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REVISING THE BORDERLINE DIAGNOSIS FOR DSM-V: AN ALTERNATIVE PROPOSAL

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Abstract

The changes in the borderline personality disorder (BPD) diagnosis proposed by the DSM-V personality disorder work group involve radical changes in format (prototype and dimensions) and descriptive characteristics (traits). Changes of this magnitude will create an unwelcome and potentially harmful discontinuity with the definition that has guided BPD research and the development of disorder-specific therapies. This paper offers an alternative proposal that was developed in collaboration with clinical and research leaders. It includes modification of existing criteria, use of a diagnostic algorithm based on phenotypes, and giving BPD a hierarchical relationship vis-à-vis other personality disorders. These changes are incremental, diminish overlap and heterogeneity, sustain clinical and research development, and will improve utilization.

Within a year after borderline personality disorder's (BPD) coming of age was celebrated by the American Journal of Psychiatry (Kernberg & Michaels, 2009; Oldham, 2009) and the American Psychiatric Association's Annual Meeting, the DSM-V Personality Disorder Work Group has proposed major changes in the BPD diagnosis (prototypes, traits, and dimensions; see dsm5.org). A thoughtful consideration of such change is timely insofar as changes have been few despite an ever-expanding body of research (Blashfield & Intoccia, 2000; Gunderson, 2009). The BPD syndrome defined in DSM-III, III-R, and IV is frequently criticized for too much overlap with other personality disorders and its polythetic algorithm allows too much heterogeneity.

The proposed changes by the DSM-V work group radically alter a definition of BPD that has survived with minimal changes since it entered the DSM system 30 years ago and from which has come a body of knowledge about heritability (Distell et al., 2008; Kendler, Meyers, Torgerson, Neade, & Reichborn-Kjennerud, 2008; Torgerson et al., 2000, 2008), prevalence (Grant et al., 2008; Lenzenweger, Lane, Loranger, & Kessler, 2007; Trull, Jahng, Tomko, Wood, & Sher, in press), developmental antecedents (Cohen, Crawford, Johnson, & Kasen, 2005; Winograd, Cohen, & Chen, 2008; Yen et al., 2002), markers of risk (Crick, Murray-Close, & Woods, 2005; Lyons-Ruth, Melnick, Patrick, & Hobson, 2007), course (Skodol et al., 2004; Zanarini et al., 2008; Zanarini, Frankenburg, Reich, & Fitzmaurice, 2010), and treatment. As to the latter, many empirically-validated psychotherapeutic treatments have been developed while an increasing number of medication studies have been instructive (Lieb, Vollm, Rucker, Timmer, & Stoffers, 2010). Moreover, because in its present form the BPD diagnosis has been validated (Gunderson 2009; New, Triebwasser, & Charney, 2008), and because it has become a recognizable clinical entity that informs the

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hopes and the therapies for patients who would otherwise be undiagnosable, changes in its definition should proceed with caution and only with sound empirical justification.

The proposal offered here is guided by the conservative standards established in DSM-IV: all changes were required to have an empirical rationale, meaning reliably assessable with demonstrated diagnostic efficacy. In addition, changes proposed here will be guided by considerations of clinical utility (i.e., familiarity, coverage for patients who benefit from BPD's treatments), continuity with past research, and the potential to diminish problems with underutilization, oversight, and stigma.

BPD's CRITERIA

The current definition of the BPD diagnosis was largely developed in the late 1970s. It developed as a result of the first empirical study (Grinker, Werble, & Drye, 1968), a swelling clinical interest (Kernberg, 1968; Masterson, 1972), a review of relevant literature (Gunderson & Singer, 1975), and the development of a reliable diagnostic method (Gunderson & Kolb, 1978, 1981). The latter provided a method to reliably distinguish BPD patients from those with schizophrenia or depression, established a cut-point/threshold for making the diagnosis, and identified seven characteristics that were highly discriminating (Gunderson & Kolb, 1978). These seven characteristics, with some modifications, and with the addition of an eighth criterion for *identity disturbance* (Spitzer, Endicott, & Gibbon, 1979), became the criteria for BPD adopted in DSM-III in 1980. Since then the only substantial change has been the addition into DSM-IV of a ninth criterion *psychotic-like experiences* in 1994.

The current criteria have all been validated, have an established research legacy, and have generally proven clinically valuable. All have been examined for their relative specificity, sensitivity, and predictive power (Clarkin, Widiger, Frances, Hurt, & Gilmore, 1983; Grilo et al., 2007; Gunderson, Zanarini, & Kisiel, 1996). The only criterion to be added since 1980, i.e., psychotic-like experiences, required significant empirical justification and demonstrating that it did not increase prevalence (Sternbach, Judd, & Sabo, 1992; Gunderson et al., 1996). These criteria have been the basis for research validating BPD as a diagnostic entity (Gunderson, 2009; New et al., 2008; Paris, 2007).

Since DSM-IV, many new studies have examined BPD phenomenology. Table 1 incorporates findings from these studies that appear to add to both the specificity and clinical valence of the current criteria.

The proposed changes in interpersonal criteria (criteria 2 & 3) derive from the growing body of research on attachment and social psychology. They can be expected to add specificity and bridge the criteria to therapies such as Mentalization Based Treatment (MBT; Bateman & Fonagy, 2004; Fonagy, Target, & Gergely, 2000) and Schema Focused Therapy (SFT; Young, Klosko, & Weishaar, 2003). The proposed changes in affective (emotion) criteria (4 & 5) derive from a growing body of research including neurophysiology and experience-sampling. While the DSM III and IV criterion for affective instability (criterion 4) has been sensitive, it has not been specific (Grilo, Becker, Anez, & McGlashan, 2009; Gunderson, Zanarini, & Kisiel, 1991). The changes proposed for this criterion capture the pervasive negativity of mood alongside specific types of instability. It will provide valuable help in distinguishing BPD from bipolar disorder—where “affective instability” has become a passport for diagnosing bipolar disorder (Zimmerman, Ruggera, Chelminski, & Young, 2010). The use of Negative links it to *neuroticism* and the use of Emotionality links this criterion to Dialectical Behavior Therapy (DBT) and the construct of *emotional dysregulation* (Linehan, 1993). The proposed change in criteria 8 (i.e., sense of badness) reflects a widely-recognized and empirically-validated aspect of the borderline patients'

sense of self and will add specificity. The change proposed for criteria 9 returns to the historically-valued observation that borderline patients lack a *sense* of reality (Frosch, 1970); that is, experiences of disconnection or dissociation, thereby shifting it from an episodic symptom into more of a personological construct (Waller & Ross, 1997).

The final change (also used in the DSM-V prototype and trail schemes), would be to move ratings of criteria from an *absent-present* dichotomy to a more dimensional four-point scale from Not to Mild to Moderate to Strong (see Table 1). This change has conceptual and statistical advantages over the current system.

The magnitude of the proposed changes in the content of the criteria is greater than those approved in DSM-III-R or DSM-IV, but are modest compared to the changes proposed by the DSM-V committee. All of the changes proposed here have been empirically shown to discriminate BPD from other disorders. None of these changes depart from the basic clinical construct each criterion was designed to represent. Still, it is unclear whether the newly added components might better be separated into their own independent criterias, e.g., alternating between overinvolved and withdrawal or chronic dysphoria.

BPD's DIAGNOSTIC ALGORITHM

DSM-IV uses a polythetic system (adding the number of criteria without weighting) with the diagnosis of BPD requiring five (or more) of the nine criteria. This means that there are 256 different combinations of criteria from which the diagnosis might be established. Two patients getting this diagnosis might overlap on as few as only one criterion. This allows too much heterogeneity in the people who can receive the diagnosis. It contrasts with the relative ease with which the diagnosis can usually be established—due to differences in the clinical weighting of criteria. One study showed that the combination of criterion 7 (self-injurious behaviors) and criterion 1 (intense unstable relations) were usually sufficient to predict the diagnosis (Grilo et al., 2007).

It would reflect what is known about BPD's latent structure and diminish the disorder's heterogeneity to subdivide the criteria set into four component sectors and base the scoring algorithm on them. Three components have been solidly established as phenotypes (signifying significant heritability) based on the results of factor analyses (Blais, Hilsenroth, & Castlebury, 1997; Clarkin, Hull, & Hurt, 1993; Hurt, Hyler, Frances, Clarkin, & Brent, 1989; Sanislow et al., 2002; Skodol et al., 2002), family studies (Gunderson et al., unpublished; Silverman et al., 1991; Zanarini et al., 2004) and increasing evidence of their endophenotypes, that is, neurobiological correlates (King-Casas et al., 2008; Koenigsberg et al., 2009; Siever, Torgerson, Gunderson, Livesley, & Kendler, 2002; Stanley & Siever, 2010). Factor analytic studies generally divide BPD psychopathology into three sector or phenotypes: *Interpersonal Hypersensitivity* (criteria 1, 2, and 3), *Affective/Emotional Dysregulation* (criteria 4, 5), and *Behavioral Dyscontrol* (criteria 6, 7). Major clinical theories (and the effective therapies that are based on them) have proposed that the *Interpersonal* (Adler, 1986; Bateman & Fonagy, 2004; Benjamin, 1993; Gunderson, 2007) or the *Affective/Emotional* (Linehan, 1993; Livesley, Jackson, & Schroeder, 1992) phenotypes are the core component. These are the phenotypes that are also more stable over time (Zanarini et al., 2007). While the *Behavioral* phenotype is not central to any major clinical theory or therapy, it gains significance by virtue of its link to a biogenetic dimension called *impulsive/aggression* (Coccaro, Bergman, & McLean, 1993, 1997), its power to explain co-aggregation patterns (White, Gunderson, Zanarini, & Hudson, 2003), and the genetic link of BPD to antisocial personality disorder (ASPD; Torgersen et al., 2008).

Kernberg's enduring theoretical and therapeutic contributions were responsible for initially introducing the *identity disturbance* criterion (criterion 8; Spitzer et al., 1979). This criterion

has proved to be reasonably efficient (Gunderson et al., 1996), but not very longitudinally persistent (Zanarini et al., 2007). The proposed change (see Table 1) should add to its specificity. The ninth criterion becomes a conceptual partner to criterion 8, and more trait-like by shifting its emphasis to recurring dissociative splits (perceiving oneself or the outer world as unreal; consistent with Akhtar & Thomson, 1982; Kernberg, 1975; Korzekwa, Dell, Links, Thabanc, & Fougere, 2010) (see Table 1). Combined they yield a fourth sector that might be identifiable as *Disturbed Self*. Such a sector has conceptual coherence, honors the contributions from Kernberg and object relations theory, and links the criteria set to Transference Focused Psychotherapy (TFP; Clarkin, Yeomans, & Kernberg, 2006). This sector also recognizes the role of dissociation that serves as a defensive flight from either aloneness (Gunderson, 1984) or feelings (Linehan, 1993). The potential centrality of this sector is reflected in the prominence it attained in the proposed DSM-V definition for all PDs (see dsm5.org) and specifically in the proposed BPD prototype (see dsm5.org).

To be diagnosed BPD, the proposed changes would require that a person continue to have ≥ 5 criteria, but require at least one criterion from at least three sectors. Several empirical questions remain; should the four phenotypes be weighed differently (as is currently done with MDD and PTSD)? Should the diagnostic threshold require psychopathology in 3 or 4 sectors? Three would be more inclusive (increased sensitivity) and might be preferred within the psychotherapeutic community; and four (increased specificity) would probably be preferred by researchers, who value narrower homogeneous samples. However these questions are answered, the proposed algorithm moves the diagnosis away from observable symptoms towards underlying phenotypes (and endophenotypes) which lie closer to the genetic template (Gottesman & Gould, 2003). Nor does the proposed shift from criteria to phenotype prematurely bias the diagnosis towards any of the competing models about which sector should be considered primary. Finally, it offers a relatively discrete way to identify a diagnostic threshold—lacking altogether in the proposed schemes of DSM-V. [This model for the BPD diagnosis is validated by family study results which show that when the phenotypes are used to establish the BPD diagnosis, it is more familial than when diagnosed with the DSM-IV criteria system (Zanarini et al presented American Psychiatric Association, 2009).]

BPD's PLACE IN THE DSM CLASSIFICATION SYSTEM

BPD does not easily lend itself to the changes (dimensions, trait profiles, and prototypes) being proposed for it in DSM-V (see dsm5.org). Many other personality disorders lend themselves to dimensional schemes because they reflect traits that extend seamlessly into normality (e.g., dependency or avoidance). Although one taxometric study indicated that BPD is more dimensionally-related to normality than categorically separate (Rothschild, Cleland, Haslam, & Zimmerman, 2003), BPD's diverse components (noted above), each of which might be dimensionalized, cannot easily be blended into any single dimension. Several factor analytic studies show that the disorder is best modeled as a single factor (Fossati et al., 1999; Clifton & Pilkonis, 2007), but we can't yet identify what that core factor is. Nor does BPD fit into the quantitatively-developed architecture of personality traits: it relates equally to both internalizing and externalizing (Depue & Lenzenweger, 2005), and to both antagonism and negative emotionality— thus being “interstitial” (Krueger & Eaton, in press). The traits definition proposed for BPD (see dsm5.org) is not empirically-based and is completely divorced from clinical concepts and literature. Prototypes have been shown to have advantages for clinicians in identification of personality disorders (Shedler & Westen, 2004), but BPD already has a validated and clinically-useful definition for which a format change of such magnitude cannot be easily justified. The prototype model returns us to the problems of DSM I and II and sets BPD and other PDs apart from all other medical diagnoses. Moreover, the specific prototype proposed for BPD

in DSM-V (see dsm5.org) is relatively discursive, theory-driven (the concepts of self and identity are central), has unknown and probably difficult-to-attain reliability, and is not tightly anchored to research citations or to databases from either DSM-IV or the Shedler-Westen Assessment Procedure (SWAP; Westen & Shedler, 1999).

Genetically, Kendler et al. (2008) have indicated that BPD shares some of the genetic template common to personality disorders but much of its genetic vulnerability is not. (Moreover, it shares just as much with the genetic template for the axis I sector of psychopathology; Kendler et al., in press.) [The idea of some still unidentified and specific core to BPD psychopathology is further supported by preliminary results of the Family Study in which the disorder appears to be more familial than are its phenotypes (Gunderson et al., 2010).] Such research findings would seem to confirm Paris's (2007) earlier conclusion that BPD appears to be "more than the sum of its parts" (p. 968).

As a clinical entity, BPD does not fit comfortably alongside the other personality disorders. Certainly it is the personality disorder that is the most dystonic (Paris, 2007) and most prevalent in clinical settings. The intensity and duration of treatment utilization by BPD patients and their severe social dysfunction (and derivative costs) underscore the disorder's extraordinary public health significance. The psychotherapeutic office-practice community is likely to use personality diagnoses as a primary way to organize their treatment, but in other settings the personality disorders are likely to be considered as either: (1) adjunctive to axis I disorders (i.e., they may help clinicians anticipate resistances), or (2) latent vulnerabilities to presumably primary Axis I disorder(s). Both conceptualizations are an argument to keep the PDs in a separate and subordinate relationship to Axis I disorders in DSM-V, as has been the case since Axis II was introduced in DSM-III. But neither of these models reflect BPD's relationship to axis I. The Collaborative Longitudinal Personality Disorders Study (CLPS) has shown that BPD has a negative effect on the course of substance use disorders (Walter et al., 2009), major depression (Gunderson et al., 2004, 2008; Morey et al., 2010), bipolar disorder (Gunderson et al., 2006) and panic disorder (Gunderson et al., unpublished), but these disorders had no, or relatively little, reciprocal effect on the course of BPD. Thus, even in the presence of major Axis I comorbidity, BPD should often be a primary target for treatment. Only ASPD might claim to have a comparably negative effect on the prognosis for Axis I disorders (Gunderson, 1984; Kernberg, 1968; Reich, 1988; Woody, McLellan, Luborsky, & O'Brien, 1985). Finally, only BPD can claim to have empirically validated disorder-specific therapies that hasten the remission of its symptoms (criteria) and decrease treatment utilization.

Because the BPD diagnosis is greatly underutilized (Zimmerman et al., 1999, 2010), and because most clinicians lack training in treating BPD and do not like working with them (Shanks, Pfohl, Blum, & Black, in press); changes in DSM-V should encourage its use by making the diagnosis more visible and accessible. In DSM-III and IV, placing BPD under the parent class of PDs may have encouraged excessive use of the residual category, PDNOS (Blashfield & Intoccia, 2000). Though in the current DSM-V proposal all the personality disorders are placed in Axis I, BPD remains buried behind an even more complex and lengthy filter that is proposed to be classified as having a personality disorder (see Shedler et al., 2010). Rather than encouraging and stimulating more appropriate utilization of the BPD diagnosis, the DSM-V work group's proposed revisions may *de facto* defeat the hope of making the BPD diagnosis harder to overlook by placing it on Axis I (Gunderson, 2009; New et al., 2008; Paris, 2007). BPD needs to be featured as a primary target for treatment. Such prominence will serve as a stimulus for training programs to include this disorder in their curricula, help support fair reimbursement policies by third party payors, and help this diagnosis gain parity.

The reasons for giving BPD a hierarchical relationship to other personality disorders are impressive, but how to do this is unclear. If it were to move to the mood disorders, it would benefit utilization and decrease stigma. However, this change would magnify the current overuse of medication and underutilization and training in psychosocial interventions. Moreover, it would bury the role of interpersonal hypersensitivity. If it was identified as an Impulse Control disorder, this too might help utilization and stigma, but would not reflect any of the major theories of etiology or therapy. It might be least prejudicial for BPD to be considered as an anxiety disorder. This could be justified conceptually, but it is not reflected well in the criteria. Perhaps the best option is to have it stand alone (or with ASPD) on Axis I with all other PDs remaining on Axis II.

CONCLUSIONS

The changes in BPD's diagnosis proposed here are offered with recognition that the proposed move towards dimensions and a scientific personology endorsed by Drs. Kupfer and Regier (Regier, Narrow, Kuhl, & Kupfer, 2009) should one day supersede and reorganize our classification. At the present, however, as per the letter from Drs. Spitzer and Frances (letter to Board of Trustees, American Psychiatric Association, July 6, 2009), a huge gap remains between the still preliminary development of that scientific nosology and its application to clinical practice.

Changes in the BPD criteria and their ratings that are proposed integrate levels of severity and have both an empirical and conceptual rationale that do not radically effect their meaning. The changes should make the criteria more specific, and thereby diminish both BPD's overlaps and its heterogeneity. The change proposed for the diagnostic algorithm moves from number of criteria to number of phenotypes; i.e., Interpersonal Hypersensitivity, Affective/Emotional Dysregulation, Behavioral Dyscontrol, and Disturbed Self. This diminishes heterogeneity, mirrors knowledge about BPD's underlying structure, and retains continuity with prior research. The specific algorithm for establishing the BPD diagnosis will require empirical exploration. The final change proposed, that is, assigning BPD a hierarchical priority to other personality disorders (with the exception, perhaps, of ASPD) will unquestionably improve utilization, awareness, and stimulate training and research.

This proposal has developed with the input of many researchers and clinicians who have made contributions to BPD (see Appendix). Each may find aspects to disagree with, but all are seriously dissatisfied with the DSM-V proposal and all support the mode from which this proposal evolved: transparent, interactive, science-based, and having face-value clinical utility to those who treat patients. The proposed changes invite empirical assessments, most significantly how the changes will effect coverage, but they all honor the basic integrity of the current diagnosis, the clinical and research base that has developed from it, and adds impetus to the growing awareness and clinical utility of the borderline diagnosis.

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APPENDIX

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TABLE 1

Proposed Changes in *DSM-IV* BPD Criteria: Four Sectors

(Proposed additions are **[bracketed]**; proposed omissions are; relevant studies are cited).

Interpersonal Hypersensitivity

(1) a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation **[i.e., distrustful perceptions of others as bad, malevolent]** (Bhar, Brown, & Beck, 2008; Butler, Brown, Beck, & Grisham, 2002; Drapeau & Perry, 2009; Domes et al., 2008; Donegan et al., 2003; King-Casas et al., 2008; Lynch, Chapman, Rosenthal, Kuo, & Linehan, 2006; Shedler & Westen, 2004; Unoka, Seresm, Aspan, Bodi, & Keri, 2009; Wagner & Linehan, 1999) **[and between overinvolvement and withdrawal]** (Conklin & Westen, 2005; Drapeau & Perry, 2009; Meyer et al., 2001)]

(2) **[anxious preoccupation with]** (Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004; Butler et al., 2002; Choi-Kain, Fitzmaurice, Zanarini, Laverdiere, & Gunderson, 2009; Morse et al., 2009; Scott, Levy, & Pincus, 2009) real or imagined abandonment **[and rejection]** (Butler et al., 2002; Stiglmayer et al., 2005; Choi-Kain, Hudson, Zanarini, & Gunderson, 2010; Stanley & Siever, 2010; Ziegler-Hill & Abraham, 2006)]

(3) chronic feeling of emptiness (Klonsky, 2008) (no change)

Affective/Emotional Dysregulation

(4) **negative emotionality (chronic dysphoria)** (Kuo & Linehan, 2009; Livesley, 2008; Nica & Links, 2009; Shedler & Westen, 2004; Zanarini et al., 2007), **with sudden shifts from** irritability or anxiety **[to depression (but not to euphoria)]** (Koenigsberg et al., 2002; Trull et al., 2008)] (usually lasting a few hours and only rarely more than a few days)

(5) inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, **[violent outbursts]**) (Koenigsberg et al., 2002; Morse et al., 2009; Shedler & Westen, 2004) (minor change)

Behavioral Dyscontrol

(6) impulsivity in at least two areas that are potentially self-damaging, e.g. [omit], sex, substance abuse, reckless driving, binge eating (minimal change)

(7) recurrent suicidal behavior, gestures, or threats, or **deliberate self-harming behavior** (minimal change)

Disturbed Self

(8) markedly and persistently unstable self-image or sense of self **[including perceptions of self as bad]** (Conklin & Westen, 2005; Gunderson & Links, 2008; Zanarini et al., 1998, 2008) (minor change)

(9) dissociative **[states of mind]** **[i.e., perceives self or the world as disconnected, unreal]** (Koenigsberg et al., 2009; Stiglmayer, Shapiro, Stieglitz, Limberger, & Bohus, 2001; Stiglmayer et al., 2008) with episodic stress-related paranoid ideation (minor change)

[*Criterion Rating* Rate the extent to which each criterion is descriptive of the patient (adapted from Shedler & Westen, 2004; Trull et al., in press):

0 = Not

1 = Mild

2 = Moderate

3 = Strong]