Psychological Medicine (2015), **45**, 2309–2319. © Cambridge University Press 2015
doi:10.1017/S0033291715000252

ORIGINAL ARTICLE

A metastructural model of mental disorders and pathological personality traits

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Background. Psychiatric co-morbidity is extensive in both psychiatric settings and the general population. Such co-morbidity 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.

Method. We conducted joint structural analyses of common mental disorders, personality disorders and pathological personality traits in a sample of 628 current or recent psychiatric out-patients.

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.

Received 13 May 2014; Revised 17 January 2015; Accepted 28 January 2015; First published online 23 April 2015

Key words: Co-morbidity, exploratory structure equation modeling, personality disorders, personality traits, structure of mental disorders.

Introduction

Psychiatric co-morbidity is extensive in the general population (Kessler *et al.* 1994, 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 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 (Krueger, 1999; Markon & Krueger, 2006; Kotov *et al.* 2011) 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 (Wolf *et al.* 1988; Kotov *et al.* 2010*a*, *b*; Markon, 2010; 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, 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 PDs (Markon, 2010; Kotov *et al.* 2011; Røysamb *et al.* 2011; Blanco *et al.* 2013)[1]†. Although each resultant model is necessarily unique given differences in the precise admixture

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of disorders (e.g. some do not include indicators of psychosis), sampling strategy (e.g. clinical *versus* epidemiological), and other features (e.g. disorder-level *versus* symptom-level analyses), two additional domains appear reasonably replicable across studies. First, Markon (2010) and Røysamb *et al*. (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.* (2011) also found that schizoid and depressive PDs loaded strongly on the factor, which accounts for the slight difference in conceptualization. Blanco *et al*. (2013) also found evidence for a factor with the strongest loadings from avoidant and dependent PDs and social phobia.

Second, in three studies (Kotov *et al.* 2011; Røysamb *et al.* 2011; Blanco *et al.* 2013), 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; Meyer & Pilkonis, 2005; Pincus, 2005; Hill *et al*. 2010; Hopwood *et al.* 2013). Therefore, based on this initial 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 the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (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 with 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 co-morbidity 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 (Wright *et al.* 2012; De Fruyt *et al.* 2013; Gore & Widiger, 2013; Thomas *et al.* 2013; Watson *et al.* 2013; 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 with clinical syndromes (Kotov *et al.* 2010*a*, *b*) and PDs (Saulsman & Page, 2004; Samuel & Widiger, 2008) 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. Siever & Davis, 1991; Harkness *et al*. 2014). This parallels efforts in the broader DSM-5 development process aimed at developing crosscutting dimensions of pathology (Narrow *et al*. 2013) 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 and genetic liabilities, and leads to more replicable and accurate etiological research (Plomin *et al.* 2009; Ofrat & Krueger, 2012).

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 out-patient sample would

https://doi.org/10.1017/S0033291715000252 Published online by Cambridge University Press

result in five easily interpretable dimensions that closely resemble the aforementioned internalizing, externalizing/disinhibition, thought disorder, antagonism and introversion/detachment domains. Specifically, we use exploratory structural equation modeling (ESEM; Asparouhov & Muthén, 2009; for an applied example, see also Marsh *et al.* 2010) to examine the joint structure of DSM-5 pathological personality traits, clinical syndromes and PDs, 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.

Table 1. *Sample demographic features* (n = 628)[a]

	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 (0.07)
Divorced	123 (19.6)
Never married	316 (5.3)

[a] One participant did not provide their sex and two did not provide their marital status.

Method

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 2 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 mean age of 43.2 years (S.D. = 12.5) and was mainly female but was 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 1 (10%) to 2 (5%) years.

Measures

Current criteria for clinical syndromes were assessed using the sixth edition of the Mini International Neuropsychiatric Inventory (MINI; Sheehan *et al.* 1998; Sheehan & LeCrubier, 2010), 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 assessments were conducted by highly trained interviewers (including the first author; A.G.C.W.), who typically were clinical psychology doctoral students. Interviewers received extensive initial training and ongoing supervision by the second author (L.J.S.), 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 κ's were high (median = 0.96; range = 0.66–1.00).

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 were treated as ordinal (i.e. absent, present, present with persistent fear of recurrence). The three psychotic disorder 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

https://doi.org/10.1017/S0033291715000252 Published online by Cambridge University Press

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 four 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 (median $\alpha = 0.86$; range = 0.77 to 0.96).

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 *versus* 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 to 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 were negligible, not all participants completed the PID-5 (about 74%) because it was presented later in the assessment protocol. It was found that the missingness on the PID-5 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 χ^2 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 0.05 indicating excellent fit and values lower than 0.08 indicating good fit, the comparative fit index, with values approaching or greater than 0.95 indicative of excellent fit, and values of 0.90 or greater indicative of acceptable fit, and the standardized root mean square residual (SRMR), with values lower than 0.05 indicative of excellent fit (Hu & Bentler, 1999).

Ethical Standards

All procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

Results

Exploratory structural equation model of mental disorders and pathological personality traits

Model fit results for ESEMs ranging from zero to seven substantive factors (i.e. not counting method factors) are listed in Table 2. Fit improved appreciably up to 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- to seven-factor models. Full results for each model can be found in the online Supplementary Tables. For the three-factor model we interpreted the factors as reflecting internalizing (strong loadings from, e.g. PID-5 anxiousness, borderline PD, major depression), externalizing (strong loadings from, e.g. PID-5 risk-taking, narcissistic PD, and loadings from alcohol and drug use) and detachment (strongest loadings from PID-5 withdrawal, PID-5 restricted affectivity and schizoid PD). In the four-factor model, internalizing, disinhibition and detachment factors remained, but now a clear psychoticism factor

https://doi.org/10.1017/S0033291715000252 Published online by Cambridge University Press

Table 2. *Model details and fit indices for exploratory factor analyses of mental disorder symptom counts and Personality Inventory for DSM-5 scales* (n = *628*)[a]

Models	*k*	df	χ^2	χ^2 *p*	RMSEA	RMSEA 90% CI	CFI	SRMR
Method factors	135	945	6808.86	<0.001	0.099	0.097–0.102	0.570	0.244
1-Factor	180	900	4735.07	<0.001	0.082	0.082–0.085	0.719	0.092
2-Factor	224	856	3746.58	<0.001	0.073	0.071–0.076	0.788	0.065
3-Factor	267	813	3205.68	<0.001	0.068	0.066–0.071	0.824	0.047
4-Factor	309	771	2642.96	<0.001	0.062	0.060–0.065	0.863	0.042
5-Factor	350	730	2205.03	<0.001	0.057	0.054–0.059	0.892	0.036
6-Factor	390	690	1930.28	<0.001	0.054	0.051–0.056	0.909	0.032
7-Factor	429	651	1691.19	<0.001	0.050	0.048–0.053	0.924	0.031

DSM-5, Diagnostic and Statistical Manual of Mental Disorders, fifth edition; *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.

[a] Model estimated using robust maximum likelihood.

emerged. The five-factor conformed to the hypothesized structure, in that there were clear factors that could be labeled internalizing (e.g. strong loadings from PID-5 anxiousness, generalized anxiety, major depression, borderline PD), disinhibition (drug use, alcohol use, antisocial PD, PID-5 risk-taking), psychoticism (psychotic symptoms, PID-5 unusual beliefs), antagonism (narcissistic, histrionic, paranoid PD symptoms, PID-5 manipulativeness) and detachment (schizoid PD symptoms, negative histrionic PD symptoms, PID-5 withdrawal, PID-5 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 PID-5 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 PID-5 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. As such, based on theoretical and quantitative grounds, we chose to retain the five-factor model consistent with hypotheses (see Table 3)[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; Clark, 2005; Harkness *et al.* 2014). 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 and thought disorder/psychosis, have been well replicated in a number of samples (Kotov *et al.* 2010*a*, *b*, 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

https://doi.org/10.1017/S0033291715000252 Published online by Cambridge University Press

Table 3. *Exploratory structural equation model of mental disorder symptom counts and pathological personality traits*[a]

	Internalizing		Disinhibition		Antagonism		Detachment		Psychoticism		Methods	
Factor loadings	λ	(S.E.)	λ	(S.E.)	λ	(S.E.)	λ	(S.E.)	λ	(S.E.)	λ	(S.E.)
PID-5 anxiousness	0.92[b]	(0.03)	−0.13	(0.06)	0.06	(0.07)	−0.11	(0.06)	−0.13	(0.05)	0.15	(0.11)
PID-5 depressiveness	0.85[b]	(0.07)	0.12	(0.06)	−0.24	(0.11)	0.01	(0.05)	−0.04	(0.11)	0.36	(0.07)
PID-5 anhedonia	0.85[b]	(0.06)	0.04	(0.06)	−0.23	(0.12)	0.13	(0.06)	−0.17	(0.13)	0.32	(0.08)
PID-5 emotional lability	0.83[b]	(0.04)	0.00	(0.06)	0.05	(0.05)	−0.24	(0.06)	0.03	(0.09)	0.09	(0.09)
Borderline PD	0.68[b]	(0.06)	0.16	(0.05)	0.15	(0.05)	0.00	(0.04)	0.05	(0.07)	0.32	(0.06)
PID-5 separation insecurity	0.68[b]	(0.04)	−0.02	(0.06)	0.14	(0.11)	−0.28	(0.05)	−0.09	(0.06)	0.19	(0.08)
Avoidant PD	0.68[b]	(0.04)	−0.10	(0.10)	−0.16	(0.08)	0.20	(0.06)	0.01	(0.06)	0.21	(0.08)
PID-5 perseveration	0.68[b]	(0.07)	−0.16	(0.07)	−0.01	(0.08)	−0.20	(0.06)	0.19	(0.10)	0.38	(0.09)
PID-5 distractibility	0.68[b]	(0.09)	0.01	(0.05)	−0.20	(0.09)	−0.19	(0.06)	0.19	(0.10)	0.36	(0.07)
Major depression	0.65[b]	(0.04)	0.18	(0.07)	−0.05	(0.06)	0.06	(0.04)	0.01	(0.05)	0.38	(0.06)
Generalized anxiety	0.62[b]	(0.06)	0.11	(0.07)	0.08	(0.06)	0.00	(0.04)	−0.09	(0.09)	0.42	(0.07)
Post-traumatic stress	0.59[b]	(0.05)	0.17	(0.05)	0.01	(0.04)	0.09	(0.04)	0.15	(0.05)	0.43	(0.06)
Dysthymia	0.58[b]	(0.05)	0.18	(0.06)	−0.02	(0.05)	0.12	(0.04)	0.00	(0.05)	0.34	(0.07)
Dependent PD	0.58[b]	(0.05)	0.07	(0.06)	−0.12	(0.07)	−0.19	(0.05)	0.09	(0.06)	0.21	(0.06)
Paranoid PD	0.56[b]	(0.11)	0.03	(0.04)	0.38[b]	(0.07)	0.24	(0.06)	−0.06	(0.06)	0.31	(0.06)
PID-5 hostility	0.55[b]	(0.08)	0.02	(0.07)	0.46[b]	(0.08)	0.10	(0.07)	−0.07	(0.07)	0.22	(0.10)
PID-5 suspiciousness	0.53[b]	(0.10)	−0.07	(0.06)	0.44[b]	(0.07)	0.24	(0.07)	0.01	(0.04)	0.08	(0.11)
Social phobia	0.51[b]	(0.05)	−0.03	(0.09)	−0.07	(0.08)	0.06	(0.06)	0.17	(0.07)	0.19	(0.06)
PID-5 submissiveness	0.45[b]	(0.11)	−0.13	(0.07)	−0.26	(0.12)	−0.36[b]	(0.06)	0.03	(0.08)	0.33	(0.06)
PID-5 perfectionism	0.44[b]	(0.08)	−0.38[b]	(0.08)	0.35[b]	(0.08)	0.00	(0.05)	0.08	(0.08)	0.17	(0.13)
PID-5 irresponsibility	0.42[b]	(0.06)	0.32[b]	(0.08)	0.05	(0.13)	−0.03	(0.05)	0.12	(0.11)	0.44	(0.06)
Schizotypal PD	0.39[b]	(0.06)	−0.05	(0.06)	0.04	(0.09)	0.08	(0.05)	0.36[b]	(0.07)	0.41	(0.05)
PID-5 impulsivity	0.39[b]	(0.06)	0.30[b]	(0.09)	0.11	(0.11)	−0.16	(0.05)	0.26	(0.08)	0.25	(0.08)
Panic attacks	0.35[b]	(0.06)	0.07	(0.09)	−0.05	(0.09)	0.04	(0.05)	0.15	(0.09)	0.17	(0.07)
Obsessive–compulsive PD	0.32[b]	(0.06)	−0.27	(0.07)	0.22	(0.08)	−0.01	(0.04)	0.06	(0.07)	0.30	(0.07)
Obsessive–compulsive	0.27	(0.06)	0.03	(0.07)	0.12	(0.06)	0.03	(0.05)	0.20	(0.07)	0.24	(0.06)
Drug use	0.04	(0.06)	0.60[b]	(0.11)	0.04	(0.10)	0.04	(0.06)	0.01	(0.09)	0.00	(0.00)
Alcohol use	0.02	(0.05)	0.41[b]	(0.11)	0.04	(0.08)	0.03	(0.05)	0.14	(0.09)	0.00	(0.00)
Antisocial PD	0.06	(0.06)	0.39[b]	(0.09)	0.20	(0.09)	0.10	(0.05)	0.23	(0.08)	0.02	(0.06)
PID-5 risk-taking	−0.16	(0.06)	0.37[b]	(0.11)	0.21	(0.12)	−0.05	(0.06)	0.26	(0.09)	0.22	(0.08)
PID-5 manipulativeness	−0.05	(0.04)	0.18	(0.15)	0.59[b]	(0.15)	−0.12	(0.07)	−0.01	(0.06)	0.51	(0.09)
PID-5 grandiosity	−0.09	(0.06)	−0.14	(0.14)	0.57[b]	(0.09)	0.02	(0.05)	0.27	(0.10)	0.37	(0.08)
PID-5 attention seeking	0.02	(0.04)	0.06	(0.16)	0.53[b]	(0.18)	−0.44[b]	(0.06)	0.03	(0.05)	0.32	(0.32)
Narcissistic PD	0.29	(0.08)	0.01	(0.07)	0.51[b]	(0.07)	−0.01	(0.05)	0.06	(0.06)	0.48	(0.05)
PID-5 deceitfulness	0.09	(0.05)	0.24	(0.14)	0.47[b]	(0.16)	−0.06	(0.06)	0.01	(0.10)	0.60	(0.08)

PID-5 callousness	0.10	(0.07)	0.14	(0.11)	0.39[b]	(0.10)	0.29	(0.05)	0.18	(0.14)	0.44	(0.07)
Histrionic PD	0.07	(0.04)	0.12	(0.12)	0.32[b]	(0.14)	−0.45[b]	(0.06)	0.07	(0.09)	0.32	(0.07)
PID-5 withdrawal	0.50[b]	(0.07)	−0.19	(0.06)	0.04	(0.05)	0.52[b]	(0.06)	0.10	(0.16)	0.28	(0.07)
Schizoid PD	0.28	(0.06)	0.05	(0.05)	0.01	(0.05)	0.50[b]	(0.05)	0.08	(0.12)	0.22	(0.07)
PID-5 restricted affectivity	0.00	(0.06)	0.05	(0.11)	−0.01	(0.09)	0.49[b]	(0.06)	0.27	(0.25)	0.50	(0.09)
PID-5 intimacy avoidance	0.15	(0.07)	−0.03	(0.07)	−0.06	(0.07)	0.38[b]	(0.06)	0.16	(0.18)	0.35	(0.07)
PID-5 unusual beliefs	0.03	(0.04)	0.04	(0.06)	0.05	(0.08)	0.05	(0.05)	0.79[b]	(0.04)	0.21	(0.14)
PID-5 perceptual dysregulation	0.34[b]	(0.05)	0.10	(0.07)	0.01	(0.04)	−0.01	(0.03)	0.61[b]	(0.06)	0.31	(0.12)
Psychotic symptoms	0.08	(0.06)	−0.07	(0.07)	−0.02	(0.10)	0.14	(0.06)	0.51[b]	(0.07)	0.06	(0.08)
PID-5 eccentricity	0.43[b]	(0.06)	−0.02	(0.05)	0.04	(0.05)	−0.09	(0.05)	0.46[b]	(0.06)	0.27	(0.09)
Factor correlations	Internalizing		Disinhibition		Antagonism		Detachment		Psychoticism			
Internalizing	1.00											
Disinhibition	0.19		1.00									
Antagonism	0.28		0.31		1.00							
Detachment	0.15		−0.01		−0.02		1.00					
Psychoticism	0.46		0.16		0.40		0.06		1.00			

λ, Factor loading; S.E., standard error; PID-5, Personality Inventory for the Diagnostic and Statistical Manual of Mental Disorders, fifth edition; PD, personality disorder.
[a] A Geomin oblique factor rotation was used. Methods factor loadings, although based on three factors, are presented in the same column for space considerations.
[b] Factor loadings >|0.30|.

spectrum, and the PID-5 psychoticism scales loaded on the same factor as psychotic symptoms and schizotypal PD criteria.

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, 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 social 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 model and by 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 PD has its strongest loading on the internalizing factor, reflecting the affective dysregulation associated with the construct, and schizotypal PD falls along the psychoticism spectrum. What is probably the case is that much of the characteristic interpersonal dysfunction that is the shared hallmark of the PDs (Benjamin, 1996; Meyer & Pilkonis, 2005; Pincus, 2005; Hopwood *et al.* 2013) 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 Røysamb *et al.* (2011) as opposed to Markon (2010). 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 probably are distinct underlying processes driving failures to socially engage (i.e. fear *versus* 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

https://doi.org/10.1017/S0033291715000252 Published online by Cambridge University Press

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 PID-5 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*a*, *b*, 2011; Wright *et al.* 2013). For the PID-5 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. Unexpected but theoretically consistent cross-loadings include the negative loadings of obsessive–compulsive PD and PID-5 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 a 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 suggest that the current parsing of disorders lacks validity and may have run its 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 (Plomin *et al.* 2009; Hyman, 2010). Furthermore, this probably 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.* 2014). 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. PID-5 depressivity,

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anxiousness) overlap with clinical syndromes, others do not have such explicit representation (e.g. PID-5 hostility, submissiveness). In many respects, the observed item similarities across domains are consistent with our view that these domains share items because they are not wholly distinct domains. Strictly differentiating personality from psychopathology probably 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.

Conclusion

In conclusion, the results of the models estimated here suggest that large proportions 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

For supplementary material accompanying this paper visit http://dx.doi.org/10.1017/S0033291715000252

Acknowledgements

This work was supported by the National Institute of Mental Health (L.J.S., R01MH80086; A.G.C.W., F32MH097325). We thank William Calabrese, Julie Gass, Jane Rotterman, Wern How Yam and Kerry Zelazny for their help in data collection and preparation.

Declaration of Interest

None.

Notes

[1] We note that Kendler *et al.* (2011) also examined the joint genetic structure of clinical syndromes and PDs 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).

[2] Participants 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.

[3] We 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.

[4] Given the demographic diversity in the sample, we re-estimated the final five-factor model while simultaneously regressing all indicators on sex, 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.

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