An update on diagnostic issues for borderline personality disorder

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The diagnosis of mental illness and structure of psychopathology in classification systems such as the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) is cross-sectional, relying on reported symptoms within a specified period of time. This fails to address why psychiatric illnesses persist in some people and why clinical presentations change so radically across time. These weaknesses are relevant to proposed changes to the diagnostic systems of the DSM and International Classification of Diseases 11th Revision (ICD-11), and particularly to personality disorders (PDs), which change over time, recur, and are comorbid and complex.

In this article we will discuss the traditional DSM approach to PD, the alternative approach set out in Section III of DSM-5,[1] and the new approach proposed for the forthcoming ICD-11. We suggest what may be a way forward for thinking about the conceptualization of PD, concluding that an integrative dimensional model may be the most clinically valuable and theoretically coherent approach.

The traditional DSM model

DSM-5 promised to revolutionize the practice of psychiatric diagnosis. However, in the eyes of many clinicians and researchers it continues to struggle because (a) it retained diagnosis on the basis of clinical observation and patient phenomenological symptom reports—that is, the disease is diagnosed as the constellation of symptoms, despite the fact that neuroscience, behavioral science, and genetic science do not support this; and (b) it kept the polythetic and dichotomous (categorical) diagnostic system (e.g., 5 out of 9 symptoms for BPD), which gives symptoms equal weight and results in the same symptoms being manifest across a range of possible disorders.

For example, in DSM-IV,[2] 1,750 combinations of symptoms could culminate in a diagnosis of post-traumatic stress disorder. In DSM-5, the possible combinations of symptoms increased to more than 10,000. The mental disorders as per DSM are not

biologically valid disease entities. Diagnostic systems cannot be purely based on phenomenology.

The criticism of the DSM categorical model is particularly pertinent in the case of PDs. The typal approach to PDs, as presented in Section II of DSM, provides 10 discrete diagnostic categories of PD. However, the attempt to categorize in this way, for example, a category such as borderline PD (BPD), is undermined by excessive comorbidity, excessive within-diagnosis heterogeneity, marked temporal instability, the lack of a clear boundary between normal and pathological personality, and poor convergent and discriminant validity.[3] This creates problems for clinicians and researchers alike; for example, the various available evidence-based treatments may have been studied in different populations and may not be equally applicable to all subtypes of BPD. However, there is a degree of consensus that BPD incorporates three related core features: emotion dysregulation, impulsivity, and social-interpersonal dysfunction. These core features are significant as they are suggestive of general difficulties in social communication that may cut across psychopathology.

The categorical model for PD reproduced in DSM-5 Section II is not empirically supported. This has been confirmed in meta-analyses.[4,5] As a recent review concluded, "…not only do PD categories covary due to shared and correlated latent dimensions but at least most of them fall apart once symptoms are analyzed".[6, p. 132,7]

DSM-5 Section III: The alternative model for PDs

In an attempt to resolve these difficulties, Section III of DSM-5 proposes an alternative model for PD consisting of three components:

1. Level of personality functioning. This has four subcomponents of identity and self-direction (both relating to the relationship to the self), and empathy and intimacy (both relating to interpersonal functioning). Severity of impairment predicts whether the individual meets the general criteria for PD, with more severe impairment predicting

whether an individual has more than one PD diagnosis, or one of the more typically severe forms of PD

2. Specific PD diagnoses, which are reduced to six (as opposed to 10 in the existing model
3. A system of pathological personality traits. These traits are organized into five domains: negative affectivity, detachment, antagonism, disinhibition and psychoticism. Within these domains, there are 25 trait facets.

From this perspective, people with BPD will be identified by impairment in personality functioning, characterized by difficulties in two or more of the following four areas:[1]

1. Identity: impoverished, poorly developed self-image, often excessive self-criticism; chronic feelings of emptiness; dissociative states under stress
2. Self-direction: instability in goals, aspirations, values, career plans
3. Empathy: impoverished ability to recognize the feelings and needs of others, especially as a result of hypersensitivity—i.e., feeling rejected or insulted; perceptions of others are negatively biased
4. Intimacy: intense, unstable, and conflicted close relationships characterized by mistrust and neediness; close relationships often viewed in extremes of idealization and devaluation, reflected in a pattern or over-involvement or withdrawal.

The trait stage of diagnosis for BPD requires fulfilment of four or more of the following seven traits: emotional lability, anxiousness, separation anxiety, depressivity, impulsivity, risk-taking, and hostility. Of the four or more traits fulfilled, at least one of these must be impulsivity, risk-taking, or hostility.

This alternative model is dimensional in nature, which is in keeping with research evidence indicating that "personality disorders are continuous with normal personality",[8, p. 364], and the personality functioning scale accommodates a severity factor, which is a good

predictor of outcome.[8] The main criticism has been that the new model, with its use of dimensional and trait approaches, is an "unwieldy conglomeration of disparate models that cannot happily coexist and raises the likelihood that many clinicians will not have the patience and persistence to make use of it in their practices".[9, p. 1026] Clinicians should not be expected to regard their patients in terms of so many subcomponents.[9] However, it also keeps a categorical/typal model (in the form of the six specific PD diagnoses) alongside the dimensional model. This hybridization requires two incompatible assumptions—that psychopathology is continuous with normality, and that a diagnosis is "a distinct type that is either present or absent, which is also discontinuous with related constructs and, in the case of personality disorder, with normal personality"[8, p. 366]—which disregards the lack of empirical evidence for discontinuous types.[8]

ICD-11

The ICD-11, which is currently in development, proposes a dimensional approach to the classification of PDs. There will be one general diagnosis for PD: the criteria for this are described as "a relatively enduring and pervasive disturbance in how individuals experience and interpret themselves, others, and the world that results in maladaptive patterns of cognition, emotional experience, emotional expression, and behaviour".[10, p. 722] These patterns are entrenched and result in significant difficulties in psychosocial functioning, particularly in interpersonal relationships; the disturbances range across personal and social situations; and are relatively stable over time.

Once the general diagnosis of PD has been made, the level of impairment is identified as mild, moderate, or severe. In addition, there is the subthreshold category of personality difficulty; this is not a disorder, and refers to a disturbance that might manifest sporadically or in particular contexts. The emphasis, therefore, is on PD in general and its severity, rather than on categories of PD. Severity is assessed on the extent of social dysfunction, the level of

risk to self and others, and the overlap of trait domains that capture an individual's PD profile. These domain traits are "not categories, but rather represent a set of dimensions that correspond to the underlying structure of personality dysfunction".[10, p. 723] The proposed domain traits are: negative affective features; dissocial features; features of disinhibition; anankastic features; and features of detachment. In individuals with more severe PD, more than one domain trait is likely to present.[10]

The proposed ICD-11 is clearly a break from previous ICD and DSM systems of diagnosis in that it ceases to use type-specific categories of PD; the single diagnostic category is of the presence or absence of PD itself, and discrimination is instead made on the basis of severity and the expression of domain traits. This resolves the issue of comorbidity across different categories of PDs. For example, BPD would classically involve an emphasis on negative affect; BPD comorbid with antisocial PD—a frequently used traditional diagnosis—might manifest as moderate or severe PD with dissocial features and features of disinhibition as well as negative affect. The ICD trait domains, although not using the language of typal categorization, can be understood as constituting a way of making sense of a patient's behaviors in terms of severity *and* typical styles of behavior and their underlying cognitive processes, which some might think, comes perilously close to categories.

Ways forward

At the heart of this discourse is whether PD can be understood as one dimensional continuum, or made up of discrete but overlapping diagnostic categories, or whether a hybrid model combining dimensional and categorical approaches is the most fitting. To date, there are few data that compare categorical, dimensional, and hybrid models of PD.

We suggest a new direction that combines (a) recent research on the structure of personality pathology and the structure of psychopathology more generally, and (b) developments related to resilience and a theory of social learning, the theory of *epistemic*

trust. The implications of these lines of thinking are that an integrated dimensional model is the most coherent from both a clinical and a research perspective: PDs are best understood as existing on a continuum of *persistence* of symptoms over time, which encompasses normal personality functioning up to the most severe personality pathology; but some form of categorization that captures an individual's profile of behavioral difficulties and forms of distress and social dysfunction is necessary to understand the manifestations of pathology and how best to understand the individual clinically and, ultimately, treat them.

The 20-year analysis of the Dunedin longitudinal study by Caspi and colleagues[11] suggested the existence of a general factor in psychopathology. Caspi and colleagues found that vulnerability to mental disorder was more convincingly described by a bi-factor model comprising a general psychopathology factor (labelled "p") and three spectral factors (internalizing, externalizing, and thought disorder), rather than by the spectral factors alone. A higher "p" factor score was associated with "more life impairment, greater familiality, worse developmental histories, and more compromised early-life brain function".[11, p. 131] This work has been confirmed by other studies extending the validity of the "p" factor concept into childhood and adolescence[12] where the measure of an overarching psychopathology factor substantially improved the prediction of mental disorder over a 3-year period.[13] In this context, "p" could stand for the *persistence* of mental disorder.

The idea of a general construct that underpins vulnerability to psychopathology has also been considered specifically in PD; a recent study by Sharp and colleagues at the Menninger Clinic explored whether there is a general PD factor that underlies different diagnoses for PD.[14] Bi-factor analyses of the DSM PD criteria confirmed several different PDs but indicated that they also load on to a general factor that includes all the BPD criteria, rather than the latter representing a separate PD category. It appears that BPD might be better understood as being at the core of personality pathology more generally, rather than as a type

of PD; this approach would help to make sense of the high levels of comorbidity found in BPD patients.

Caspi and colleagues found that individuals who scored highly on the general psychopathology scale were characterized by "three traits that compromise processes by which people maintain stability—low Agreeableness, low Conscientiousness, and high Neuroticism … that is, high-p individuals experience difficulties in regulation/control when dealing with others, the environment, and the self".[11, p. 131] Such a profile, of course, captures the core features of BPD: emotion dysregulation, impulsivity, and social dysfunction. BPD is similar to high "p", and as BPD features appear to be core to all PDs, we may infer that there is at least a superficial association between high "p" scores and the likelihood of a PD diagnosis, which in turn predicts an increased likelihood or persistence of a future mental disorder.

Construed in this way, moving from a cross-sectional to a developmental psychopathology frame, enables us to reverse our lens and shift from investigating the mechanisms that lead to adversity-related illness to investigating the mechanisms that protect against the impact of adversity, that is, resilience—the work of adaptive mechanisms with a biological basis that protect the individual from disorder despite the experience of hardship. We suggest that the measurement captured in general factors for psychopathology ("'p") is the same construct that determines an individual's resilience, or lack of it. Can we reconceptualize the construct of high "p" (suggesting persistence), that is, PD with BPD features, as *the relative absence of a capacity to withstand adversity*, or a *lack of resilience*?

ICD-11 will suggest an explicit link between PD and compromised interpersonal or social function. We can readily reverse this and see PD as an incapacity to adapt to changing social contexts. An individual with PD is impaired in appraising social situations, less able to extract relevant social information from their current interpersonal context, and compromised

in evaluating social information to update their interpersonal schemas or expectations. Consequently, they appear rigid, leading to the assumption that their pathology is rooted in the most stable psychic structure we can conceptualize: their personality. Yet, we know from follow-along studies that personality disorder is hardly stable.[15] What we do have evidence of is the increased likelihood of persistence of continuous dysfunction in this group. Resilience assumes that protection from adversity is commensurate with the availability of and capacity to make use of social and environmental support. Those least capable of appraising social contexts and learning from social experience will be at greatest risk of managing adversity poorly and most vulnerable to succumbing to social challenge, with mental disorders being triggered by adversity.

Is there a known psychological mechanism that could (at least hypothetically) account for this conceptualization? We suggest that the constructs that studies indicate represent psychopathology are measurements of an individual's level of epistemic trust, by which we mean trust in the authenticity and personal relevance of interpersonally transmitted knowledge.[16,17] This describes an individual's openness to learning from another person, acquiring information, and receiving and internalizing this new knowledge. To modify a person's behavior, social information must be coded as personally relevant and generalizable (i.e., applicable to a range of social contexts). However, access to this privileged route of communication that leads to learning and change cannot be universal. It is restricted to people whose communication we can trust as accurate and reliable—individuals to whom we extend *epistemic trust.*[18]

The evolutionary purpose of epistemic trust is to enable social learning in an ever-changing social and cultural context, by stimulating individuals to be open to acquiring new knowledge from their (social) environment; they update expectations from trustworthy sources, but show appropriate suspicion and vigilance, and reject new information as not

relevant to them, when it comes from those who have not demonstrated their trustworthiness. The epistemic channel cannot be left open by default: it is adaptive for humans to adopt a position of *epistemic vigilance* unless they are reassured otherwise.

The disruption of epistemic trust, or the emergence of outright epistemic mistrust as a result of environmental adversity, genetic propensity, or both, can lead to a fundamental breakdown in the capacity for the ongoing exchange of social communications, which can create the appearance of rigidity, inflexibility, or being hard to talk to and difficult to help. This is because, to be able to trust knowledge, we are biologically programmed to look for cues in the communicator's behavior that proves their interest in our wellbeing. We tend to extend trust to those who demonstrate interest in us and can see the world from our perspective. If they show us that they understand our point of view, we will be able to *listen* to them and not just to hear their words. Emotion dysregulation, impulsivity, and social dysfunction interact—it is hard (and possibly pointless) to try to work out which comes first. They are jointly cause and consequence. But they compromise an individual's capacity to detect genuine interest and to approach social communication with epistemic trust. It follows from this perspective that PD may be a state of profound and chronic epistemic mistrust that bars individuals from social communication, making them appear rigid and "hard to reach". Perhaps these patients require longer-term therapy, whatever their presenting symptoms, to help overcome their vigilance in relation to learning from their therapist. The therapist needs to be exceptionally explicit in adopting the patient's perspective, which will serve to generate epistemic trust and open the patient to social learning. It is only by addressing this limitation of social communication that epistemic vigilance can be lifted so that the benefits of improved social knowledge can be experienced within the wider social environment.[18,19]

We are all more or less epistemically trustful or distrustful. The epistemic trust model of PD thus requires an integrative and dimensional approach. This involves thinking not in terms of

classes of patients based on traditional phenomenological indicators and behavioral trajectories common to different clinical phenotypes, but assuming an underlying common factor of vulnerability to adverse social conditions, a lack of resilience (too much "p"), and additional neurobiological drivers that generate different symptom profiles. Both are relevant and must be examined using state-of-the art models of the dimensional structure of psychopathology in real-life psychiatric settings.

References

1. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders.* 5th ed. Washington, DC: American Psychiatric Association; 2013.

2. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders.* 4th, text rev. ed. Washington, DC: American Psychiatric Association; 2000.

3. Skodol AE, Clark LA, Bender DS, et al. Proposed changes in personality and personality disorder assessment and diagnosis for DSM-5 Part I: Description and rationale. *Personal Disord.* 2011;2:4-22. doi: 10.1037/a0021891.

4. O'Connor BP. A search for consensus on the dimensional structure of personality disorders. *J Clin Psychol.* 2005;61:323-345. doi: 10.1002/jclp.20017.

5. O'Connor BP, Dyce JA. A test of models of personality disorder configuration. *J Abnorm Psychol.* 1998;107:3-16.

6. Wright AGC, Zimmermann J. At the nexus of science and practice: Answering basic clinical questions in personality disorder assessment and diagnosis with quantitative modeling techniques. In: Huprich SK, ed. *Personality disorders: Toward theoretical and empirical integration in diagnosis and assessment*. Washington, DC: American Psychological Association; 2015:109-144.

7. Sheets E, Craighead WE. Toward an empirically based classification of personality pathology. *Clin Psychol (New York).* 2007;14:77-93. doi: 10.1111/j.1468-2850.2007.00065.x.

8. Livesley WJ. Disorder in the proposed DSM-5 classification of personality disorders. *Clin Psychol Psychother.* 2012;19:364-368. doi: 10.1002/cpp.1808.

9. Shedler J, Beck A, Fonagy P, et al. Personality disorders in DSM-5. *Am J Psychiatry.* 2010;167:1026-1028. doi: 10.1176/appi.ajp.2010.10050746.

10. Tyrer P, Reed GM, Crawford MJ. Classification, assessment, prevalence, and effect of personality disorder. *Lancet.* 2015;385:717-726. doi: 10.1016/S0140-6736(14)61995-4.

11. Caspi A, Houts RM, Belsky DW, et al. The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Clin Psychol Sci.* 2014;2:119-137. doi: 10.1177/2167702613497473.

12. Lahey BB, Rathouz PJ, Keenan K, et al. Criterion validity of the general factor of psychopathology in a prospective study of girls. *J Child Psychol Psychiatry.* 2015;56:415-422. doi: 10.1111/jcpp.12300.

13. Patalay P, Fonagy P, Deighton J, et al. A general psychopathology factor in early adolescence. *Br J Psychiatry.* 2015;207:15-22. doi: 10.1192/bjp.bp.114.149591.

14. Sharp C, Wright AGC, Fowler JC, et al. Borderline personality pathology as the 'g' factor of personality disorder. *J Abnorm Psychol.* 2015;124:387-398. doi: 10.1037/abn0000033.

15. Gunderson JG, Stout RL, McGlashan TH, et al. Ten-year course of borderline personality disorder: Psychopathology and function from the Collaborative Longitudinal Personality Disorders study. *Arch Gen Psychiatry.* 2011;68:827-837. doi: 10.1001/archgenpsychiatry.2011.37.

16. Sperber D, Clement F, Heintz C, et al. Epistemic vigilance. *Mind Lang.* 2010;25:359-393. doi: 10.1111/j.1468-0017.2010.01394.x.
17. Csibra G, Gergely G. Natural pedagogy as evolutionary adaptation. *Philos Trans R Soc Lond B Biol Sci.* 2011;366:1149-1157. doi: 10.1098/rstb.2010.0319.
18. Fonagy P, Luyten P, Allison E. Epistemic petrification and the restoration of epistemic trust: A new conceptualization of borderline personality disorder and its psychosocial treatment. *J Pers Disord.* 2015;29:575-609. doi: 10.1521/pedi.2015.29.5.575.
19. Fonagy P, Luyten P. A multilevel perspective on the development of borderline personality disorder. In: Cicchetti D, ed. *Developmental psychopathology*. 3rd ed. New York, NY: John Wiley & Sons; 2016.