggest

that the construct is unidimensional, internally consistent, and reasonably stable. Furthermore, PAPD appears systematically related to borderline and narcissistic personality disorders, sets of personality traits, and childhood experiences consistent with several theoretical formulations, dysfunction, substance abuse disorders, and history of hospitalizations. Overall, results support the construct validity of PAPD.

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Passive Aggressive personality disorder (PAPD) was eliminated as an official psychiatric diagnosis and relegated to the appendix of the fourth edition of the DSM (American Psychiatric Association, 1994), even though passive aggressive behavior continues to play an important role in several theories of personality disorder (PD) (e.g., Benjamin, 1993; Millon, 1981; Morey, Hopwood, & Klein, 2007; Pretzer & Beck, 1996). Some authors have questioned the decision to remove PAPD from DSM-IV (e.g., Wetzler & Morey, 1999). In particular, Blashfield & Intoccia (2000) noted that this decision was made in the context of a dramatically expanding diagnostic system and increased research on some PDs, and that PAPD appears to meet the conventional standards for diagnostic inclusion (Blashfield, Sprock, & Fuller, 1990; Feighner et al., 1972; Robins & Guze, 1970) as well as most other PDs do.

The term *passive aggressive* originated in the American military to describe soldiers who did not comply with superiors' commands. Early theoretical development of passive aggressive clinical characteristics occurred in the psychoanalytic literature—for example, Abraham's (1924) description of melancholiacs or Fenichel's (1945) discussion of the oral character. Several contemporary authors have contributed to the theoretical foundation of PAPD. Benjamin's (1993) interpersonal theory posits that PAPD results from a developmental pattern involving punishments for anger, failure to submit, and efforts to establish autonomy, resulting in heightened power sensitivity. In Beck's cognitive theory (Pretzer & Beck, 1996), PAPD is also thought to occur because of beliefs related to power and autonomy. In particular, this theory holds that PAPD individuals view themselves as vulnerable to control and others as demanding and interfering. Kernberg's psychoanalytic theory (1976) emphasizes the existence of a moderately integrated superego which can only modulate the ego in a somewhat primitive manner, resulting in pathological defenses. The most directly pertinent of these to PAPD appear to be "partial [passive] expressions of instinctual impulses [such as aggression]" (p. 144–145, bracketed material added). Little research has empirically assessed the congruence of these theories with PAPD, but they do appear to formulate similar etiological and phenomenological hypotheses. Specifically, each holds that passive aggressive behavior results from a disruption in learning how to navigate hierarchical relationships during childhood. This disruption leads to ineffective self-assertion and, thereby, negativistic mood states and cognitions.

One controversy that apparently contributed to the removal of PAPD from DSM-IV involved diagnostic content. Although the list of characteristics signifying passive aggressive behavior is substantial (McCann, 1988), Millon (1981) viewed passive aggressive behavior as a single symptom diagnostically embedded in a larger syndromal context, which he labeled negativistic. Millon's negativistic personality disorder includes characteristics in addition to passive aggressive behavior, such as irritability, anger, pessimism, and discontent (Millon, 1981). In the end, the authors of DSM-IV Axis II, including Millon, added characteristics reflecting this wider operationalization of the disorder, and removed several DSM-III symptoms that targeted passive aggressive behavior more directly. This group also agreed to move PAPD to the appendix, concluding that further study was needed to justify including PAPD as an official diagnosis.

Research implicates both genetic and environmental factors in the genesis of PAPD. The heritability of the disorder was estimated at .50 in school-age twins (Coolidge, Thede, & Jang, 2001; see also Czajkowski et al., 2008). Consistent with various theoretical formulations, PAPD is also associated with environmental factors, including ineffective parenting behavior

(Johnson et al., 2008) in general as well as child abuse (Grover et al., 2007), harsh/aversive parenting (Johnson et al., 2006a), and neglect (Johnson et al., 2000) in particular.

Data on the structure and reliability of PAPD as represented in the DSM-IV appendix are mixed. Rotenstein and colleagues (2007) reported that a two-factor structure for the disorder most parsimoniously described their patient data. The internal consistency of PAPD symptoms in their data was quite low (.50), very likely as a result of this structure. Interestingly, one of these factors reflects the original conception of PAPD (viz., passive resistance), and the other reflects additional symptoms added to widen the diagnosis to Negativistic PD (viz., resentment). However, using a different estimation method and a more severe sample, Fossati and colleagues (2000) reported unidimensionality of PAPD and acceptable internal consistency (.85). Thus, the structure of PAPD remains an important and unresolved question. Data on the temporal stability of PAPD are also limited.

PAPD appears to have prevalence rates similar to other PDs in clinical (Morey, 1988; Widiger & Rogers, 1989) and community (Torgersen, Kringlen, & Cramer, 2001) samples. PAPD is also linked to a host of external validating markers. For example, it is associated with an increased risk for substance use (Cohen et al., 2007; Franken & Hendriks, 2000), anxiety disorder (Johnson et al., 2006b), depression (Kasen et al., 2001), and narcissistic (Fossati et al., 2000; Morey, 1988; Rotenstein et al., 2007) and borderline PDs (Czajkowski et al., 2008; Grilo, Sanislow, & McGlashan, 2002; Morey, 1988; Rotenstein et al.). Little is known about effective treatments for PAPD. Although data suggest that fluoxetine reduces passive aggressive symptoms among depressed patients (Fava et al., 2002), systematic studies of psychotherapy for PAPD are rare and no firm conclusions can be drawn.

Bradley, Shedler, and Westen (2006) found that clinician-rated PAPD personality characteristics converged with ratings of DSM-IV PAPD symptoms but had very limited overlap with other PDs. Furthermore, analyses of their data yielded a distinct group of patients who had classic PAPD characteristics. These authors concluded that their data suggest clinicians tend to regard PAPD as being uniquely associated with a particular pattern of personality characteristics. Consistent with theoretical articulations by Benjamin, Beck, and others, PAPD is more strongly associated with autonomy/agency than sociotropy/communion personality dimensions (Morey, 1985; Morse, Robins, & Gittes-Fox, 2002) and has been linked to authority conflicts among adolescents (Vereycken, Vertommen & Corvelyn, 2002). However, research has been limited; the relations of PAPD to other common models of personality have not been thoroughly tested.

The purpose of this study was to further assess the construct validity (i.e., the extent to which a measurement method corresponds to a natural construct, as demonstrated by various indicators of reliability and validity) of PAPD and to address previously inconsistent or controversial findings in the literature. One- and two-factor models were compared, and the internal consistency of diagnostic criteria was computed to address mixed findings regarding the unidimensionality of PAPD. Criterion prevalence and factor pattern coefficients were computed to examine the content coverage of diagnostic criteria. To address limited data on the stability of the disorder, test-retest correlations of the diagnostic symptom count were computed across two and four years of prospective follow-up. To address previous suggestions that PAPD is best considered a subtype of other PDs (e.g., narcissistic; Fossati et al., 2000), its overlap with other PDs was investigated. Finally, this study assessed a wider net of external variables previously linked to PAPD, particularly with regard to personality traits, childhood etiological factors, and clinical outcomes, than has been previously the case in single studies. Given previous research and theoretical formulations, it was anticipated that PAPD would be related to narcissistic and borderline PDs, negative affect and traits related to power, autonomy,

and negative interpersonal interactions, depression, anxiety, substance use, and negative childhood experiences.

## METHOD

### Participants

Participants were 733 patients with either PDs ( $n = 629$ ) or major depression without PD ( $n = 104$ ) at baseline, recruited from multiple inpatient and outpatient clinical sites for the Collaborative Longitudinal Personality disorder Study (CLPS) project (see Gunderson, et al., 2000, for sample details<sup>1</sup>). The average age of participants was 32.5 ( $SD = 8.1$ ; range = 18–45). Women represented 64% ( $n = 467$ ) of the sample; 69% ( $n = 505$ ) of participants were white, 15% ( $n = 108$ ) were black, 13% ( $n = 94$ ) were Hispanic, 2% ( $n = 17$ ) were Asian, and the rest ( $n = 9$ ) reported other ethnicities. PAPD was unrelated to demographic variables ( $p$ 's  $> .10$ ).

### Measures

**Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV)**—The DIPD-IV (Zanarini, et al., 1996) is a semi-structured interview that assesses PD criteria including PAPD, which to count toward the diagnosis must be continuously present over at least the previous two years and characteristic of most of adult life. The median inter-rater correlation of dimensional PAPD symptoms at baseline was .93; the 10-day test-retest reliability was .76 (Zanarini, et al., 2000). Notably, these values are similar to those of other PD diagnoses in the current ( $Mdn$  inter-rater  $r = .89$ ;  $Mdn$  test-retest = .77) and previous (e.g., Grilo, Añez, & McGlashan, 2003) samples. PAPD was also assessed 2 ( $n = 598$ ) and 4 years ( $n = 550$ ) following the baseline evaluation. Baseline PAPD severity (i.e., symptom counts) did not significantly differ ( $p > .10$ ) for patients who persisted relative to those who dropped out of the study.

**NEO Personality Inventory, Revised (NEO-PI-R)**—The NEO-PI-R (Costa & McCrae, 1992) was designed to comprehensively assess the five-factor model (FFM) of personality. Internal consistency reliabilities for the five traits (i.e., neuroticism, extroversion, openness to experience, agreeableness, and conscientiousness) in this sample ranged from .87 to .92 (Morey et al., 2002). The five trait scores represented dependent variables in the current study.

**Schedule for Nonadaptive and Adaptive Personality (SNAP)**—The SNAP (Clark, 1993) is a 375-item self-report questionnaire designed to assess personality traits in both the higher-order/temperamental and lower-order/ abnormal range. Internal consistency in our study sample was consistent with results described in the SNAP manual (Clark, 1993): medians of .89 for the higher order temperament scales and .84 for the lower order trait scales (Morey et al., 2003). SNAP traits were used as dependent variables.

**Longitudinal Interval Follow-Up Examination (LIFE)**—The LIFE (Keller et al., 1987), a structured interview, assesses functioning in interpersonal, recreational, and occupational domains. Baseline functioning scores were combined with self-reported functioning scores as described below. History of psychiatric hospitalizations and presence of suicidal behavior (including overt attempts and self-harming gestures) during the four years following baseline assessment were also measured with the LIFE.

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<sup>1</sup>Gunderson and colleagues (2000) describe data for 668 baseline participants; 65 participants were later included in the sample to increase the representation of individuals with ethnic minority status, and those individuals are included in the present report.

**Social Adjustment Scale, Self-Report (SAS-SR)**—The SAS-SR (Weissman & Bothwell, 1976), a self-report instrument with adequate psychometric properties, yields estimates of interpersonal, occupational, and recreational functioning. Median internal consistency reliability from CLPS baseline data for these composites was .70. Baseline domain scores from the SAS-SR were factor analyzed in combination with those from the LIFE. Three factors emerged with eigenvalues  $> 1$ , and after oblique rotation, structure coefficients suggested that these factors represented social, work, and recreational domains free from method effects. Standardized factor scores were used in this study to represent these functional domains.

**Childhood Experiences Questionnaire-Revised (CEQ-R)**—The CEQ-R (Zanarini et al., 1989), a clinician-rated interview, was administered at baseline to assess a range of retrospectively reported experiences during childhood and adolescence (ages 0–17). Several composites were constructed from CEQ-R items for this study to represent broad and reliable indicators of caretaker neglect ( $k = 18$ , Cronbach's  $\alpha = .92$ ), caretaker abuse ( $k = 30$ ,  $\alpha = .92$ ), and witnessing violence ( $k = 9$ ,  $\alpha = .84$ ).

**Structured Clinical Interview for DSM-IV Axis I (SCID-I)**—The SCID-I (First et al., 1996) is a structured interview assessing DSM-IV Axis I disorders. The presence of any baseline mood, anxiety, and substance use disorders were used as dependent variables in this study.

## Analyses

All analyses used diagnostic symptom counts rather than categorical cutoffs for PDs because of empirical evidence from this (Morey et al., 2007) and other (Widiger & Simonsen, 2005) samples that PDs are dimensional constructs. The structure of PAPD was tested using maximum likelihood and weighted least squares confirmatory factor analysis (CFA). In particular, goodness of fit of a one-factor model relative to the two-factor model observed by Rotenstein and colleagues (2007) was assessed. Because of controversies around covariance modeling dichotomous items (e.g., West, Finch, & Curran, 1995) and the use of different methods to analyze the structure of PAPD by Fossati and colleagues (2000; weighted least squares) and Rotenstein et al., (maximum likelihood), both methods were used. The Rotenstein 2-factor model was compared to a one-factor model; no other multidimensional models were investigated. Cronbach's alpha internal consistency and Pearson correlation stability coefficients were then computed for PAPD. Endorsement rates and factor coefficients were computed to assess the validity and prevalence of PAPD criteria.

Diagnostic co-occurrence was assessed with zero-order correlations between DSM-IV PDs and PAPD. To assess the unique relations between PAPD and other PDs, a multiple regression model was constructed in which DSM-IV PDs predicted PAPD. Correlations were also computed between PAPD and FFM and SNAP personality traits. Two more regression models were constructed to test unique trait-PAPD relations, one with FFM traits as predictors and the other using SNAP traits. Correlations were also computed between PAPD and multiple validating variables that were selected based on previous research. These included functioning composites, psychiatric disorders, history of hospitalization, prospective suicide gestures, and childhood experiences. Finally, to test the incremental validity of PAPD, hierarchical regression models in which PAPD was entered after the 10 official PDs in the prediction of personality traits and other validating variables were constructed. Beta coefficients for PAPD in models in which it provided a significant increase in model variance explained represented this increment. A Type I error rate of .01 was used to account for multiple hypothesis tests, and effect sizes were computed for all analyses.

## RESULTS

CFA results for the unidimensional model of PAPD symptoms suggested reasonable fit using both maximum likelihood ( $\chi^2_{(14)} = 55.88, p < .001, CFI = .93, RMSEA = .06$ ) and weighted least squares (RMR = .01, AGFI = .98) estimation methods. Although the two-factor model improved the fit somewhat in the maximum likelihood model according to the  $\chi^2$  test ( $\chi^2_{(1)} \text{ difference} = 11.18, p < .001$ ), other fit indices did not change sufficiently to indicate that this more complex model substantially improved description of the data ( $\chi^2_{(13)} = 44.70, p < .001, CFI = .94, RMSEA = .06$ ). (Hu & Bentler, 1998). Furthermore, the coefficients for the weighted least squares model did not differ. As such, the unidimensional model was considered the more parsimonious representation. The internal consistency of PAPD criteria was .69 and the mean corrected item-total correlation was .40, acceptable values given the number of symptoms.

As shown in Table 1, factor coefficients suggested that each criterion significantly contributed to the diagnosis. Each PAPD criterion was endorsed by between 13 and 30% of patients, indicating that each symptom was represented in a meaningful proportion of this clinical sample. Retest correlations over 2- ( $r = .46$ ) and 4-year ( $r = .41$ ) intervals reveal comparable if somewhat lower stability than for other PDs in this sample (Mean of 10 official PDs = .59 at 2 years and .47 at 4 years; Morey et al., 2007).

Table 2 shows the relations of PAPD to personality disorders and traits. Amongst DSM-IV personality disorders, PAPD is most similar to Cluster B PDs, and regression analyses showed that it had the strongest unique relationships with borderline and narcissistic PDs. Zero-order correlations suggested substantial relations with a host of traits, particularly those related to negative affect and a disagreeable interpersonal style. Regression analyses in which traits predicted PAPD clarified these relations. Unique relations were observed for FFM neuroticism, disagreeableness, and SNAP negative temperament, manipulateness, mistrust, and aggression. Table 2 also shows that PAPD incremented other PDs in predicting lower agreeableness and conscientiousness and higher neuroticism, negative temperament, manipulateness, and aggression.

Associations between PAPD and functioning variables are depicted in Table 3. Zero-order correlations indicate that PAPD negatively affects functioning in social, work, and recreational domains. PAPD was significantly and positively associated with substance use disorders and negative childhood experiences. PAPD was not significantly related to mood and anxiety disorders or suicidal behaviors at a Type I error rate of .01, although there were trends (i.e.,  $p < .05$ ) for mood disorder ( $r = .09$ ) and suicidal behaviors ( $r = .08$ ). PAPD only significantly incremented the other PDs in predicting work functioning ( $\beta = -.10$ ); its increment of other validating variables was not statistically significant. However, it is important to note that incremental validity is a fairly stringent yardstick: three other PDs (paranoid, dependent, and histrionic) failed to increment other PDs to predict any of these variables, and only borderline significantly incremented other PDs in predicting more than half of them.

## DISCUSSION

This study examined the construct validity of PAPD in a large clinical sample. Analyses focused on controversies with regard to the construct, such as unidimensionality, internal consistency, criterion prevalence, overlap with other PDs, and bivariate, unique, and incremental relations to external validators. Results generally support the construct validity of DSM-IV PAPD.

Analyses suggested that PAPD is unidimensional. Rotenstein and colleagues (2007) interpreted their data as supporting a two-factor model despite similar fit statistics to those in the current



data. In both samples, the  $\chi^2$  difference test suggested improved fit in the less parsimonious model, yet minimal improvement was observed on the other fit indices (current sample differences for CFI = .01; RMSEA = .00; Rotenstein et al., differences for CFI = .02; RMSEA = .01). Furthermore, overall fit statistics were very similar across both models in both samples. Our interpretation of unidimensionality was based on the common observation that the  $\chi^2$  test is overly sensitive to poor model fit, and consequent recommendations to weigh other indices more heavily (e.g., Hu & Bentler, 1998). Fossati et al., (2000), using a different method (weighted least squares) than Rotenstein and colleagues, reported different fit statistics (e.g., RMR and AGFI); but the fit of a unidimensional model in their sample, as in ours, was excellent and their factor co-efficients and internal consistency were higher than either this or the Rotenstein et al., sample. Notably, subjects in the Rotenstein sample were all outpatients and manifested somewhat lower overall severity and rates of PAPD, which may have resulted in poorer structural fit and internal consistency than in the current or Fossati samples, which comprised mixed inpatients and outpatients.

Findings on the structure of PAPD have important theoretical implications. As discussed above, one major controversy that led to the decision to move the diagnosis to the DSM-IV appendix involved content coverage. Concerns about the breadth of the construct led to a reconsideration of its content. In particular, Millon felt that the criteria reflected a narrow range of behaviors embedded in a larger, negativistic context. His view sufficiently influenced the Axis II work group that only three symptoms from DSM-III survive in the DSM-IV criteria (1, 3, and 4). Other symptoms, such as feeling underappreciated, resenting others, complaining of misfortune, and alternating between defiance and obedience were added. At the same time, several symptoms that relate to passive aggressive behavior as traditionally conceived were removed, including purposefully ineffective work, unjustified protests, avoiding obligations by forgetting, believing that the person does a better job than is objectively being done, resenting suggestions for improvement, and obstructing others' efforts.

This change in content likely affected the empirical structure of the construct, although if it was broadened in DSM-IV, one would expect the DSM-III criteria to be less ambiguously unidimensional. Syndromes need not be multidimensional in order to be considered valid, although they often are (e.g., borderline PD; Sanislow et al., 2002), and current findings may be regarded as consistent with the original objection of Millon and others regarding PAPD. However, whether it is regarded as a unidimensional trait or a multidimensional syndrome, evidence from the current study suggests that PAPD as represented in DSM-IV demonstrates reasonable validity and is relevant for clinical assessment. However, since the DSM-IV's definition did not fully encompass the defiant obstructive motivations behind passive behaviors, the present study's results are commensurably constrained by that conceptual and descriptive revision. The relative validity of varying representations of PAPD, including those of DSM-III and -IV, is an important question for continued research that cannot be answered in the CLPS data.

A related issue involves how to best operationalize a construct that is theoretically associated, perhaps more in DSM-III than DSM-IV, with psychological ambivalence. It might be argued that an assumption of internal consistency is questionable for such a construct. Further research on the effect of ambivalence on psychometric scales is an interesting area of ongoing research (e.g., Hopwood & Morey, 2007) that might be usefully applied to PAPD.

Fossati and colleagues (2000) concluded that PAPD represents a vulnerable subtype of narcissistic PD. It is important to note that this conclusion relied partially on analyses regarding the overlap of categorical diagnoses, despite many of their other analyses focusing on the dimensionality of PAPD. Furthermore, the best fitting model in their data specified PAPD and narcissistic personality disorder (NPD) as separable but related constructs. Consistent with

their conclusion, NPD had the strongest partial coefficient ( $\beta = .25$ ) in a regression analysis of PDs predicting PAPD in the current data. However, several other PDs also uniquely contributed to predicting PAPD, notably including borderline ( $\beta = .22$ ). Grilo and colleagues (2001) reported that the mean inter-item correlation in a subsample ( $n = 668$ ) of individuals used in the current report was .24, whereas the median correlation of PAPD items with those from other PDs ranged from .05 (dependent) to .17 (borderline and narcissistic). Similar results have been observed in a different sample (Becker et al., 1999). In summary, our results converge with multiple studies suggesting both discriminant validity of PAPD and strong relations between PAPD and borderline and narcissistic PDs. Further studies testing the hypothesis that PAPD is closely linked to vulnerable narcissism (Cain, Pincus, & Ansell, 2008), as suggested by Fossati and colleagues and perhaps particular subtypes or dimensions of borderline PD, may clarify its relation to these constructs.

Overall, our data suggest that PAPD as represented in the appendix of DSM-IV can be reliably assessed and demonstrates comparable stability to other PDs. Each of its criteria were endorsed at a non-trivial rate and meaningfully contributed to the construct. PAPD relates to a host of important clinical variables, such as functioning, substance use, and history of hospitalization. Consistent with previous research (Kasen et al., 2001), modest relations were also demonstrated with mood disorder. Furthermore, consistent with theories of PAPD and recent research, PAPD is associated with a unique pattern of personality traits, even after controlling for other PDs, that clinicians are likely to consider important. The constellation of traits and PDs related to PAPD suggests that it characterizes people with a high level of negative affect and ineffective and ambivalent interpersonal behavior likely to dissatisfy others. This is generally consistent with clinical descriptions by Benjamin (1993), Kernberg (1976), and Pretzer and Beck (1996). Relations with negative childhood experiences are also consistent with both previous research and these theoretical formulations.

In summary, the current data indicate PAPD is a useful clinical construct that deserves careful consideration in clinical assessment and may merit further consideration in discussions of PD nosology. Results suggest research on the construct should continue, particularly in regard to additional correlates of PAPD using both DSM-III and DSM-IV criteria sets, course, development and investigation of effective treatment strategies, and behavioral genetic and developmental studies that could provide further data regarding its etiology. Sampling issues may affect observed results. For example, PDs were non-randomly selected in the CLPS data, potentially reducing the power to find significant PAPD correlates and providing a conservative estimate of the magnitude of observed effects. Research unlike the current and previous studies in which PAPD is targeted by study design, rather than sampled in data that were initially gathered for other purposes, are likely to be particularly informative.

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**TABLE 1**

## PAPD Item Characteristics.

<b>Item</b>	<b>% Endorsement</b>	<b>Standardized Factor Coefficient</b>
Passive resistance	16.85	.40
Feels misunderstood and unappreciated	29.14	.61
Sullen and argumentative	28.18	.62
Scorns authority	22.44	.57
Resentment of more fortunate	22.57	.44
Complains of misfortune	26.54	.44
Alternates between defiance and contrition	13.41	.35

*Note.* Factor coefficients are from maximum likelihood estimated model. No coefficient differed from these values  $> .01$  using weighted least squares estimation.

TABLE 2

Relation of PAPD to Personality Disorders and Traits.

	<i>r</i>	PD/Trait $\beta$	PAPD Incremental $\beta$
DSM-IV		$R^2 = .46$	—
Paranoid	.42	.16	—
Schizotypal	.31		—
Schizoid	.18		—
Avoidant	.26		—
Dependent	.35	.10	—
Obsessive-Compulsive	.21	.09	—
Histrionic	.45	.11	—
Borderline	.50	.22	—
Narcissistic	.49	.25	—
Antisocial	.36	.11	—
FFM		$R^2 = .23$	
Neuroticism	.32	.28	.11
Extraversion			
Openness to Experience			
Agreeableness	-.38	-.34	-.17
Conscientiousness	-.22		-.21
SNAP		$R^2 = .28$	
Negative Temperament	.36	.15	.15
Manipulativeness	.36	.18	.13
Mistrust	.34	.14	
Eccentric Perceptions	.21		
Aggression	.43	.22	.21
Self-harm	.30		
Dependency	.19		
Positive Temperament			
Detachment			
Exhibitionism			
Entitlement	.12		
Disinhibition	.30		
Impulsivity	.23		
Propriety			
Workaholism			

Note.  $p < .01$  for all correlations given. Column three provides results from three regression analyses in which DSM-IV PDs and traits from the FFM and SNAP predicted PAPD symptoms. Column four represents the Beta weight of PAPD in hierarchical regression models in which PAPD significantly incremented all other PDs ( $F$  test for change in  $R^2$   $p < .01$ ).



TABLE 3

Relation of PAPD to Functioning, Psychiatric Disorders, Clinically Relevant Behaviors, and Childhood Experiences.

	$r^a$
Interpersonal Functioning	-.30
Work Functioning*	-.17
Recreational Functioning	-.22
Mood Disorder	
Anxiety Disorder	
Substance Use Disorder	.13
Presence of Prospective Suicidal Behavior over 4 years	
History of Psychiatric Hospitalization	.14
Childhood Neglect	.13
Childhood Abuse	.13
Witnessing Violence during Childhood	.13

Note.  $p < .01$  for all correlations given.

<sup>a</sup>Pearson correlations were used for continuous outcomes (i.e., functioning scores); point-biserial correlations were used for dichotomous outcomes. Distributions for dichotomous outcomes were: mood disorder ( $N_{\text{positive}} = 498$ ,  $N_{\text{negative}} = 163$ ), anxiety disorder ( $N_{\text{positive}} = 423$ ,  $N_{\text{negative}} = 310$ ), substance use disorder ( $N_{\text{positive}} = 168$ ,  $N_{\text{negative}} = 493$ ), prospective suicide gestures ( $N_{\text{positive}} = 113$ ,  $N_{\text{negative}} = 549$ ), history of hospitalization ( $N_{\text{positive}} = 301$ ,  $N_{\text{negative}} = 432$ ), neglect ( $N_{\text{positive}} = 451$ ,  $N_{\text{negative}} = 213$ ), abuse ( $N_{\text{positive}} = 450$ ,  $N_{\text{negative}} = 214$ ), witnessing violence ( $N_{\text{positive}} = 432$ ,  $N_{\text{negative}} = 232$ ). Missing data accounted for sample size differences for each outcome variable.

\* This correlation remained significant with the effects of other PDs controlled.