

# The Diagnosis of Borderline Personality Disorder: Problematic but Better Than the Alternatives

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**Objective.** *The purpose of this review is to examine empirical evidence concerning critiques of the diagnosis of borderline personality disorder (BPD): for uncertain validity, and for overlap with other mental disorders.*

**Method.** *A review of the literature on the validity and comorbidity of BPD was conducted.*

**Results.** *Since BPD is a complex multidimensional construct, its validity is inevitably problematic, but no more so than most other psychiatric diagnoses. The comorbidity of BPD is probably an artefact of the current classification system, and there is no convincing evidence that BPD is a variant of an Axis I disorder.*

**Conclusions.** *Although further research should lead to changes in classification, the diagnosis of BPD retains significant clinical utility.*

**Keywords** Borderline personality disorder, Psychiatric diagnosis

## INTRODUCTION

Borderline personality disorder (BPD) is a complex and multidimensional syndrome that includes affective, impulsive, and cognitive phenomena (1,2). For this reason, patients with this disorder can have a wide range of symptoms, associated with high levels of Axis I comorbidity (3). This degree of overlap with other mental disorders has made the BPD diagnosis controversial, leading some theorists (4,5) to reject the construct entirely.

This review will critically examine the main arguments against the validity of BPD, and will assess whether this population of patients can be better described using other diagnoses. The main arguments against the construct have been that: a) “borderline” is a misnomer; b) the category has not been validated; c) the diagnosis lacks precise boundaries; d) cases may be atypical forms of other mental disorders. Each of these issues will be addressed separately. The review will suggest that the critiques of the BPD construct can equally be applied to other mental disorders, and will argue for the clinical utility of making this diagnosis.

## The Term “Borderline”

The very term “borderline” has contributed to debate about the validity of BPD. Akiskal (4), with a flair for language, described the borderline diagnosis as “an adjective in search of a noun.” In fact, there is no border on which a patient can be “borderline.”

The origin of the term derives from the first description of this group of patients by Adolf Stern (6). Noting their resistance to analytic therapy, Stern suggested this form of pathology falls on a “border” between psychosis and neurosis. This formulation was concordant with a concept, then current in psychoanalysis, that all mental disorders lie on a continuum (7). This point of view was never accepted by mainstream psychiatry, and it fell entirely out of favor when neo-Kraepelinian ideas became predominant (8). BPD was not accepted into the American diagnostic classification prior to DSM-III (9); internationally, the diagnosis was only grudgingly included in ICD-10 (10), as a sub-category of “emotionally unstable personality disorder.”

Yet BPD is far from the only misleading diagnostic term in psychiatry. While no one currently believes that schizophrenia reflects its literal meaning, that is, a “split head,” the term retains currency. Renaming diagnoses makes most sense when we have a sufficient understanding of etiology and pathogenesis, so that the new diagnostic term can actually describe the nature

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of a disease process. For example, once the mechanism of peripheral edema related to cardiac illness was understood, the term “dropsy” was replaced by the more descriptive term “congestive heart failure” (CHF). (Even so, CHF remains a syndromal construct, since it only describes pathogenesis.) Until we understand the causes of mental disorders, we accomplish little by simply changing their names.

### *Is the Diagnosis of BPD Valid?*

The definition of BPD that was eventually accepted into DSM-III (9) was largely based on the work of John Gunderson (11), who showed that one can operationalize BPD with observable criteria, using a semi-structured interview with established psychometric properties. The criteria in DSM-IV-TR (12) have remained much the same (except for the addition of a criterion describing cognitive symptoms), and the ICD-10 definition of “emotionally unstable personality disorder, borderline type” (10) is not notably different.

Of course, reliability proves little about validity. Decades ago, Robins and Guze (13) proposed that valid diagnoses in psychiatry need to meet five criteria: 1) clearcut clinical description; 2) laboratory studies; 3) delimitation from other disorders; 4) follow-up studies documenting a characteristic outcome; 5) family prevalence studies. BPD fails on most of these grounds: it overlaps with other mental disorders, lacks a biological profile, and does not show a specific family history (1). At best, BPD is a coherent syndrome with a fairly typical outcome.

Yet if we were to apply the Robins and Guze criteria to most mental disorders, few would be considered valid. Even the most intensely studied categories, such as schizophrenia and bipolar disorder, have serious problems with overlap, lack laboratory tests to identify them, and do not consistently conform to an expected family pattern (14). While the proposal of Robins and Guze was sensible (and remains so today), psychiatry is not advanced enough to apply such stringent criteria, and will not be for decades to come. Thus, while BPD lacks established validity, it is no better and no worse in this respect than other widely accepted diagnoses.

### *Boundaries and Comorbidity*

In addition to its Axis I comorbidity, the BPD diagnosis with overlaps other Axis II disorders (15). This observation has led some researchers (16,17,18) to recommend that personality disorders should be classified dimensionally rather than categorically. In this scenario, patients would be described by profiles of scores on personality traits, rather than being fitted into a specific category. But while this proposal may eventually win the day, it has thus far suffered from lack of agreement as to which dimensional schema are most appropriate.

Yet the problem of comorbidity and overlap is far from unique to the BPD diagnosis. In medicine, similar symptoms can have entirely different causes. Clinical phenomena such as the mood changes seen in depression or bipolar disorder may be no more specific than fever or inflammation. Given the state of research in psychiatry, DSM-III (9) made a practical decision to create a diagnostic system based almost entirely on phenomenology. But reliance on observable data was intended as a provisional solution, pending a future understanding of etiology and pathogenesis. Inevitably, the result was that most mental disorders were syndromal, and lacked unique etiological and pathogenetic mechanisms. Moreover, since the system allows for few hierarchical rules, the classification system for mental disorders in DSM-IV-TR actually encourages comorbidity.

Thus, the comorbidity of BPD, both on Axis I and Axis II, does not invalidate the diagnosis, any more than the comorbidity of major depression. The real problem is our lack of basic knowledge about the nature of the pathology being observed. In psychiatry, the term “comorbidity” does not mean that patients have more than one disease. Internists describe comorbidity between hypertension and arteriosclerosis, but these are two diseases whose etiology and pathogenesis are reasonably well known, so that each interacts to raise the risk for the other. Such concepts are not yet applicable to psychiatry, where diseases are syndromal and poorly understood.

### *Is BPD a Variant of an Axis I Disorder?*

Given the presence of many egodystonic symptoms, BPD does not seem to correspond to the classic concept of personality disorders as dysfunctional (but egosyntonic) personality traits. Might BPD therefore be a variant of other mental disorders on Axis I? Over the years, it has been suggested that its pathology can be better understood as related to psychosis, to depression, to bipolar disorder, to anxiety disorders, or to other impulsive disorders.

The original concept of BPD as a border between neurosis and psychosis led to the use of the diagnostic term “pseudoneurotic schizophrenia” (19). But this terminology confused personality disorders primarily affecting mood and impulsivity (i.e. BPD) with categories such as schizotypal personality that primarily affect cognition (20). While patients with BPD often have micropsychotic symptoms (21), neither family history studies nor biological markers support a link with schizophrenia (22,23).

Since depression is a common reason for clinical presentation in BPD, it has been suggested that BPD could be an atypical form of unipolar depression (4). This argument was based on the high frequency of family history of depression in BPD patients, as well as on commonalities in biological markers, such as a shorter REM latency (4). Some years ago, two theoretical reviews (24,25) presented a detailed critique of this hypothesis. The key point concerned the phenomenological distinction between patterns of depressive symptoms with and without BPD, largely based on temporal patterns. In classical

depression, mood is stable over weeks and is relatively unresponsive to the environment. In contrast, mood in BPD is highly mercurial. Many environmental events can cause a change in mood, and one typically sees a mixture of affects—not only sadness or anxiety, but also anger, brief periods of elation, and feelings of numbness. Thus, mood can be strikingly unstable in the course of a single day, depending on life events. Moreover, depression in BPD typically presents as chronically lowered mood rather than acute episodes (3), and early-onset dysthymia is often a marker for BPD (26). Finally, depression in BPD does not respond in the same way as classical depression to antidepressant drugs (27,28).

Recently, it has been proposed that borderline pathology falls within the spectrum of bipolar illness (29). This argument is based on an expansion of the narrower diagnostic construct of bipolar disorder into a much broader range of conditions termed the *bipolar spectrum* (30). In this model, the range of bipolar spectrum disorders would be extended to include bipolar III (antidepressant-induced hypomania), as well as bipolar IV (ultra-rapid-cycling bipolar disorder). The last category, bipolar IV seems to describes the mood swings typical of BPD (i.e., rapid shifts over hours). This expanded definition might include many, if not most, patients with BPD (31).

The issue here is whether the lability of mood seen in BPD is identical to the phenomena observed in mood disorders. Affective instability (AI) is a characteristic feature of BPD that has been shown to distinguish this diagnosis from classical bipolar disorder as well as from other personality disorders (32). Siever and Davis (33) as well as Linehan (34) have hypothesized that AI is a trait dimension that underlies the pathology seen in BPD. But the idea that AI is actually a milder form of the mood swings seen in bipolar illness has not been demonstrated (32).

Other lines of evidence have also failed to support the idea that BPD and bipolar disorder reflect the same underlying psychopathology (35). Thus, family prevalence data show that impulsive disorders are more common than mood disorders (22,23) in the first degree relatives of patients with BPD. The longitudinal course of BPD only rarely shows evolution into bipolar disorder (35). Treatment studies have not shown that mood stabilizers have the same efficacy for BPD that they do for bipolar disorder (28).

Patients with BPD can also be comorbid for anxiety disorders, including panic, generalized anxiety, and obsessive-compulsive disorder (3), although it has never been suggested that BPD is variant of these diagnoses. The concept that BPD might be a “complex” form of post-traumatic stress disorder has been suggested by frequency of childhood abuse histories in these patients (36). The problem with that proposal is its assumption that trauma is the primary cause of BPD, rather than one among many risk factors. Research shows that biological, psychological, and social factors are all involved in the etiology of BPD, while severe trauma histories are only found in about a third of cases (1).

Another possible “border” of BPD lies with other impulsive disorders. Impulsivity may be as central to the clinical picture of BPD as affective instability (33,37). High rates of substance use and antisocial personality are found in the families of borderline patients (22,23), and levels of impulsivity predict long-term outcome in BPD (38). Moreover, most neurobiological studies of BPD that have examined biological and genetic markers have reported stronger links with impulsive traits than with clinical diagnosis (39). What might distinguish BPD most is the combination of affective instability and impulsivity in one clinical syndrome (33).

### ***Why Clinicians Are Reluctant to Diagnose BPD***

In practice, clinicians can be reluctant to diagnose BPD. There are several reasons for their hesitation. First, making an accurate Axis II diagnosis requires experience. Personality disorders tend to lack the precise phenomenologically based criteria associated with some categories. For example, it has been shown that structured interviews pick up many cases of BPD missed in ordinary practice (40). Second, resistance to seeing patients as having a personality disorder may be based on the idea that these conditions are untreatable (41) (or at least not treatable using the pharmacological tools that have come to dominate the treatment of so many other disorders). While there is good evidence for the efficacy of psychotherapy in BPD (34,42) not every clinical setting today has the human resources to provide this form of treatment.

Clinicians also wish to reduce stigma for patients. It is an unfortunate reality that a diagnosis of BPD can sometimes lead to rejection by the mental health system. Some may believe if BPD were to be reclassified, for example, as a mood disorder, patients might benefit from being seen as having a “chemical imbalance” instead of having a problematical personality. What this idea fails to consider is that stigma cannot be removed by reclassification. Patients with the problems seen in BPD will continue to be just as difficult, albeit under a different diagnostic label.

### ***Reasons for Making the Diagnosis of BPD***

There are real advantages in making the diagnosis of BPD: providing a conceptual framework for psychopathology, predicting outcome, predicting response to pharmacotherapy, and supporting the prescription of psychotherapy.

The first advantage concerns the classification of complex forms of psychopathology. It was once a tradition for physicians to say, “know syphilis and you know medicine,” given the effects of that disease on many organ systems. Today, psychiatrists might make a similar claim for BPD. One of its principal characteristics is its protean nature. Instead of dismantling the construct into multiple symptomatic components, it is more useful to make use of the concept of personality disorder.

Patients with BPD present with a wide range of clinical phenomena, each of which seems to point to a different diagnosis. Yet none of these symptoms actually occur in isolation. The usefulness of diagnosing any personality disorder, including BPD, is that the construct can account for the co-occurrence of a wide range of affective, impulsive, and cognitive symptoms in the same patient. None of the alternatives offered thus far account for the range of clinical phenomena seen in this population.

The second advantage concerns the prediction of outcome. One of the key features of any personality disorder is a chronic course. The distinction between Axis II and Axis I disorders is far from absolute, since patients with BPD often have a waxing and waning course (43), while Axis I conditions can also be chronic (44). However, BPD has a characteristic course over time, with symptoms peaking in early adulthood, followed by gradual recovery in middle age (1). This information is important and useful, both for practitioners and for patients.

The third value of diagnosing BPD lies in predicting response to pharmacotherapy. A robust finding in the literature is that medication for depression is less effective in the presence of any personality disorder (27). Although patients with BPD may respond to these agents (45), results are not consistent (28). Perhaps the most robust finding in the literature is that (as recently confirmed by Zanarini and Frankenburg (46), patients with BPD often benefit from low-dose neuroleptics. The problem is that since none of these agents yields the same results as they do in the disorders for which they were originally developed, BPD patients end up receiving polypharmacy (47), and it has not been shown that such regimes are clinically effective. Clinicians and patients need to take these facts into account.

The fourth advantage of diagnosing BPD is the evidence that psychotherapy can be an effective form of treatment for the disorder (34,42). As clinicians have long observed, and as this research confirms, addressing issues related to dysfunctional personality traits can lead to as much, if not more, symptomatic relief than psychopharmacological management.

Whatever the problems with the BPD diagnosis, there are also problems with *not* diagnosing this disorder. Professionals treating patients meeting criteria for this disorder need to benefit from the large empirical literature bearing on this complex clinical problem. Moreover, clinicians who see these cases as examples of Axis I pathology may have mistaken expectations about course and treatment response. Finally, we need to inform and educate patients and their families about this condition (48).

### ***BPD as a Multidimensional Disorder***

BPD is only one of many psychiatric diagnoses that lack established validity. As is the case for these other categories, there are good reasons to retain this diagnostic term until a better construct becomes available.

The literature criticizing the construct of BPD has placed too much emphasis on comorbidity. Far from being a measure of independent disease entities, this phenomenon is an artefact of our lack of knowledge about disease processes (49). The more symptoms patients have, the more likely they are to meet criteria for multiple disorders, so that comorbidity tends to reflect overall severity of illness. This is a problem for most of the categories in DSM-IV-TR. Given this dilemma, and the likelihood that disease processes usually affect more than one dimension of pathology, focusing on only one aspect of symptomatology, and making it a defining feature, is a perilous approach to classification.

Thus, the comorbidity of BPD may only reflect the reality of a disorder that affects many symptomatic dimensions. As shown by family prevalence and outcome data, the phenotypes behind BPD must be complex. In particular, if BPD were an atypical form of an Axis I disorder, "pharmacological dissection" should show that it responds to similar medications. Instead, BPD responds partially to a wide number of agents developed for other diagnoses, and does not have a definitive response to any of them (28).

### ***CONCLUSIONS***

The diagnosis of BPD will eventually be replaced by another approach to personality diagnosis. For the present, however, it can be considered as the best among alternatives. There is little benefit in re-diagnosing patients with other mental disorders, when they do not respond to the same treatments.

Ultimately, when we find reliable ways to delineate and demarcate mental disorders, our classification will no longer need to depend on phenomenology alone. Differential diagnosis would come to depend on biological markers such as laboratory testing, imaging, and genotyping. The current state of medicine is the future of psychiatry.

The questions raised in this article can only be addressed through further empirical research. More studies need to be carried out on genetic and biological factors, on development, on longitudinal course, and on treatment. Only then will we be in a position to replace BPD with something better: either a newly named disorder, or, more likely, a set of disorders.

While waiting for future developments, we still need to diagnose and treat patients. BPD can claim the same status as current psychiatric diagnoses: the one that Winston Churchill once proposed for democracy, "the worst form of government, except for all the others that have been tried from time to time."

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