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Review

# The Fate of Borderline Pathology in Dimensional Classification Systems: A Narrative Review

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## Highlights

### What are the main findings?

- Borderline symptoms do not form a distinct factor in structural analyses but consistently load on a general personality disorder factor, functioning as markers of overall severity of personality dysfunction rather than defining a separate diagnostic entity.
- The regional concept of the borderline level of personality functioning converges with the Level of Personality Functioning (Criterion A) in DSM-5 AMPD and the severity continuum in ICD-11, demonstrating that borderline pathology represents a dimension of structural vulnerability across personality disorders.

### What are the implications of the main findings?

- Retaining borderline pathology as a recognizable clinical pattern within dimensional classification systems preserves continuity with established treatment and research traditions while providing clinicians with a meaningful marker of moderate-to-severe personality dysfunction.
- Dimensional assessment of personality disorders should integrate both severity rating and trait-based description, with borderline features serving as clinically useful indicators of regression proneness and the need for specialized psychotherapeutic intervention.

## Abstract

Recent revisions of personality disorders (PD) classifications have moved from categorical diagnoses toward dimensional models, raising renewed questions about the nosological status and clinical utility of borderline personality disorder (BPD). This narrative review traces the development of the borderline construct from early descriptions of patients positioned between neurosis and psychosis, through its theoretical consolidation within the concept of borderline personality organization, to the operationalization of BPD in DSM III and subsequent diagnostic revisions. A central section summarizes contemporary controversies regarding the validity and utility of BPD features. Arguments for abandoning the diagnosis emphasize the absence of a distinct borderline factor in factor analytic studies, the tendency of the construct to capture fluctuating symptoms and patterns of behaviour rather than stable maladaptive personality traits, the stigmatizing and non selective use of the label, and the lack of disorder specific treatment approaches. In contrast, converging evidence supports the view that core borderline symptoms frequently function as markers of general PD pathology and of the severity of impairments in self and interpersonal functioning. The paper integrates the regional tradition of the borderline level of personality functioning, conceptualizing borderline pathology as a dynamic dimension of dysfunction with potential transient regressions, and links this concept to the Level of Personality Functioning (LPF, Criterion A) within the DSM 5 Alternative Model for Personality Disorders (AMPD). Retaining borderline pathology as a dimension

may support contemporary PD assessment by offering a clinically recognizable marker of overall dysfunction, a guide for rating severity, an indicator of personality structure and need for psychotherapy, without disrupting continuity with an extensive clinical and research tradition.

**Keywords:** borderline pathology; borderline personality organization; borderline personality disorder; borderline pattern descriptor; level of personality functioning; ICD-11 classification; severity; general factor of personality disorder

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## 1. Introduction: Nosological History

Dimensional reforms of personality disorders (PD) classifications have challenged the status of long standing categorical diagnoses, with BPD becoming a focal point of debate. This review provides a brief nosological history of the borderline construct, summarizes key arguments for and against retaining BPD as a diagnostic entity, and proposes a synthesis that integrates regional models of borderline level of personality functioning with contemporary dimensional frameworks.

Within official diagnostic classifications, the term borderline first appeared in the diagnosis of BPD, introduced as a distinct entity in DSM-III [1980], largely through the work of Spitzer and colleagues, building on earlier research by Gunderson and Kernberg's concept of borderline personality organization (BPO) [1]. Spitzer defined criteria for separating a class of severely disturbed patients into two groups, borderline and schizotypal personality disorder and early findings suggested that BPD could be differentiated from schizophrenia, depression, and other PDs by specific identity disturbances and interpersonal problems [2]. In ICD systems the construct appeared in ICD-10 within emotionally unstable PD, with borderline and impulsive subtypes [3]. In DSM-5, the categorical model was retained, while the AMPD introduced a hybrid approach that includes a limited set of six PD categories, Antisocial, Avoidant, Borderline, Narcissistic, Obsessive-Compulsive, and Schizotypal PD, combined with a dimensional assessment of personality functioning and pathological traits. In ICD-11 categorical PD diagnoses were removed and replaced by a severity-based model with trait domain qualifiers, Negative Affectivity, Detachment, Dissociality, Disinhibition, and Anankastia, alongside the optional Borderline Pattern qualifier, which was included to preserve continuity with prior clinical and research traditions [4].

Looking at the period preceding formal classification systems, the term borderline first appeared in 1938, in Stern's description of the "borderline group of neurosis, referring to patients who could not be clearly assigned to either the schizophrenic or the psychoneurotic group [5]. Subsequent labels such as pseudoneurotic schizophrenia [6], borderline schizophrenia [2], and psychotic character [7] placed the phenomenon within the psychoses because of a presumed potential for psychotic decompensation, the syndrome remained poorly delineated, without a stable or widely shared definition.

The nosological history of the construct could be traced through the historical framework proposed by Gunderson who outlines multiple phases in the evolution of the borderline concept, reflecting shifts in theory, research focus, and clinical attitudes and organizes into six periods [8]. In the first period, before 1970, borderline was used to describe patients viewed as being between neurosis and schizophrenia, with Kernberg's model gradually influencing psychiatric discourse, sometimes dismissed within psychiatry as a psychoanalytic colloquialism [9]. A key advance in understanding borderline pathology nevertheless emerged precisely from Kernberg's concept of Borderline Personality Organisation (BPO) [10], characterized by identity diffusion, primitive defenses centered on splitting (distinguishing it from neurotic organization), and preserved reality testing (distinguishing it from psychotic organization) [11]. Splitting is treated as a central clinical marker and a putative driver of cognitive, emotional, and behavioural manifestations: contradictory representations of self and object, emotional lability and impulsivity, and chaotic interpersonal relationships marked by primitive ambivalence and instability [12]. Grinker classified patients along a continuum from neurotic to psychotic and empirically identified four core elements of the

borderline syndrome: anger, disturbed relationships, identity problems, and depressive loneliness [13]. In the second period (1970s--1980s), the focus shifted from personality organization toward a syndrome perspective, while Akiskal observed that "borderline is an adjective in search of a noun"[14]. During that period, BPD entered DSM-III and ceased to be conceptualized as a variant of schizophrenia, although speculations about atypical depression began to appear [14]. The third period [1980–1990] is marked by Vaillant's remark that "it is wise never to call a patient borderline"[15] reflecting the accumulation of knowledge about countertransference difficulties in treating this group, while research simultaneously supported the consistency of the syndrome, its distinction from schizophrenia and depression, and a high rate of abuse histories (around 70%). In the fourth period (1990s), neurobiological models of affective dysregulation emerged and the differential diagnosis broadened to include the bipolar II spectrum, raising the question of whether a patient could be considered borderline if they responded to medication, while the development of specialized psychotherapies partly supported the construct's validity. In the fifth period (after 2000), BPD was framed as a "heritable brain disease with a good prognosis" [16]. The sixth period (from 2010 onward) can be summarized by the idea that "borderline patients have long been to psychiatry what psychiatry has been to medicine," highlighting their neglected public health significance [8].

From 2010 onward, concerted efforts began toward the abolition of categorical models and the dimensionalization of PDs. These efforts were built upon earlier foundational work, including factor-analytic studies and critical reviews of categorical classification [17]. This period was marked by the acceptance of the Alternative Model for Personality Disorders (AMPD) as a proposal for further research in DSM-5 Section III, in which BPD was retained as one of six personality disorder categories alongside a dimensional trait framework, and by the work of the WHO ICD-11 Working Group on the Classification of Personality Disorders, whose first formal proposal for reclassification was presented by Tyrer et al [18]. This process culminated in in 2022 with the adoption of a fully dimensional classification of personality disorders in ICD-11, when all categorical diagnoses were abolished. However, a Borderline Pattern qualifier was preserved [19]. This period was also characterized by a convergence between personality and PD research, aligning dimensional models of personality pathology with the Five-Factor Model [20]. Following ICD-11 adoption, some Working Group members have argued that the Borderline Pattern qualifier is redundant, fully captured by severity and the trait domains of Negative Affectivity, Disinhibition, and Dissociality [21–23]. Authors opposing abandonment of the construct argue that doing so would constitute an unacceptable rupture with what is likely the most extensive documented clinical and research literature in the PD field [1,24–27].

## 2. Validity and Clinical Utility of the Borderline Construct

A research group associated with Peter Tyrer is among the most prominent advocates for excluding BPD from diagnostic classification systems, claiming that it lacks scientific validity. In ICD-11, the retention of a borderline pattern descriptor, notably not a formal diagnosis, is described by this group as a result of political compromise [19]. These authors cite three broad reasons for excluding BPD, and the term itself, from classification [22].

First, PDs are linked to maladaptive traits that are relatively stable over time, whereas the features used to describe BPD are not tied to a specific personality trait but rather to fluctuating symptoms and behaviours. As Mulder and Tyrer put it, BPD criteria are "not longstanding personality dispositions, but oscillating symptoms and behaviour." A constant and undisputed diagnostic aspect of true personality disturbance is the presence of traits that are generally stable over time and, when disturbance becomes disorder, maladaptive. The widely fluctuating features of emotional instability do not belong in this paradigm [22].

Second, factor-analytic studies have not isolated a distinct borderline factor. The evidence suggests that BPD symptoms form a coherent syndrome when examined in isolation but "disappear" into a general factor when criteria from other PDs are included. Sharp and colleagues identified a strong latent factor underlying the nine DSM BPD criteria. However, when symptoms of other PDs

were incorporated into a model that also included a general factor, BPD items loaded entirely onto the general factor, and no specific borderline factor remained [28].

Third, beyond issues of diagnostic validity, these authors argue that the diagnosis lacks clinical utility: it is overly broad, applied non-selectively, and thus contributes to confusion and stigma, particularly among health professionals. They further question its usefulness on the grounds that it provides little guidance regarding specific treatment. In this view, specialized psychotherapies for BPD are better understood as transdiagnostic approaches, and no psychopharmacological treatment has demonstrated consistent disorder-specific efficacy. Indeed, the authors contend that the only accurate aspect of the diagnosis is its name, as it inadvertently points to the construct's nonspecificity [22]. This raises a legitimate question: how should one conceptually organize the large body of clinical and research findings that continue to be recognized in everyday practice as "borderline," findings that do not simply vanish when the diagnostic entity is removed from classification?

Having summarized the main arguments for excluding the borderline construct, we now turn to evidence that may complicate the straightforward conclusion that borderline pathology is merely redundant with severity and trait domains.

### 3. Level of Personality Functioning

Dimensional approaches to PD were developed and advocated for more than three decades within a regional tradition emphasizing the borderline level of personality functioning. Research from this perspective supported the dimensional nature of PD and suggested that many symptoms traditionally viewed as specific to BPD also characterize other PDs. They conceptualized the core personality deficit as a shared dimension (broadly resembling BPO) that cuts across PD categories, which they termed the borderline level of personality functioning [11,29–31].

Within this model, BPD is not treated as a discrete entity but as a level on a continuum between normal and neurotic functioning on one side and psychotic functioning on the other. The concept of continuum implies the absence of rigid boundaries, and functioning is viewed as dynamic rather than fixed, allowing for regressions and progressions across levels. Levels of functioning are described as relatively stable systems whose modulation depends on internal and external factors [12,32]. Borderline phenomena are framed as nonspecific markers of severity and dysfunction across PDs. Individuals with PD predominantly function at a borderline level of personality functioning while differing in trait based types of adaptation (corresponding to trait-based descriptions) [30,33].

A key element of this model is that the borderline level of functioning is conceptualized as a dynamic dimension, making transient regression to this level possible, with accompanying symptoms, most often in the context of affective disorders. This framework provides a conceptual basis for the frequent comorbidity between borderline phenomena and affective disorders [34]. Such transient regression to the borderline level in patients whose primary diagnosis is an affective disorder, regression that remits with treatment of the affective disorder, has been termed pseudo-borderline syndrome [34]. This framework further proposes that emotional dysregulation and constitutionally low tolerance of negative affect characterize all individuals with PD, forming a vicious cycle that may explain the clinical phenomenon of recurrent regression [35].

The two-step diagnostic logic described in the regional tradition, first assessing the level of personality functioning, then specifying the type of trait-based adaptation, aligns closely with contemporary dimensional models. In the AMPD, assessment begins with Criterion A, the Level of Personality Functioning (LPF), before proceeding to Criterion B, pathological trait domains. In the ICD-11, clinical decision-making similarly starts with determining whether a PD is present and, crucially, rating its severity, with trait domain qualifiers added as a secondary step. Both systems rate severity across graded levels that parallel the levels of personality organization described by Kernberg: moderate, severe, and extreme impairment in AMPD, and mild, moderate, and severe PD in ICD-11, correspond to high, middle, and low borderline personality organization respectively. [36,37]. This congruence is now supported by empirical convergence between measures of personality organization and both classification systems [38–40]. In each case, the level of personality

functioning is primary for prognosis and treatment planning, while trait-based description is secondary, an approach that Kernberg's concept of BPO and the regional concept of the level of functioning had articulated decades earlier [30,31,36].

This convergence is consistent with the finding that borderline symptoms consistently load on the general PD factor in bifactor analyses. Sharp has argued that borderline criteria capture precisely what LPF is designed to measure, that is, self and interpersonal functioning, which is why they represent general personality pathology rather than forming a distinct specific factor [28]. Conceptually, the LPF domains of self-functioning (identity and self-direction) and interpersonal functioning (empathy and intimacy) correspond to the self and object representations that psychodynamic models place at the core of personality organization. This suggests that what psychodynamic tradition describes as borderline and what dimensional models measure as impaired personality functioning is the same underlying dysfunction, approached from different theoretical perspectives [41].

#### 4. Discussion

The stance toward borderline personality pathology varied not only between authors who supported or opposed its removal from diagnostic classifications, but also shifted within individual authors over time. Tyrer's trajectory illustrates this evolution. Initially, he argued that BPD and schizotypal PD were unique among PDs, separated from more continuously distributed conditions by a "zone of rarity" a discontinuity in the PD spectrum that warranted their recognition as distinct clinical entities. Subsequently, he proposed reconceptualizing the condition as one of persistently unstable mood, coining the term "fluxithymia," and recommended its reclassification within the affective disorders spectrum rather than outright elimination [42]. "Finally, he called for abandoning the construct entirely as "spurious" and "unsupported by science" [22].

The three principal arguments advanced for excluding the borderline construct, that its features represent fluctuating symptoms rather than stable traits, that a distinct borderline factor does not emerge in factor analytic studies, and that the diagnosis lacks clinical utility, can each be examined on their own grounds.

The argument that borderline features are fluctuating symptoms rather than stable personality dispositions conflates two distinct levels of analysis. What fluctuates is the symptomatic manifestation, affective storms, impulsive acts, transient psychotic episodes, but what remains stable is the underlying structural vulnerability that makes such fluctuations possible. The capacity for regression to a given level of dysfunction is itself a stable structural characteristic what varies is only the occasion of its activation. This dynamic, in which an enduring vulnerability manifests episodically, is precisely what static trait domain profiles cannot represent. Indeed, regression proneness, one of the most clinically consequential features of borderline pathology and a major source of treatment difficulties, is omitted from the nine DSM-5 BPD criteria and from the ICD-11 borderline pattern specifier alike, and is even less likely to be captured by trait domain descriptors alone. Regression proneness has been empirically demonstrated through projective tests such as the Rorschach and TAT, in which BPD patients show primary process thinking with activation of primitive defenses and disturbed object relations[1]. The concept of „pseudo borderline syndrome" illustrates both this distinction and the broader principle that the level of personality functioning must be understood as a dynamic dimension. Individuals whose baseline functioning is neurotic may transiently regress to the borderline level under the pressure of affective disorder, with borderline symptoms remitting once the affective episode is treated [34].

The second argument, that a distinct borderline factor fails to emerge in factor analytic studies, is largely accurate but does not carry the implications its proponents assume. The absence of a specific borderline factor does not mean that borderline symptoms vanish from factorial solutions; rather, it reveals something fundamental about the nature of borderline pathology itself. Moreover, this claim is contradicted by the authors' earlier findings. Although Mulder and Tyrer [2023] assert that such a factor never appears, a "borderline factor" did in fact emerge in a validation study of the five proposed

ICD-11 trait domains conducted by the same group [43]. This factor predominantly comprised items from borderline, narcissistic, and histrionic PDs, aggregating a broad set of symptoms that merged dissociality and disinhibition while also correlating with negative affectivity and an antisocial factor. The authors themselves interpreted this factor as reflecting the heterogeneity of BPD and speculated that it might represent a general PD factor, as it included a wide spectrum of pathological traits and dysfunctional behavioural patterns across multiple PD types rather than being uniquely specific to BPD [43].

More broadly, the factor analytic literature reveals a consistent three factor structure of PD pathology: an externalizing factor, an internalizing factor, and a detachment factor. Of these, the externalizing dimension is the most robust and clearly delineated. Borderline symptoms do not disappear in these solutions but recur in varying configurations, loading most frequently on the externalizing dimension alongside histrionic, narcissistic, antisocial, and paranoid symptoms [44–46]. Beyond the externalizing factor, borderline symptoms also load on an internalizing factor, where they co occur with avoidant and dependent traits. In our own study revalidating the ICD-11 proposal using exploratory factor analysis, borderline symptoms did not disperse across other factors but instead formed a coherent factor together with avoidant and dependent features, which we termed a borderline internalizing factor [47]. When modelled within the broader structure of common mental disorders, BPD consistently loads on both the distress subfactor of the internalizing dimension and the externalizing dimension, a dual placement now codified in the HiTOP model. As the HiTOP consortium has noted, BPD is "best considered an indicator of both internalizing and, to a lesser degree, the general externalizing superspectrum, likely with different components of the disorder being related to these two spectra." [48].

Evidence from bifactor modeling offers a way to integrate these findings by clarifying why borderline features do not form a separate factor yet remain omnipresent across factorial solutions. Sharp and colleagues reported that borderline criteria form a strong latent factor when examined in isolation, but load almost entirely on a general PD factor once criteria from other PDs are included, suggesting that borderline symptoms index general impairment in personality functioning rather than a specific syndrome [28]. A large bifactor study by Jahng and colleagues, based on the U.S. National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), further supports this view [49]. They identified a strong general factor spanning ten PD diagnoses that accounted for a substantial proportion of the comorbidity between PD and substance use disorders. After partialling out this general factor, a residual factor linked to Cluster B PDs remained, plausibly reflecting impulsivity or disinhibition, which showed an independent positive association with all substance dependence diagnoses across both sexes and age groups. With the residual factor controlled, the PDs loading most strongly on the general factor were avoidant, dependent, paranoid, and schizoid, conditions characterized by marked interpersonal dysfunction [49]. Borderline pathology thus operates simultaneously at two levels: it saturates the general factor that indexes overall severity of personality dysfunction, and its impulsive action components contribute to a specific externalizing residual. Similar results emerged from our study of a clinical PD sample, in which canonical analysis of covariance revealed a broad general PD factor predominantly driven by borderline, avoidant, dependent, detached, and anankastic traits and linked to high neuroticism and low conscientiousness, and a separate externalizing factor linking narcissistic, histrionic, and antisocial traits to high extraversion, high openness, and low agreeableness [50].

This cross spectral placement receives independent support from research on higher order personality metatraits. Building on Digman's observation that the Big Five factors cluster into two higher order metatraits, Alpha (stability; N-, A+, C+) and Beta (plasticity; E+, O+), Strus and colleagues developed the Circumplex of Personality Metatraits (CPM), an octant model that maps both normal personality and personality pathology onto a common circular dimensional space [51,52]. In this model, internalizing PDs cluster around the maladaptive pole Gamma minus (N+, E-, O-, A-, C-), representing general dysfunction and disharmony, while externalizing PDs cluster around Delta minus (N+, E+, O+, A-, C-), representing disinhibition and sensation seeking.

Borderline PD does not fall neatly into either cluster but occupies an intermediate position at the interface between the two, converging with the cross spectral placement consistently observed in factor analytic and HiTOP studies [53,54].

The third argument concerns clinical utility: the borderline pattern is said to be applied non selectively, to offer limited clinically useful information, to generate confusion, and to be a source of stigma, and should accordingly be abandoned [21–23]. Given that ICD-11 includes a severity continuum and clearly defined trait domains, these authors argue that the borderline pattern adds no additional diagnostic information and that severity plus domain configuration can replace the descriptor entirely.

However, the empirical findings invoked in support of redundancy can equally be read as supporting a different conclusion. In a study published shortly before ICD-11 adoption, the same group examined overlap between BPD symptoms and total PD symptoms and found a strong association between hallmark borderline features, unstable identity and fear of abandonment, and overall PD severity [21]. The more severe the PD, the more likely borderline symptoms are to manifest. Rather than demonstrating that the borderline pattern is dispensable, this finding is consistent with the interpretation that it functions as a clinically recognizable marker of moderate to severe PD, precisely the role proposed by the regional concept of the borderline level of personality functioning [30] and by Sharp's argument that borderline symptoms capture the general PD factor because they index impairments in self and interpersonal functioning [28,55,56].

The claim that the borderline pattern is fully captured by severity plus three trait domains rests on a compositional assumption: if a construct can be decomposed into its elements, the elements render the construct unnecessary. Several lines of evidence challenge this assumption. First, the decomposition is incomplete. Gutiérrez et al. found that ICD-11 trait domains accounted for only 65% of the variance in validated BPD measures, whereas the Borderline Pattern Scale explained 74%, a gap suggesting that domain level descriptors do not fully capture what the borderline pattern indexes [57]. Second, the same configuration of domains can describe clinically distinct presentations: Negative Affectivity combined with Disinhibition and Dissociality may characterize a patient with identity diffusion, affective instability, and chronic self harm, but equally one with substance dependence, anger dyscontrol, and intermittent antisocial behavior, presentations that differ in risk profile, treatment needs, and prognosis [25]. Third, borderline symptoms show incremental validity over trait dimensions in predicting functional outcomes: Morey et al. demonstrated that BPD features and Five Factor Model traits each predicted prospective functioning beyond what the other captured, with impulse action features such as suicidality and substance misuse showing relative independence from neuroticism. Descriptive overlap does not entail predictive redundancy [58].

## 5. Conclusions

This review has argued that borderline pathology, far from being a spurious category to be 'buried,' represents a clinically meaningful dimension that indexes the general factor of personality dysfunction - a dimension now formally recognized as Level of Personality Functioning in AMPD and as the severity continuum in ICD-11. The convergence between the regional concept of the borderline level of functioning and contemporary dimensional frameworks suggests that what has historically been called 'borderline' is not a discrete disorder but a marker of severity and structural vulnerability, capturing impairments in identity, interpersonal functioning, and affect regulation that cut across PD types. That this construct 'dissolves' into a general factor in structural analyses does not diminish its clinical utility. On the contrary, it reveals its nature as a reliable indicator of core personality dysfunction. Retaining borderline pathology as a recognizable clinical pattern within dimensional systems preserves continuity with an extensive treatment and research literature, provides clinicians with a configuration that specifies what severity looks like in practice, and maintains the dynamic perspective in which the capacity for regression is itself a stable structural characteristic, even when its manifestations are episodic.

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## References

1. Leichsenring, F.; Fonagy, P.; Heim, N.; Kernberg, O.F.; Leweke, F.; Luyten, P.; Milrod, B.; Steinert, C.; Strehlow, J.; Tan, Y.M.; et al. Borderline personality disorder: A comprehensive review of diagnosis and clinical presentation, etiology, treatment, and current controversies. *World Psychiatry* **2024**, *23*, 4–25. <https://doi.org/10.1002/wps.21156>
2. Spitzer, R.L.; Endicott, J.; Gibbon, M. Crossing the border into borderline personality and borderline schizophrenia: The development of criteria. *Arch. Gen. Psychiatry* **1979**, *36*, 17–24. PMID: 760694 DOI: 10.1001/archpsyc.1979.01780010023001
3. Lai, C.M.; Leung, F.; You, J.; Cheung, F. Are DSM-IV-TR Borderline Personality Disorder, ICD-10 Emotionally Unstable Personality Disorder, and CCMD-III Impulsive Personality Disorder Analogous Diagnostic Categories Across Psychiatric Nomenclatures? *J. Pers. Disord.* **2012**, *26*, 551–567. <https://doi.org/10.1521/pedi.2012.26.4.551>
4. Bach, B.; Kramer, U.; Doering, S.; di Giacomo, E.; Hutsebaut, J.; Kaera, A.; De Panfilis, C.; Schmahl, C.; Bekaert, P.; Kongerslev, M.T.; et al. The ICD-11 classification of personality disorders: A European perspective on challenges and opportunities. *Borderline Personal. Disord. Emot. Dysregul.* **2022**, *9*, 12. <https://doi.org/10.1186/s40479-022-00182-0>
5. Stern, A. Psychoanalytic investigation of and therapy in the border line group of neuroses. *Psychoanal. Q.* **1938**, *7*, 467–489. <https://doi.org/10.1080/21674086.1938.11925367>
6. Hoch, P.; Polatin, P. Pseudoneurotic forms of schizophrenia. *Psychiatr. Q.* **1949**, *23*, 248–276. <https://doi.org/10.1007/BF01563119>
7. Frosch, J. Psychoanalytic considerations of the psychotic character. *J. Am. Psychoanal. Assoc.* **1970**, *18*, 24–50. <https://doi.org/10.1177/000306517001800102>
8. Gunderson, J.G. Borderline personality disorder: Ontogeny of a diagnosis. *Am. J. Psychiatry* **2009**, *166*, 530–539. <https://doi.org/10.1176/appi.ajp.2009.08121825>
9. Stone, M.H. Borderline personality disorder: History of the concept. *Med. Psychiatry* **2005**, *31*, 1. <https://doi.org/10.1201/b14134-4>
10. Kernberg, O. Borderline personality organization. *J. Am. Psychoanal. Assoc.* **1967**, *15*, 641–685. <https://doi.org/10.1177/000306516701500309>
11. Lečić, T.D. Modern concept of personality disorders: The past rediscovered. *Psihijatrija Danas* **2004**, *36*, 243–260.
12. Divac, J.M.; Tatar, R.S.; Lečić, T.D.M.; Manojlović, J. Borderline level of functioning: Manifestations of splitting in transactions with personality disorders. *Psihijatrija Danas* **1995**, *27*, 9–26.
13. Grinker, R.R., Sr.; Webble, B.; Drye, R. *The Borderline Syndrome. A Behavioral Study of Ego-Functions*; Basic Books: New York, NY, USA, 1968.
14. Akiskal, H.S.; Chen, S.E.; Davis, G.C.; Puzantian, V.R.; Kashgarian, M.; Bolinger, J.M. Borderline: An adjective in search of a noun. *J. Clin. Psychiatry* **1985**, *46*, 41–48.
15. Vaillant, G.E. The beginning of wisdom is never calling a patient a borderline; or, the clinical management of immature defenses in the treatment of individuals with personality disorders. *J. Psychother. Pract. Res.* **1992**, *1*, 117.

16. Torgersen, S.; Lygren, S.; Øien, P.A.; Skre, I.; Onstad, S.; Edvardsen, J.; Tambs, K.; Kringlen, E. A twin study of personality disorders. In *Personality and Personality Disorders*; Routledge: London, UK, 2013; pp. 244–253. <https://doi.org/10.1053/comp.2000.16560>
17. Widiger, T.A.; Trull, T.J. Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *Am. Psychol.* **2007**, *62*, 71. <https://doi.org/10.1037/0003-066X.62.2.71>
18. Tyrer, P.; Crawford, M.; Mulder, R.; Blashfield, R.; Farnam, A.; Fossati, A.; Kim, Y.-R.; Koldobsky, N.; Lecic-Tosevski, D.; Ndeti, D.; et al. The rationale for the reclassification of personality disorder in the 11th revision of the International Classification of Diseases (ICD-11). *Pers. Ment. Health* **2011**, *5*, 246–259. <https://doi.org/10.1002/pmh.190>
19. Tyrer, P.; Mulder, R.; Kim, Y.-R.; Crawford, M.J. The development of the ICD-11 classification of personality disorders: An amalgam of science, pragmatism, and politics. *Annu. Rev. Clin. Psychol.* **2019**, *15*, 481–502. <https://doi.org/10.1146/annurev-clinpsy-050718-095736>
20. Widiger, T.A.; Costa, P.T. Integrating normal and abnormal personality structure: The five-factor model. *J. Pers.* **2012**, *80*, 1471–1506. <https://doi.org/10.1111/j.1467-6494.2012.00776.x>
21. Mulder, R.T.; Horwood, L.J.; Tyrer, P. The borderline pattern descriptor in the International Classification of Diseases, 11th Revision: A redundant addition to classification. *Aust. N. Z. J. Psychiatry* **2020**, *54*, 1095–1100. <https://doi.org/10.1177/0004867420951608>
22. Mulder, R.; Tyrer, P. Borderline personality disorder: A spurious condition unsupported by science that should be abandoned. *J. R. Soc. Med.* **2023**, *116*, 148–150. <https://doi.org/10.1177/01410768231164780>
23. Tyrer, P.J.; Mulder, R.T. The problem with borderline personality disorder. *World Psychiatry* **2024**, *23*, 445–446. <https://doi.org/10.1002/wps.21249>
24. Bateman, A.W. Throwing the baby out with the bathwater? *Pers. Ment. Health* **2011**, *5*, 274–280. <https://doi.org/10.1002/pmh.184>
25. Herpertz, S.C.; Huprich, S.K.; Bohus, M.; Chanen, A.; Goodman, M.; Mehlum, L.; Moran, P.; Newton-Howes, G.; Scott, L.; Sharp, C. The challenge of transforming the diagnostic system of personality disorders. *J. Pers. Disord.* **2017**, *31*, 577–589. [https://doi.org/10.1521/pedi\\_2017\\_31\\_338](https://doi.org/10.1521/pedi_2017_31_338)
26. Sharp, C.; Oldham, J. Nature and assessment of personality pathology and diagnosis. *Am. J. Psychother.* **2023**, *76*, 3–8. <https://doi.org/10.1176/appi.psychotherapy.20220016>
27. Ruffalo, M.L. The validity of borderline personality disorder: Robins and Guze applied. *BJPsych Bull.* **2026**, 1–3. <https://doi.org/10.1192/bjb.2025.10206>
28. Sharp, C.; Wright, A.G.; Fowler, J.C.; Frueh, B.C.; Allen, J.G.; Oldham, J.; Clark, L.A. The structure of personality pathology: Both general ('g') and specific ('s') factors? *J. Abnorm. Psychol.* **2015**, *124*, 387. <https://doi.org/10.1037/abn0000033>
29. Svrakic, D.; Divac-Jovanovic, M. Personality Disorders: Model for Conceptual Approach and Classification: Part II: Proposed Classification. *Am. J. Psychother.* **1994**, *48*, 562–580. <https://doi.org/10.1176/appi.psychotherapy.1994.48.4.562>
30. Divac-Jovanovic, M.; Svrakic, D.; Lecic-Tosevski, D. Personality disorders: Model for conceptual approach and classification: Part I: General model. *Am. J. Psychother.* **1993**, *47*, 558–571. <https://doi.org/10.1176/appi.psychotherapy.1993.47.4.558>
31. Lecic-Tosevski, D. Description of specific personality disorders. In *New Oxford Textbook of Psychiatry*; Gelder, M.G., Andreasen, N.C., López-Ibor, J.J., Geddes, J.R., Eds.; Oxford University Press: Oxford, UK, 2000; pp. 927–953.
32. Lečić, T.D.M.; Divac, J.M.; Ilić, Z.P.; Stojanović, D.; Draganić, G.S. Personality disorders in DSM and ICD classifications—'A tale of two cities'. *Psihijatrija Danas* **1996**, *28*, 445–471.
33. Lečić-Toševski, D.; Divac-Jovanović, M. Modifikovana psihodinamska psihoterapija poremećaja ličnosti—Dileme i izazovi. *Psihijatrija Danas* **2011**, *43*, 65–82.
34. Lecic-Tosevski, D.; Divac-Jovanovic, M. Borderline personality disorder and depression. *Psychiatriki* **1995**, *6*, 154–160.
35. Lecic-Tosevski, D.; Divac-Jovanovic, M. Effects of dysthymia on personality assessment. *Eur. Psychiatry* **1996**, *11*, 244–248. [https://doi.org/10.1016/0924-9338\(96\)82330-3](https://doi.org/10.1016/0924-9338(96)82330-3)

36. Kernberg, O.F.; Caligor, E. A psychoanalytic theory of personality disorders. In *Major Theories of Personality Disorder*, 2nd ed.; Guilford Press: New York, NY, USA, 2005; pp. 114–156.
37. Hörz-Sagstetter, S.; Ohse, L.; Kampe, L. Three Dimensional Approaches to Personality Disorders: A Review on Personality Functioning, Personality Structure, and Personality Organization. *Curr. Psychiatry Rep.* **2021**, *23*, 95. <https://doi.org/10.1007/s11920-021-01250-y>
38. Kampe, L.; Zimmermann, J.; Bender, D.; Caligor, E.; Borowski, A.-L.; Ehrental, J.C.; Benecke, C.; Goth, K.; Hennig-Fast, K.; Buchheim, A.; et al. Comparison of the structured DSM-5 clinical interview for the Level of Personality Functioning Scale with the Structured Interview of Personality Organization. *J. Pers. Assess.* **2018**, *100*, 642–649. <https://doi.org/10.1080/00223891.2018.1489257>
39. Nazari, A.; Huprich, S.K.; Hemmati, A.; Rezaei, F. The construct validity of the ICD-11 severity of personality dysfunction under scrutiny of object-relations theory. *Front. Psychiatry* **2021**, *12*, 648427. <https://doi.org/10.3389/fpsy.2021.648427>
40. Ohse, L.; Kerber, A.; Zimmermann, J.; Kampe, L.; Mohr, J.; Busch, O.; Bender, D.S.; Caligor, E. Empirical convergence between Kernberg's model of personality organization and the alternative DSM-5 model for personality disorders. *Pers. Ment. Health* **2025**, *19*, e1641. <https://doi.org/10.1002/pmh.1641>
41. Sharp, C. Current trends in BPD research as indicative of a broader sea-change in psychiatric nosology. *Pers. Disord. Theory Res. Treat.* **2016**, *7*, 334. <https://doi.org/10.1037/per0000199>
42. Tyrer, P. Why borderline personality disorder is neither borderline nor a personality disorder. *Pers. Ment. Health* **2009**, *3*, 86–95. <https://doi.org/10.1002/pmh.78>
43. Mulder, R.T.; Horwood, J.; Tyrer, P.; Carter, J.; Joyce, P.R. Validating the proposed ICD-11 domains. *Pers. Ment. Health* **2016**, *10*, 84–95. <https://doi.org/10.1002/pmh.1336>
44. Dowson, J.; Berrios, G. Factor structure of DSM-III-R personality disorders shown by self-report questionnaire: Implications for classifying and assessing personality disorders. *Acta Psychiatr. Scand.* **1991**, *84*, 555–560. <https://doi.org/10.1111/j.1600-0447.1991.tb03194.x>
45. O'Boyle, M. DSM-III-R and Eysenck personality measures among patients in a substance abuse programme. *Pers. Individ. Differ.* **1995**, *18*, 561–565. [https://doi.org/10.1016/0191-8869\(94\)00178-U](https://doi.org/10.1016/0191-8869(94)00178-U)
46. Livesley, W.J.; Jang, K.L.; Vernon, P.A. Phenotypic and genetic structure of traits delineating personality disorder. *Arch. Gen. Psychiatry* **1998**, *55*, 941–948. <https://doi.org/10.1001/archpsyc.55.10.941>
47. Pesic, D.; Lecic-Tosevski, D.; Kalanj, M.; Vukovic, O.; Mitkovic-Voncina, M.; Peljto, A.; Mulder, R. Multiple faces of personality domains: Revalidating the proposed domains. *Psychiatr. Danub.* **2019**, *31*, 182–188. <https://doi.org/10.24869/psy.2019.182>
48. Krueger, R.F.; Hobbs, K.A.; Conway, C.C.; Dick, D.M.; Dretsch, M.N.; Eaton, N.R.; Forbes, M.K.; Forbush, K.T.; Haltigan, J.D.; Kotov, R.; et al. Validity and utility of Hierarchical Taxonomy of Psychopathology (HiTOP): II. Externalizing superspectrum. *World Psychiatry* **2021**, *20*, 171–193. <https://doi.org/10.1002/wps.20844>
49. Jahng, S.; Trull, T.J.; Wood, P.K.; Tragesser, S.L.; Tomko, R.; Grant, J.D.; Bucholz, K.K.; Sher, K.J. Distinguishing general and specific personality disorder features and implications for substance dependence comorbidity. *J. Abnorm. Psychol.* **2011**, *120*, 656–669. <https://doi.org/10.1037/a0023539>
50. Pešić, D.; Lečić-Toševski, D.; Kalanj, M.; Ristić, I.; Vuković, O.; Pejusković, B. Analysis of the Relationship between Higher-Order Factor Structure of Personality Disorders and the Five-Factor Model of Personality. *Brain Sci.* **2023**, *13*, 605. <https://doi.org/10.3390/brainsci13040605>
51. Strus, W.; Ciecuch, J.; Rowiński, T. The Circumplex of Personality Metatraits: A Synthesizing Model of Personality Based on the Big Five. *Rev. Gen. Psychol.* **2014**, *18*, 273–286. <https://doi.org/10.1037/gpr0000017>
52. Strus, W.; Ciecuch, J. Towards a synthesis of personality, temperament, motivation, emotion and mental health models within the Circumplex of Personality Metatraits. *J. Res. Pers.* **2017**, *66*, 70–95. <https://doi.org/10.1016/j.jrp.2016.12.002>
53. Zawadzki, B. The location of personality disorders in the Circumplex of Personality Metatraits. *Rocz. Psychol.* **2017**, *20*, 493–512. <https://doi.org/10.18290/rpsych.2017.20.2-7en>
54. Brud, P.P.; Ciecuch, J. Borderline personality disorder and its facets in the context of personality metatraits and pathological traits. *Pers. Individ. Differ.* **2023**, *202*, 111958. <https://doi.org/10.1016/j.paid.2022.111958>

55. Sharp, C.; Wall, K. DSM-5 Level of Personality Functioning: Refocusing Personality Disorder on What It Means to Be Human. *Annu. Rev. Clin. Psychol.* **2021**, *17*, 313–337. <https://doi.org/10.1146/annurev-clinpsy-081219-105402>
56. Wright, A.G.; Hopwood, C.J.; Skodol, A.E.; Morey, L.C. Longitudinal validation of general and specific structural features of personality pathology. *J. Abnorm. Psychol.* **2016**, *125*, 1120. <https://doi.org/10.1037/abn0000165>
57. Gutiérrez, F.; Aluja, A.; Ruiz Rodríguez, J.; Peri, J.M.; Gárriz, M.; Garcia, L.F.; Sorrel, M.A.; Sureda, B.; Vall, G.; Ferrer, M.; et al. Borderline, where are you? A psychometric approach to the personality domains in the International Classification of Diseases, 11th Revision (ICD-11). *Pers. Disord. Theory Res. Treat.* **2023**, *14*, 355. <https://doi.org/10.1037/per0000592>
58. Morey, L.C.; Hopwood, C.J.; Markowitz, J.C.; Gunderson, J.G.; Grilo, C.M.; McGlashan, T.H.; Shea, M.T.; Yen, S.; Sanislow, C.A.; Ansell, E.B.; et al. Comparison of alternative models for personality disorders, II: 6-, 8- and 10-year follow-up. *Psychol. Med.* **2012**, *42*, 1705–1713. <https://doi.org/10.1017/S0033291711002601>

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