

RUNNING HEAD: Mapping a Way Forward for the Clinical Neuroscience of PDs

Neurobiological Investigations of Dimensionally Conceptualized Personality Pathology:

Mapping a Way Forward for the Clinical Neuroscience of Personality Disorders

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Despite significant efforts, relatively limited progress has been made with regard to attempts to fully understand the links between personality dysfunction and neurobiology. A growing consensus, buttressed by a reliable empirical literature, is that advancement has been impeded by a reliance on the categorical classification of personality disorders (PDs) as described by earlier versions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and the *International Statistical Classification of Diseases and Related Health Problems* (ICD) rather than an inherent limitation in the endeavor. As described briefly below, and demonstrated through the series of papers included in this special section of *Journal of Personality Disorders*, dimensional models of personality pathology offer a promising strategy for addressing these challenges and accelerating the clinical neuroscience of personality disorders.

Historical Roots

The concept of personality pathology has its roots dating back to the beginning of formal training models in psychiatry (for a brief historical review, see Lilienfeld & Litzman, 2018). For example, in Kraepelin's (1907) influential psychiatry textbook, he described personality disorders (PDs) not as distinct conditions, but instead as mild manifestations (*formes frustes*) of mental disorders. Consistent with Kraepelin's early consideration of PDs lying on a continuum with other mental disorders, a clear foreshadowing of dimensional models, Schneider (1923) later distinguished abnormal from normal personality with the former as simply more extreme than the latter. Taken together, it was clear from early on in the scholarly study of personality pathology that the differentiation between normal and abnormal is one with regard to degree *not* kind. Nonetheless, PDs became formally instantiated within diagnostic nosology in the third

edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980) as manifest polythetic dichotomies; that is, categorical diagnoses defined by observable sets of symptoms whereby an individual must have, for example, four of five symptoms to meet criteria for the diagnosis. Whereas few changes were instituted with the publication of DSM-IV (American Psychiatric Association, 1993), in which the inclusion of PDs and categorical diagnostic entities persisted, it was not without good reason that Alan Frances, Chairman of DSM-IV, openly raised concerns with the categorical model included in the manual describing categorical PD diagnoses as “an inherently futile attempt to type a continuum that is without clear boundaries.” With regard to a dimensional model for considering PDs, he went on to assert that it is “not whether, but when and which” (Frances, 1993, pg. 110).

Overcoming Limitations of Categorical Models

As described in detail elsewhere (e.g., Clark, 2007; Tyrer et al., 2006; Widiger & Simonsen, 2005; Widiger, 2007; Widiger & Trull, 2007), problems with categorical PDs have been well-documented and are generally well-known in the field. Although a detailed description of these problems is outside the scope of this brief article, critical limitations with regard to categorical PDs include: the use of arbitrary boundaries to distinguish between normal and abnormal functioning, excessive comorbidity among PDs as well as between PDs and clinical disorders, extensive within-disorder heterogeneity, relatively low diagnostic stability, and limited treatment utility (for a comprehensive review, see Clark, 2007). All told, it has become abundantly clear that continuing to categorize is clearly limiting to the field.

Personality pathology is not categorical in nature; indeed, this is true for all forms of psychopathology (Eaton, Krueger, South, et al., 2011; Haslam, McGrath, Viechtbauer, & Kuppens, in press; Hopwood et al., 2018; Krueger et al., 2018). Thus, not surprisingly, categorical diagnoses do not represent appropriate phenotypic targets for neurobiological investigations aimed at elucidating neural correlates of personality pathology. Unfortunately, this therefore obstructs our ability to develop effective biologically-informed interventions. Dimensional models of personality pathology offer a promising strategy for addressing these challenges (Clark, 2007; Latzman, DeYoung, & The HiTOP Neurobiological Foundations Workgroup, 2020). Indeed, dimensional models provide a much superior approach to conceptualizing PDs and address many of the limitations posed by categorical PD diagnosis. For example, the use of binary diagnostic categories reduces statistical power, as well as reliability and validity, relative to the use of dimensional scores (Markon, Chmielewski, & Miller, 2011). This improvement is particularly critical for clinical neuroscience specifically, given the commonality of relatively small effect sizes and the broader importance of reproducibility of our science (Button et al., 2013).

Dimensional models are further consistent with a variety of influential initiatives including the National Institute of Mental Health Research Domain Criteria (RDoC, Insel et al., 2010), meant to explicate a dimensional organization of systems of relevance to psychopathology across units of analysis and the Hierarchical Taxonomy of Psychopathology (Kotov et al., 2017; Krueger et al., 2018), an empirically-derived dimensional organizational psychopathology framework. Dimensional approaches of these sorts not only more accurately represent the nature of psychopathology, including

personality pathology, but also provide more optimal biological signal for neurobiological investigations (Latzman et al., 2020; Zald & Lahey, 2017) and improved clinical utility (Ruggero et al., 2019; Mullins-Sweatt et al., 2020).

Towards a Dimensional Nosology of Personality Disorder

As a result of a large and reliable empirical literature emphasizing the superiority of dimensional over categorical models of classification of personality disorders, it is now generally widely accepted that the diagnosis and classification of personality pathology is best accomplished within a dimensional framework (Clark, 2007; Kotov et al., 2017; Krueger et al., 2018; Widiger & Simonsen, 2005; Widiger, 2007;). This understanding is reflected, potentially most notably, in recent movements across diagnostic systems. Indeed, the alternative model of personality disorders (AMPD) in *DSM-5* (American Psychiatric Association, 2013), as well as the recently approved *ICD 11* (World Health Organization, 2018) Personality Disorder Model, both conceptualize PDs in terms of dimensions of trait personality, continuous with normal personality variation, along with a general dimension of severity (Krueger & Markon, 2014; Tyrer, Mulder, Kim, & Crawford, 2019).

In an attempt to address the significant limitations of traditional categorical models, the AMPD, situated in Section III of *DSM-5* (“emerging models and measures”), characterizes personality disorders as constellations of pathological dispositional traits within a dimensional framework. The AMPD includes two primary components: Criterion A, which concerns impairments with regard to the sense of self and interpersonal relatedness, and Criterion B which concerns maladaptive personality trait dimensions. With regard to Criterion B, specifically, it comprises five higher-order pathological

personality domains encompassing 25 lower-order trait facets (American Psychiatric Association, 2013; Krueger et al., 2012): *Negative Affect, Detachment, Antagonism, Disinhibition, and Psychoticism*.

Even greater progress towards an empirically-based classification approach is evident in the classification of personality disorders in ICD-11 in which a dimensional model will completely replace the previous categorical approach (World Health Organization, 2018). Despite being developed independently, ICD-11 personality domains are strikingly similar to the DSM-5 AMPD domains. Indeed, the only significant difference in ICD-11 is that Psychoticism is replaced by *Anankastia*, a trait domain reflecting a narrow focus on one's rigid standard of perfection and of right and wrong, and on controlling one's own and others' behavior, and controlling situations to ensure conformity to these standards. The inclusion of this domain reflects the World Health Organization considering psychoticism to be part of the schizophrenia spectrum rather than reflecting personality pathology, per se. Not surprisingly, ICD-11 and DSM-5 AMPD traits have been found to be largely commensurate (Bach et al., 2017) allowing for models to be used interchangeably in service of advancing our understanding of personality pathology.

Special Section on Neurobiological Investigations of Dimensionally Conceptualized Personality Pathology

In light of these important movements in the field, this special section of *Journal of Personality Disorders* focuses on leveraging dimensional models of personality pathology for neurobiological investigations. The series of papers included in this special section provide novel and meaningful perspectives on the nature and meaning

of connections between neurobiology and dimensional conceptions of personality pathology, as discussed below.

In one of the very first studies of this kind, Bower and colleagues (this issue) demonstrate that AMPD traits, which are based on factor analyses of self-report data, need to be configured differently to interface better with neurobiological signals, indexed in this case using variants of the P300 brain response. Their findings highlight the potential of such an approach to inform theoretical models of PD (in terms of reliable, distinct and biologically-based dimensions of personality pathology) and to aid discovery of biomarkers of specific dimensions of PD that can bring rigor to the assessment of personality psychopathology within clinical settings.

Afzali and colleagues (this issue) probe, for the first time, associations between the functional connectivity of resting-state networks and the externalizing dimension in adolescents using a general-specific dimensional framework. Their findings underscore the importance of both lower and higher order factors of PD in neuroscientific studies as we progress towards a comprehensive neurobiologically-informed dimensional model of personality pathology. Bertuli and colleagues show, based on the pattern of brain (left anterior insula) activity during experienced (self) and observed (others') pain in a non-clinical sample of people with varying levels of dimensions of psychopathy, that psychopathy is characterized by deficits in spontaneous empathizing with others' distress. Importantly, this finding was specific to the coldheartedness and self-centered impulsivity dimensions, (but not fearless dominance sub-factor) of psychopathy, again highlighting the significance of considering lower-order dimensions. Crucially, the activation deficit (and by inference, the behavioral deficit) diminished when the

instructions to participants required effortful processing of others' distress, suggesting potential targets for intervention efforts.

Lastly, Allen et al. (this issue) point out in their theoretical paper that the majority of existing personality neuroscience studies have been conducted using an 'associational approach' and in a theoretical vacuum. Consequently, the lion's share of findings in the literature, while helping to elucidate the neural circuits related to specific PD traits, have failed to provide mechanistic accounts of personality function, or dysfunction. They argue that the field now needs to move on from 'description' to 'explanation' and advance to theory-informed hypothesis-driven studies that can formally assess, and experimentally manipulate (for example, using pharmacological challenges), the hypothesized causal pathways connecting a PD dimension to its corresponding behavioral systems and neurobiological substrates.

Brief Recommendations for Advancing the Clinical Neuroscience of Personality Pathology

It likely goes without saying that for the PD field to progress it is imperative that we move beyond considering categorical diagnoses in our research. We offer some recommendations for increasing the biological signal associated with PDs, signal that is substantially obstructed as a result of the many limitations of categorical diagnoses, as well as refining the dimensional model/s of PD so they can usefully link neurobiological findings to clinical observations and potential interventions.

First, it is recommended that neurobiological studies of personality pathology move away from case-control designs based on categorical diagnoses. In case-control designs, a variety of extraneous factors confound differences between cases and

controls on dimensions of interest. Instead, as demonstrated in the current collection of papers, researchers should focus on sampling from the general population or unselected patient populations, potentially oversampling participants in the range of high risk on the dimensions of interest (Latzman et al., 2020). This will result in, among other benefits, more efficient and impactful efforts and promises to streamline the search for mechanisms and treatment targets, for example by establishing independent contributions of different PD trait dimensions to chosen behavior and neurobiological signals (Bertuli et al, 2020) and vice versa (Bowyer et al., 2020).

Second, given extensive covariation of traditional clinical and personality disorders (formally distinguished on Axis I and Axis II of DSM-IV, respectively), along with converging evidence that many forms of psychopathology can be integrated within a common meta-structure (e.g., Conway, Latzman, & Krueger, 2019; Kotov et al., 2011; 2017; Krueger et al., 2018; Markon et al., 2010), it is recommended that researchers consider dimensional assessments of both pathological personality traits as well other forms of clinical symptomatology. Integrated research of this kind has the potential to advance both process- and treatment-oriented efforts by allowing a clearer understanding of the trait dimension/s associated with particular maladaptive behavior/s or symptoms, the conditions during which such associations may emerge, and the severity (of traits) at which dimension-specific interventions may be needed.

Third, it is imperative for investigators to consider the multidimensional nature of many of the constructs they study. Indeed, whereas dimensional methodologies significantly help to overcome the extensive within disorder heterogeneity plaguing categorical PDs, many dimensional constructs are broad and multidimensional. As a

particularly clear example, as described above, while the AMPD includes five higher-order pathological personality domains, each domain includes a number of lower-order trait facets, 25 in total. Subdimensions may show not only specific but, at times, contrasting associations with variables of interest (e.g., Litzman, Patrick, & Lilienfeld, 2019). Ignoring lower-order subdimensions may thus result in erroneous conclusions or loss of important information, for example, in relation to conduct problems factor of adolescent externalizing domain (Afzali et al., 2020).

Fourth, investigators are encouraged to include assessments across measurement modalities (e.g., report-based, task-based, neurophysiological) in their research moving beyond one-to-one studies of traits and neurophysiological assessments. Given an increased focus on deviations across domains of functioning contributing to various disorders, oftentimes termed “transdiagnostic mechanisms,” researchers are encouraged to assess and integrate across modes of assessment. Indeed, it is only through consideration of various levels or units of analysis, from genes through observable behaviors, that a full characterization of clinical phenomenon of interest is possible (Insel et al., 2010; Perkins, Litzman, & Patrick, 2020). That is, consideration of the way in which liability factors, and associated clinical manifestations, are expressed across measurement modalities can help to advance the elucidation of neuropathogenic processes associated with personality pathology (Perkins, Joyner, et al., 2020). Recent emphasis on studies that can provide mechanistic accounts of personality function/dysfunction demands a multimodal approach and multilevel analysis to fully describe the adaptive or maladaptive functions of PD trait dimensions and the psychological and neurobiological processes involved in their implementation

(Allen et al., 2020). It will further be important to consider the way in which developmental processes may represent mediating pathways with regard to transactional associations between the environment and neurobiology (e.g., Fonagy, Luyten, & Allison, 2015).

Fifth, it is strongly recommended that future investigations use sufficiently large sample sizes, and ensure a good coverage of scores on the trait domain/s of interest, to yield reliable observations, especially in relation to true associations of small effect sizes, and enable discovery of both linear and non-linear relationships. Furthermore, given known sex differences in human brain structures and function (Richie et al., 2018; Lotze et al., 2029) and possibly in certain PD traits or their expression (Sansone and Sansone, 2011), consideration of participant's sex will also be important for studies aiming to clarify the neurobiology of personality pathology.

Finally, to the extent possible, we recommend that study protocols and data (with metadata) from ongoing and future studies, as well as from previous studies where feasible, are made available through online data repositories and contribution to public datasets to increase transparency and enable large multilevel investigations.

Conclusions and Future Directions

It is now abundantly clear that personality pathology is best considered dimensionally rather than categorically. A burgeoning literature further suggests that it is through an empirically-based, dimensional consideration of personality pathology that we will be able to advance our understanding of the clinical neuroscience of personality pathology. It has been almost 30 years since Alan Frances' famous acknowledgement,

the “when” should be now and, thanks to efforts represented in Section III of *DSM-5* and the newly approved ICD PD model, the which is becoming more and more clear. It is hoped that this special section will serve to catalyze the field resulting in an acceleration of progress in explicating the clinical neuroscience of personality pathology. It is further hoped that this special section will affirm *Journal of Personality Disorders’* interest in remaining at the forefront of the science of personality pathology and the *Journal’s* encouragement to authors to continue to consider JPD as an outlet for papers in this area.

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