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A Metastructural Model of Mental Disorders and Pathological Personality Traits

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Abstract

Background—Psychiatric comorbidity is extensive in both psychiatric settings and the general population. Such comorbidity challenges whether DSM-based mental disorders serve to effectively carve nature at its joints. In response, a substantial literature has emerged showing that a small number of broad dimensions—internalizing, externalizing, and psychoticism—can account for much of the observed covariation among common mental disorders. However, the location of personality disorders within this emerging metastructure has only recently been studied, and no studies have yet examined where pathological personality traits fit within such a broad metastructural framework.

Methods—We conducted joint structural analyses of common mental disorders, personality disorders, and pathological personality traits in a sample of 628 current or recent psychiatric outpatients.

Results—Bridging across the psychopathology and personality trait literatures, the results provide evidence for a robust five-factor metastructure of psychopathology, including broad domains of symptoms and features related to internalizing, disinhibition, psychoticism, antagonism, and detachment.

Conclusions—These results reveal evidence for a psychopathology metastructure that (a) parsimoniously accounts for much of the observed covariation among common mental disorders, personality disorders, and related personality traits, and (b) provides an empirical basis for the organization and classification of mental disorder.

Psychiatric comorbidity is extensive in the general population (Kessler et al., 1994 Kessler et al., 2005), and in clinical samples poly-diagnosis is the rule rather than the exception (Zimmerman & Mattia, 1999). This complicates clinical communication, treatment selection, and frustrates efforts to uncover the pathophysiology, etiology, and maintenance

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mechanisms of mental illness (Hyman, 2010). One promising approach for resolving these issues involves using formal statistical modeling to clarify the natural structure of mental disorders (Krueger & Markon, 2006; Wright & Zimmermann, 2015). This approach has been profitably applied to both child (Achenbach, 1966; Lahey et al., 2008) and adult (Kotov et al., 2011; Krueger, 1999; Markon & Krueger, 2006) disorders. In adult psychopathology, a well-replicated structure has emerged based on the clustering of disorders and their symptoms into *internalizing* (e.g., unipolar mood disorders, anxiety disorders), *externalizing* (e.g., substance use, antisocial behavior), and *thought disorder/psychosis* (e.g., psychotic disorders, schizotypal personality disorder) spectra (Kotov et al., 2010; Markon, 2010; Wolf et al., 1988; Wright et al., 2013). This structure has demonstrated strong empirical and statistical evidence for its validity; importantly the resulting spectra or domains appear to predict treatment response and match genetic models of these disorders (Kendler et al., 2003; Kendler et al., 2011).

Recently developed quantitative models of psychopathology have expanded the basic *internalizing*, *externalizing*, and *thought disorder/psychosis* structure by incorporating additional diagnoses, most notably personality disorders (PDs), and have begun to uncover additional spectra. To date only four published studies have explored the structure of psychopathology using a broad suite of clinical syndromes and personality disorders (Blanco et al., 2013; Kotov et al., 2011; Markon, 2010; Røysamb et al., 2011).¹ Although each resultant model is necessarily unique given differences in the precise admixture of disorders (e.g., some do not include indicators of psychosis), sampling strategy (e.g., clinical vs. epidemiological), and other features (e.g., disorder-level vs. symptom-level analyses), two additional domains appear reasonably replicable across studies. First, Markon (2010) and Røysamb and colleagues (2011) each identified a new spectrum they respectively termed *pathological* or *anhedonic introversion*. In both cases, avoidant and dependent PDs were strong markers of the factor, although Røysamb et al. also found that schizoid and depressive PDs loaded strongly on the factor, which accounts for the slight difference in conceptualization. Blanco and colleagues (2013) also found evidence for a factor with the strongest loadings from avoidant and dependent PDs and social phobia.

Second, in three studies (Blanco et al., 2013; Kotov et al., 2011; Røysamb et al., 2011), a domain related to *antagonism*, as labeled by Kotov and colleagues, has emerged. Again, slight differences emerge in the makeup of this domain across studies, although narcissistic and histrionic PDs consistently exhibit the strongest loadings. Additional markers for this domain, but varying slightly across studies, include obsessive-compulsive, borderline, paranoid, and (to a lesser extent) antisocial PDs. What these disorders share to varying degrees is an antagonistic interpersonal style that puts afflicted individuals at odds with others. Notably, *introversion* and *antagonism*, which emerge with the addition of PDs, each deal with maladaptive social/interpersonal functioning, consistent with the view that the PDs reflect the interpersonal disorders (Benjamin, 1996; Hill, Pilkonis, Bear, 2011; Hopwood et al., 2013; Meyer & Pilkonis, 2005; Pincus, 2005). Therefore, based on this initial

¹We note that Kendler et al. (2011) also examined the joint genetic structure of clinical syndromes and personality disorders in what are very similar models. We do not discuss this as a separate study here given that Kendler et al. employ the same sample as, and arrive at similar conclusions to, Røysamb et al. (2011).

accumulation of studies that have included PDs in structural models of psychopathology and a strong theoretical rationale, the domains of *introversion* and *antagonism* appear to be good candidates to include alongside *internalizing*, *externalizing*, and *thought disorder/psychosis* as broad, replicable domains of psychopathology.

Taken together, these domains bear a remarkable conceptual resemblance to the pathological personality trait domains included in DSM-5 Section III system of PDs (American Psychiatric Association, 2013). The five domains outlined in this system include *Negative Affectivity*, *Antagonism*, *Detachment*, *Disinhibition*, and *Psychoticism*, and were empirically derived from quantitative modeling of more specific PD features (i.e., clinical specifiers) outlined by the DSM-5 Personality and PD workgroup (Krueger et al., 2011; 2012). Compared to this PD trait framework, the psychopathology spectra of *internalizing*, *antagonism*, *anhedonic/pathological introversion*, *externalizing*, and *thought disorder*, respectively, reflect strong conceptual matches. However, although intuitively compelling, these putative matches between psychopathology spectra and personality domains have not been empirically demonstrated.

Notably, an empirical demonstration that the major spectra underlying psychiatric comorbidity in common clinical syndromes and PDs align with the domains of pathological personality trait models would represent a major advance in clarifying the phenotypic structure of psychopathology. A model such as this would provide the foundation for a comprehensive bridge between mental disorders and elementary domains of individual differences in basic functioning. For example, the DSM-5 pathological trait model has been linked empirically to a large scientific literature on structural models of normal personality and temperament (De Fruyt et al., 2013; Gore & Widiger, 2013; Thomas et al., 2013; Watson et al., 2013; Wright et al., 2012; Wright & Simms, 2014), which builds on a larger literature linking pathological and basic personality traits (e.g., Markon et al., 2005). Adding to the strength of our proposal, that the structures underlying traits and much of psychopathology align, basic trait domains demonstrate strong associations to clinical syndromes (Kotov et al., 2010) and PDs (Samuel & Widiger, 2008; Saulsman & Page, 2004) in meta-analyses. Based on these accumulated findings, some have suggested that there is potential to organize both basic domains of individual differences and psychopathology using a finite number of functional domains or spectra rooted in basic psychological and physiological systems (e.g., Harkness, Reynolds, & Lilienfeld, 2013; Siever & Davis, 1991). This parallels efforts in the broader DSM-5 development process aimed at developing crosscutting dimensions of pathology (Narrow et al., 2012) and in the National Institute of Mental Health's Research Domain Criteria (RDoC; Insel et al., 2010; Sanislow et al., 2010). Further, an empirically based dimensional structure increases the potential to link with biological correlates, genetic liabilities, and leads to more replicable and accurate etiological research (Orfat & Krueger, 2012; Plomin et al., 2009).

The potential for an organizing metastructure that encompasses basic and pathological functioning would go a long way towards linking disparate scientific literatures and in so doing provide an organizing scheme for refining the study of psychopathology. In the current study, we tested whether such a model was viable by examining the joint structure of mental disorders and the DSM-5 pathological personality traits. We hypothesized that a

factor analysis of interview-diagnosed major clinical syndromes and PDs and patient-reported pathological trait scales in a large general psychiatric outpatient sample would result in five easily interpretable dimensions that closely resemble the aforementioned *internalizing*, *externalizing/disinhibition*, *thought disorder/psychoticism*, *antagonism*, and *introversion/detachment* domains. Specifically, we use exploratory structural equation modeling (ESEM; Asparouhov & Muthén, 2009; see also Marsh et al. 2010 for an applied example) to examine the joint structure of DSM-5 pathological personality traits, clinical syndromes, and personality disorders, while accounting for method variance across instruments. We hypothesize that disorders that mark the *internalizing* spectrum (e.g., mood, anxiety disorders) will load on the same factor as traits that indicate *Negative Affectivity* (e.g., Emotional Lability, Separation Insecurity), and that markers of *Externalizing* (e.g., alcohol use, antisocial PD) and *Disinhibition* (e.g., Risk Taking, Impulsivity), *antagonism* (e.g., narcissistic PD and histrionic PD) and trait *Antagonism* (e.g., callousness, manipulativeness), *pathological introversion* (e.g., avoidant PD, schizoid PD) and *Detachment* (e.g., Withdrawal, Restricted Affectivity), and *Thought Disorder* (e.g., psychotic symptoms, schizotypal PD) and *Psychoticism* (e.g., Unusual Beliefs, Perceptual Dysregulation) will load together on the same factors, respectively.

Methods

Sample and Procedure

Participants for the present study were recruited by distributing flyers at mental health clinics across Western New York and were eligible to participate if they reported psychiatric treatment within the past two years. Exclusionary criteria were age under 18 years and evidence that the data collected were untrustworthy.² The final sample included 628 participants with a *M* age of 43.2 years (*SD* = 12.5) and was majority female but were diverse in terms of racial, educational, and marital features (Table 1). The majority of the sample was currently in treatment (80%) or had been within the last one (10%) to two (5%) years.

Measures

Current criteria for clinical syndromes were assessed using the sixth edition of the Mini International Neuropsychiatric Inventory (MINI; Sheehan & LeCrubier, 2010; Sheehan et al. 1998), which was adapted (with permission) to (a) assess the DSM-5 criteria for the sampled disorders, and (b) relax certain skip-out rules so that all relevant symptoms were assessed (e.g., all symptoms of depression were assessed regardless of whether participants initially endorsed depressed mood or anhedonia). Criteria for the DSM-5 Section II PDs were assessed using a modified protocol for the Structured Clinical Interview for DSM-IV-TR Personality Disorders (SCID-II; First et al., 2002). Participants initially completed the SCID-II personality questionnaire, and interviewers followed up on all items for potential diagnoses to ascertain their presence and that they caused the individual dysfunction. Both

²Participants were excluded if (a) preliminary analyses indicated excessively inconsistent responding based on ad hoc inconsistency indices, (b) they had excessive missing responses on patient-report scales (i.e., more than 50%), or (c) they exhibited behaviors in session that suggested that their responses were not trustworthy (e.g., under the influence of substances). Sixty-seven participants were removed for data untrustworthiness.

assessments were conducted by highly trained interviewers (including the first author), who typically were clinical psychology doctoral students. Interviewers received extensive initial training and ongoing supervision by the second author, which included weekly case conferences and tape review throughout the course of the study. Independent reviewers recoded a total of 120 cases with excellent reliability. Disorder-level Kappa's were high ($Mdn = .96$; $range = .66-1.00$).

The MINI covers mood, anxiety, substance use, and psychotic disorders. All disorders assessed by the MINI were assessed dimensionally to allow for gradations in disorder severity, with the exception of psychotic delusions, hallucinations, and negative symptoms, which were treated as binary (i.e., absent or present), and panic attacks, which we treated as ordinal (i.e., absent, present, present with persistent fear of recurrence). The three psychotic disorders symptom sets (delusions, hallucinations, and negative symptoms) were combined to form an ordinal indicator of psychosis severity. Alcohol and drug abuse and dependence symptoms were collapsed to form single severity dimensions for each, consistent with DSM-5 formulations. Current manic episodes were excluded due to low rates of endorsement, which affected the reliability of estimated associations with other disorders and caused problems with model estimation. All SCID-II assessed PDs were treated as dimensional criterion counts.

The DSM-5 Section III pathological personality traits were assessed using the Personality Inventory for the DSM-5 (PID-5; Krueger et al., 2012). The PID-5 is a patient-report instrument that includes 220 questions measuring 25 PD traits, organized based on factor analytic evidence into five broad domains: Negative Affectivity, Detachment, Antagonism, Disinhibition, and Psychoticism. Each trait facet is measured by 4 to 14 questions. PID-5 items are rated on a four-point scale ranging from 0 (very false or often false) to 3 (very true or often true). Higher scale scores are indicative of greater personality pathology. Adequate to good internal consistencies were achieved in the current sample ($Mdn \alpha = .86$; $range = .77$ to $.96$).

Data Analysis

We used ESEM (Asparouhov & Muthén, 2009) to examine the joint structure of the clinician-assessed mental disorders and the patient-reported pathological personality traits. ESEM is a recently developed technique that permits models to include both exploratory (i.e., data-driven) and confirmatory (i.e., investigator-defined) factors. For the current study, ESEM offers the advantage of being able to estimate an exploratory model of the joint structure of mental disorders and pathological personality traits, while including “measurement factors” that account for the difference between assessment methods (i.e., clinician assigned symptoms vs. patient-reported traits). Therefore, we ran a final ESEM model with three factors for measurement, one each with the interview-based variables loading on it, and one with all of the PID-5 scales loading on it. Additionally, the ESEM models included an exploratory portion of the structure that allowed all variables to freely load on each estimated factor to allow the data to determine the optimal pattern of loadings. We estimated models with zero through seven exploratory factors. Measurement factors were estimated as orthogonal to each other and the exploratory factors. For the exploratory

portion of the model we used an oblique Geomin rotation due to the expectation that factors would be correlated, and Geomin's balance of factor and variable complexity in its rotational criterion (Sass & Schmitt, 2010).³

All models were estimated in Mplus 7.11 (Muthén & Muthén, 2012). Due to the ordinal nature of two variables (all other variables were measured continuously), we used a robust maximum likelihood estimator (MLR in Mplus), which provides fit statistics and standard error estimates adjusted for non-normality in the data. Additionally, although missing data in the interviews was negligible, not all participants completed the PID5 (~74%) because it was presented later in the assessment protocol. It was found that the missingness on the PID5 was associated with severity of interview assessed psychopathology, and therefore it was treated as missing at random, and handled via full-information maximum likelihood in our models.

Adjudication between ESEM models was based on multiple fit indices in addition to interpretability. Because a non-significant chi-square statistic is rarely obtained in real-world clinical data (Brown, 2006), we relied on the root mean square error of approximation (RMSEA) and the associated 90% confidence interval, with values lower than .05 indicating excellent fit and values lower than .08 indicating good fit; the comparative fit index (CFI), with values approaching or greater than .95 indicative of excellent fit, and values of .90 or greater indicative of acceptable fit, and the SRMR, with values lower than .05 indicative of excellent fit (Hu & Bentler, 1999).

Results

Exploratory Structural Equation Model of Mental Disorders and Pathological Personality Traits

Model fit for ESEMs ranging from zero to seven substantive factors (i.e., not counting method factors) are listed in Table 2. Fit improved appreciably through seven factors, although it was acceptable according to two indices (RMSEA, SRMR) starting with a three-factor model. We therefore gave close scrutiny to results from the three- through seven-factor models. Full results for each model can be found in the supplementary tables. For the three-factor model we interpreted the factors as reflecting *internalizing* (strong loadings from, e.g., PID5 Anxiousness, Borderline PD, Major Depression), *externalizing* (strong loadings from, e.g., PID5 Risk-Taking, Narcissistic PD, and loadings from Alcohol and Drug Use), and *detachment* (strongest loadings from PID5 Withdrawal, PID5 Restricted Affectivity, and Schizoid PD). In the four-factor model, *internalizing*, *externalizing*, and *detachment* factors remain, but now a clear *psychoticism* factor emerges. The five-factor conformed to the hypothesized structure, in that there were clear factors that could be labeled *internalizing* (e.g., strong loadings from PID5 Anxiousness, generalized anxiety, major depression, Borderline PD), *disinhibition/externalizing* (drug use, alcohol use, antisocial PD, PID5 Risk-Taking), *psychoticism* (psychotic symptoms, PID5 Unusual Beliefs), *antagonism* (narcissistic, histrionic, paranoid PD symptoms, PID5

³We also ran all models with an alternative oblimin rotation to determine whether results were robust to rotational criteria. Results suggested that the two rotations provided highly similar solutions that would result in the same conclusions.

Manipulativeness), and *detachment* (schizoid PD symptoms, negative histrionic PD symptoms, PID5 Withdrawal, PID5 Restricted Affect).

In the six-factor model the solution retained its structure with the exception that a small *histrionism* factor (marked mostly by Histrionic PD and PID5 Attention Seeking) split off from the Antagonism spectrum. Similarly, in the seven-factor model, the primary structure was retained, excepting that a small factor we labeled *suspiciousness* emerged, with only PID5 Suspiciousness having its primary loading on the factor. Thus, although the best model fit was obtained for a seven-factor solution, factors that emerged after the five-factor solution were either highly specific or suggestive of over-factoring. Thus, based on theoretical and quantitative grounds, we chose to retain the five-factor model consistent with hypotheses.⁴

Discussion

Based on an emerging body of research suggesting that five replicable domains of psychopathology account for the structure of common clinical syndromes, psychosis, and PDs, and that these domains bear close conceptual resemblance to the major domains of personality traits, we estimated a joint structural model to test whether the same dimensions could account for patterns of covariation across these traditionally disparate systems. Our results demonstrate that an underlying metastructure explains the shared features of personality and psychopathology and may help uncover the basic structure for much of human psychological maladaptation. Many other clinical theorists and researchers have hypothesized this relationship, going back as far as antiquity, with Hippocrates and Galen, and continuing through to more contemporary thought as well (e.g., Eysenck, 1967; Siever & Davis, 1991; Harkness et al., 2013; Clark, 2005). Yet this is the first study to demonstrate this fact using a reasonably comprehensive grouping of psychiatric disorders and suite of personality traits. Ultimately, these results move us towards greater theoretical integration across psychiatric and behavioral sciences, and have important implications for refining the classification of mental disorders and refocusing the targets of mechanistic research.

Our findings suggest that the combination of mental disorders and pathological personality traits can be combined within the same structural framework. Moreover, the alignment of the disorder spectra with the trait domains closely follows our predictions based on the disorder specific impairments and the trait scale content. As noted in the introduction, three of the spectra found here, *internalizing*, *externalizing/disinhibition*, and *thought disorder/psychosis* have been well replicated in a number of samples (Kotov et al., 2010, 2011; Markon, 2010; Wright et al., 2013). As it pertains to these dimensions, our results accord well with prior findings, such that in our model the pattern of PID-5 scale loadings on these three domains were mostly as expected. Traits tapping Negative Affectivity loaded on the same factor as disorders that mark *internalizing*, Disinhibition scales loaded on the same factor as disorders considered part of the *externalizing* spectrum, and the PID5 Psychoticism scales loaded on the same factor as psychotic symptoms and schizotypal PD criteria.

⁴Given the demographic diversity in the sample, we re-estimated the final five-factor model while simultaneously regressing all indicators on gender, age, and race to ensure that the structure did not substantively change. Results of this model were highly consistent with the presented model, with some coefficients changing only in the third and second decimal points.

A comprehensive inclusion of all of the PDs sets this structural model apart from the majority of prior work. Although some PDs loaded most strongly on the *internalizing*, *externalizing/disinhibition*, and *thought disorder* spectra, expanding the model to include these disorders also requires an expansion to include the primarily interpersonal domains of *antagonism* and *detachment*. These spectra are an important addition to the structure of psychopathology, in so far as they reflect maladaptive variants of core domains of human functioning (Bakan, 1966; Wiggins, 1991). The emergence of these additional domains further emphasizes that personality pathology is intimately linked with interpersonal dysfunction, which is a view reflected in the alternative DSM-5 Section III PD model and many theorists. However, PDs are not exclusively related to the primarily interpersonal factors of *antagonism* and *detachment*, but rather are infused throughout the structure of mental disorders. For example, borderline has its strongest loading on the Internalizing factor, reflecting the affective dysregulation associated with the construct, and schizotypal falls along the *psychoticism* spectrum. What is likely the case is that much of the characteristic interpersonal dysfunction that is the shared hallmark of the PDs (Benjamin, 2005; Hopwood et al., 2013; Meyer & Pilkonis, 2005; Pincus, 2005) exists outside of this structural hierarchy, and rather is reflected in social-cognitive processes related to self and other perception.

Nevertheless, the domains of *antagonism* and *detachment* reflect important new additions to the quantitatively derived structure of psychopathology. In our model the *antagonism* factor, defined most strongly by narcissistic and histrionic features and antagonistic traits, is consonant with prior results (Kotov et al., 2011). In contrast, the domain of *detachment* observed here related more focally to low positive emotionality and withdrawal as opposed to social avoidance and interpersonal submissiveness. Thus, our results align more closely with Roysamb and colleagues (2011) as opposed to Markon (2011). In the final model, social avoidance (e.g., avoidant PD, social phobia) emerged more strongly as a fear domain within the internalizing spectrum. Taken together, these results suggest the need for refinement of content related to impoverished social relating in psychopathology. Specifically, there likely are distinct underlying processes driving failures to socially engage (i.e., fear vs. lack of social reward).

Despite the pattern of loadings that were generally highly consistent with expectations, several deviations and cross-loadings are notable. For instance, it was not uncommon for the PID-5 scales from *disinhibition* and *psychoticism* to have a “split” loading between their predicted location and the internalizing domain. As it pertains to the PID5 Psychoticism scales, this may reflect distress captured in responses to patient-report scales of this nature. Prior work has shown high correlation between *internalizing* and *thought disorder* spectra (e.g., Kotov et al., 2010, 2011; Wright et al., 2013). For the PID5 scales of Irresponsibility and Impulsivity, it may be that cross-loadings arise because *internalizing* impairs task accomplishment, and past research has demonstrated that impulsivity can be driven by negative affect (Whiteside & Lynam, 2001), respectively. Therefore, these cross-loadings are generally understandable based on past findings. Further theoretically consistent cross-loadings include the negative loadings of obsessive-compulsive PD and PID5 Perfectionism on our *disinhibition* factor. However, obsessive-compulsive disorder and obsessive-

compulsive PD generally had modest loadings, suggesting an area in need of continued inquiry.

This points to a general need for a detailed refining of these domains. Deriving domains from current psychiatric constructs is an exercise in rough estimation at best. It is akin to using a hacksaw to carve nature at her joints, when what are needed are refined tools that serve like scalpels. This is the view espoused in the RDoC effort, where the goal is to refine the measurement of core domains, which can then be used as framework to bootstrap a new nosology that would rest on a firm scientific foundation (Insel et al., 2010). The results here would suggest that this approach may be viable, and the patterns of covariation among mental disorders along with their integration with basic domains of functioning could serve to expedite this process. Indeed, one the major limitations of the current psychiatric nosology is that it was created without consideration for normative functioning, and as a result, the extant structure of disorders remains divorced from the basic mental, behavioral, and physiological processes that necessarily give rise to mental disorder when they go awry. The initial description of putatively discrete syndromes based on clinical observation was an essential initial step in outlining important clinical constructs. However, the patterns of observed covariation among disorders, shared treatment responses, and widespread failure to find specific biomarkers, suggests the current parsing of disorders lacks validity and may have run their course in terms of scientific yield (Hyman, 2010).

Moving forward, what is needed is a revised research agenda based on refining the definition and measurement of a finite set of general domains rooted in biopsychosocial processes and mechanisms. In turn, this would lay the foundation for studies that selected individuals along these spectra for intensive study in order to maximize precision of measurement and statistical power, as opposed to case control designs with noted limitations in interpretability and potential for linking to biology (Hyman, 2010; Plomin et al., 2009). Furthermore, this likely would result in a model of psychopathology that more closely approximates the gradations observed in clinical practice, allowing for fine-grained assessment of individual differences in functioning, ranging from the healthy to the pathological (Harkness et al., 2013). The linking of personality trait domains and disorder spectra provides an important demonstration of the viability of this proposal, serving as a much-needed bridge between basic processes and maladaptivity.

Any study of this type is necessarily limited by the nature of the data on which the model was estimated. A strength of this study was the large clinical sample with rich levels of psychopathology of various types, assessed by structured clinical interviews. However, not all expressions of psychopathology were assessed or included. Notable exclusions included mania, somatic disorders, eating disorders, the autism spectrum, and tic disorders. Also, because the DSM-5 traits were assessed via self-report only, an open question remains regarding whether an identical structure would emerge if clinician ratings were included. Emerging results suggest that structural analyses of these traits as rated by clinicians result in a very similar structure, providing confidence in the results (Morey et al., 2013). Patient reports of traits hypothesized to indicate the *disinhibition* and *psychoticism* domains might be influenced in large part by levels of distress as opposed to purely problems in cognition, and clinician ratings might be able to more cleanly assess these domains.

Several other considerations arise from our study. First, the structural model we arrived at here has emerged from exploratory analyses, and therefore the results should be considered an initial demonstration of a viable “metastructure” that necessitates replication and confirmation in other samples of a diverse nature. We hope that other investigators will be motivated by our findings to pursue refined models in a confirmatory framework. Second, we note that our sample size, although large, is modest when considering model complexity, which further indicates the need for replication in larger samples. Finally, some may wonder to what degree the model we have estimated here truly integrates traditionally diverse domains (i.e., psychopathology and traits) as opposed to merely demonstrating that the trait scales used here share the same item content as the criteria for mental disorders. Although the DSM-5 traits were designed to capture the important features of PD, and it is clearly the case that some trait scales (e.g., PID5 Depressivity, Anxiousness) overlap with clinical syndromes, others do not have such explicit representation (e.g., PID5 Hostility, Submissiveness). In many respects, the observed item similarities across domains is consistent with our view that these domains share items because they are not wholly distinct domains. Strictly differentiating personality from psychopathology likely is an overstatement of true differences between them given that all of these features are phenotypes with roots in what are necessarily the same biological and social substrates.

In conclusion, the results of the models estimated here suggest that large portions of the recognized mental disorders can be organized within a framework shared by personality trait domains. These spectra that cut across personality and psychopathology provide fundamental orienting dimensions for organizing classification and guiding research in the service of identifying core mechanisms. Although further refinement of the precise structure of these dimensions is warranted, the outlines of the picture appear clear. A comprehensive framework of individual differences in normative and maladaptive functioning provides much needed integration of psychiatric nosology with the basic sciences that should be its foundation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Sample demographic features.

	n (%)
Sex	
Female	399 (63.5)
Male	228 (36.3)
Race	
White	391 (62.3)
Black	215 (34.2)
Other	22 (3.5)
Education Level	
< High School	101 (16.1)
High School	345 (54.9)
College	124 (19.7)
> College	58 (9.2)
Marital Status	
Married	114 (18.2)
Widowed	28 (4.5)
Separated	45 (.07)
Divorced	123 (19.6)
Never Married	316 (5.3)

Note. N = 628. 1 participant did not provide their sex and two did not provide their marital status.

Table 2

Model details and fit indices for exploratory factor analyses of mental disorder symptom counts and Personality Inventory for DSM-5 scales.

Models	<i>k</i>	<i>df</i>	χ^2	χ^2/p	RMSEA	RMSEA 90% CI	CFI	SRMR
Method Factors	135	945	6808.86	<.001	.099	.097-.102	.570	.244
1-Factor	180	900	4735.07	<.001	.082	.082-.085	.719	.092
2-Factor	224	856	3746.58	<.001	.073	.071-.076	.788	.065
3-Factor	267	813	3205.68	<.001	.068	.066-.071	.824	.047
4-Factor	309	771	2642.96	<.001	.062	.060-.065	.863	.042
5-Factor	350	730	2205.03	<.001	.057	.054-.059	.892	.036
6-Factor	390	690	1930.28	<.001	.054	.051-.056	.909	.032
7-Factor	429	651	1691.19	<.001	.050	.048-.053	.924	.031

Note. *N*=628. *k* = number of estimated parameters; *df* = model degrees of freedom; RMSEA = Root Mean Square Error of Approximation; CI = Confidence Interval; CFI = Comparative Fit Index; SRMR = Standardized Root Mean Square Residual. Model estimated in using robust maximum likelihood.

Exploratory structural equation model of mental disorder symptom counts and pathological personality traits..

Table 3

<i>Factor Loadings</i>	Internalizing		Disinhibition		Antagonism		Detachment		Psychoticism		Methods	
	λ	(SE)	λ	(SE)	λ	(SE)	λ	(SE)	λ	(SE)	λ	(SE)
PID5 Anxiousness	.92	(.03)	-.13	(.06)	.06	(.07)	-.11	(.06)	-.13	(.05)	.15	(.11)
PID5 Depressiveness	.85	(.07)	.12	(.06)	-.24	(.11)	.01	(.05)	-.04	(.11)	.36	(.07)
PID5 Anhedonia	.85	(.06)	.04	(.06)	-.23	(.12)	.13	(.06)	-.17	(.13)	.32	(.08)
PID5 Emotional Lability	.83	(.04)	.00	(.06)	.05	(.05)	-.24	(.06)	.03	(.09)	.09	(.09)
Borderline PD	.68	(.06)	.16	(.05)	.15	(.05)	.00	(.04)	.05	(.07)	.32	(.06)
PID5 Separation Insecurity	.68	(.04)	-.02	(.06)	.14	(.11)	-.28	(.05)	-.09	(.06)	.19	(.08)
Avoidant PD	.68	(.04)	-.10	(.10)	-.16	(.08)	.20	(.06)	.01	(.06)	.21	(.08)
PID5 Perseveration	.68	(.07)	-.16	(.07)	-.01	(.08)	-.20	(.06)	.19	(.10)	.38	(.09)
PID5 Distractibility	.68	(.09)	.01	(.05)	-.20	(.09)	-.19	(.06)	.19	(.10)	.36	(.07)
Major Depression	.65	(.04)	.18	(.07)	-.05	(.06)	.06	(.04)	.01	(.05)	.38	(.06)
Generalized Anxiety	.62	(.06)	.11	(.07)	.08	(.06)	.00	(.04)	-.09	(.09)	.42	(.07)
Post-Traumatic Stress	.59	(.05)	.17	(.05)	.01	(.04)	.09	(.04)	.15	(.05)	.43	(.06)
Dysthymia	.58	(.05)	.18	(.06)	-.02	(.05)	.12	(.04)	.00	(.05)	.34	(.07)
Dependent PD	.58	(.05)	.07	(.06)	-.12	(.07)	-.19	(.05)	.09	(.06)	.21	(.06)
Paranoid PD	.56	(.11)	.03	(.04)	.38	(.07)	.24	(.06)	-.06	(.06)	.31	(.06)
PID5 Hostility	.55	(.08)	.02	(.07)	.46	(.08)	.10	(.07)	-.07	(.07)	.22	(.10)
PID5 Suspiciousness	.53	(.10)	-.07	(.06)	.44	(.07)	.24	(.07)	.01	(.04)	.08	(.11)
Social Phobia	.51	(.05)	-.03	(.09)	-.07	(.08)	.06	(.06)	.17	(.07)	.19	(.06)
PID5 Submissiveness	.45	(.11)	-.13	(.07)	-.26	(.12)	-.36	(.06)	.03	(.08)	.33	(.06)
PID5 Perfectionism	.44	(.08)	-.38	(.08)	.35	(.08)	.00	(.05)	.08	(.08)	.17	(.13)
PID5 Irresponsibility	.42	(.06)	.32	(.08)	.05	(.13)	-.03	(.05)	.12	(.11)	.44	(.06)
Schizotypal PD	.39	(.06)	-.05	(.06)	.04	(.09)	.08	(.05)	.36	(.07)	.41	(.05)
PID5 Impulsivity	.39	(.06)	.30	(.09)	.11	(.11)	-.16	(.05)	.26	(.08)	.25	(.08)
Panic Attacks	.35	(.06)	.07	(.09)	-.05	(.09)	.04	(.05)	.15	(.09)	.17	(.07)
Obsessive-Compulsive PD	.32	(.06)	-.27	(.07)	.22	(.08)	-.01	(.04)	.06	(.07)	.30	(.07)
Obsessive-Compulsive	.27	(.06)	.03	(.07)	.12	(.06)	.03	(.05)	.20	(.07)	.24	(.06)
Drug Use	.04	(.06)	.60	(.11)	.04	(.10)	.04	(.06)	.01	(.09)	.00	(.00)

Factor Loadings	Internalizing		Disinhibition		Antagonism		Detachment		Psychoticism		Methods	
	λ	(SE)	λ	(SE)	λ	(SE)	λ	(SE)	λ	(SE)	λ	(SE)
Alcohol Use	.02	(.05)	.41	(.11)	.04	(.08)	.03	(.05)	.14	(.09)	.00	(.00)
Antisocial PD	.06	(.06)	.39	(.09)	.20	(.09)	.10	(.05)	.23	(.08)	.02	(.06)
PID5 Risk-Taking	-.16	(.06)	.37	(.11)	.21	(.12)	-.05	(.06)	.26	(.09)	.22	(.08)
PID5 Manipulativeness	-.05	(.04)	.18	(.15)	.59	(.15)	-.12	(.07)	-.01	(.06)	.51	(.09)
PID5 Grandiosity	-.09	(.06)	-.14	(.14)	.57	(.09)	.02	(.05)	.27	(.10)	.37	(.08)
PID5 Attention Seeking	.02	(.04)	.06	(.16)	.53	(.18)	-.44	(.06)	.03	(.05)	.32	(.32)
Narcissistic PD	.29	(.08)	.01	(.07)	.51	(.07)	-.01	(.05)	.06	(.06)	.48	(.05)
PID5 Deceitfulness	.09	(.05)	.24	(.14)	.47	(.16)	-.06	(.06)	.01	(.10)	.60	(.08)
PID5 Callousness	.10	(.07)	.14	(.11)	.39	(.10)	.29	(.05)	.18	(.14)	.44	(.07)
Histrionic PD	.07	(.04)	.12	(.12)	.32	(.14)	-.45	(.06)	.07	(.09)	.32	(.07)
PID5 Withdrawal	.50	(.07)	-.19	(.06)	.04	(.05)	.52	(.06)	.10	(.16)	.28	(.07)
Schizoid PD	.28	(.06)	.05	(.05)	.01	(.05)	.50	(.05)	.08	(.12)	.22	(.07)
PID5 Restricted Affectivity	.00	(.06)	.05	(.11)	-.01	(.09)	.49	(.06)	.27	(.25)	.50	(.09)
PID5 Intimacy Avoidance	.15	(.07)	-.03	(.07)	-.06	(.07)	.38	(.06)	.16	(.18)	.35	(.07)
PID5 Unusual Beliefs	.03	(.04)	.04	(.06)	.05	(.08)	.05	(.05)	.79	(.04)	.21	(.14)
PID5 Perceptual Dysregulation	.34	(.05)	.10	(.07)	.01	(.04)	-.01	(.03)	.61	(.06)	.31	(.12)
Psychotic Symptoms	.08	(.06)	-.07	(.07)	-.02	(.10)	.14	(.06)	.51	(.07)	.06	(.08)
PID5 Eccentricity	.43	(.06)	-.02	(.05)	.04	(.05)	-.09	(.05)	.46	(.06)	.27	(.09)
Factor Correlations	Internalizing		Disinhibition		Antagonism		Detachment		Psychoticism			
Internalizing	1.00											
Disinhibition	.19		1.00									
Antagonism	.28		.31		1.00							
Detachment	.15		-.01		-.02		1.00					
Psychoticism	.46		.16		.40		.06		1.00			

Note. A Geomin oblique factor rotation was used. λ = factor loading; SE = standard error. Factor loadings > |.30| bolded. Methods factor loadings, although based on three factors, are presented in the same column for space considerations. PD = Personality disorder; PID5 = Personality Inventory for the DSM-5.