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BIPOLAR DISORDERS (R HIRSCHFELD, SECTION EDITOR)

Differential Diagnosis of Bipolar II Disorder and Borderline Personality Disorder

Adam Bayes[1,2] · Gordon Parker[1,2] · Joel Paris[3]

Abstract

Purpose of Review Differentiating bipolar (BP) disorders (in particular BP II) from borderline personality disorder (BPD) is a common diagnostic dilemma. We sought to critically examine recent studies that considered clinical differences between BP II and BPD, which might advance their delineation.

Recent Findings Recent studies focused on differentiating biological parameters—genetics, epigenetics, diurnal rhythms, structural and functional neuroimaging—with indicative differences not yet sufficient to guide diagnosis. Key differentiating factors include family history, developmental antecedents, illness course, phenomenological differences in mood states, personality style and relationship factors. Less differentiating factors include impulsivity, neuropsychological profiles, gender distribution, comorbidity and treatment response.

Summary This review details parameters offering differentiation of BP II from BPD and should assist in resolving a frequent diagnostic dilemma. Future studies should specifically examine the BP II subtype directly with BPD, which would aid in sharpening the distinction between the disorders.

Keywords Bipolar disorder · Borderline personality disorder · Diagnosis · Affective instability

Introduction

The relationship between borderline personality disorder (BPD) and bipolar (BP) disorder is much debated including whether they are independent or interdependent conditions. For example, the fluctuating mood symptoms observed in those with BPD has led some to position it as an 'ultra-rapid cycling' subtype of BP disorder, arguing for it to be placed on the BP spectrum [1]. However, as reviewed by Paris and Black [2], there appear to be certain BPD features (e.g. micropsychotic symptoms and interpersonal difficulties) that are not clearly explainable as related to mood fluctuations and thereby challenge it being viewed as a BP condition. Furthermore, a recent latent class analysis of BPD and BP symptoms in 34,653 individuals generated a three-factor solution (i.e. BPD, depression and mania)—with correlations between factor pairs indicative of two separate syndromes (BPD and BP disorder) [3•].

A frequent clinical diagnostic dilemma is whether a patient has one or the other disorder [4, 5]. Both can present with altered mood states, deliberate self-harm (DSH) and suicidality and evidence impulsivity such as sexual disinhibition, excessive spending and alcohol and drug misuse [6]. Features common to both disorders (e.g. affective instability (AI) and impulsivity) are considered 'transdiagnostic', and their presence may compromise diagnostic accuracy [7]. At cross-sectional assessment, periods of AI in BPD patients can resemble hypomania [8], leading to a false diagnosis of a BP disorder [9], while inter-episode residual symptoms in those with a BP condition, such as chronic dysphoria, may also compromise diagnosis [10].

Differentiating BP I disorder from BPD is usually more straightforward due to BP I typically being of greater severity and the frequent presence of psychotic features during mania. Distinguishing BP II disorder from BPD is

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✉ Adam Bayes
a.bayes@unsw.edu.au

[1] School of Psychiatry, University of New South Wales (UNSW), Sydney, Australia

[2] Black Dog Institute, Sydney, NSW, Australia

[3] Institute of Community and Family Psychiatry, SMBD-Jewish General Hospital, McGill University, Montreal, Quebec, Canada

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Table 1 Features differentiating bipolar II disorder from borderline personality disorder

Feature	Bipolar II disorder	Borderline personality disorder
Heritability	More likely for there to be a first-degree relative with BP disorder (especially BP II)	Less likely to have a first-degree relative with BP disorder; more likely for relatives to have impulse control disorders (antisocial personality and substance abuse), unipolar depression or borderline features
Age of onset	Onset usually in late teenage years with a distinct 'trend-break'	Lack of clear onset, with emotional difficulties since childhood
Mood state precipitant	More likely autonomous (but can also be reactive)	More likely to be triggered by interpersonal interactions (such as perceived abandonment)
History of childhood sexual abuse	Lower likelihood	Higher likelihood
Gender	Slight female predominance	Female preponderance in clinical settings
History of other developmental trauma (e.g. physical and emotional abuse)	Lower likelihood	Higher likelihood
Early parental relationships	Distant/rejecting parent unlikely	Distant/rejecting parent likely
Childhood depersonalization	Unlikely	Likely
Psychotic features	Hypomanic states lack psychotic features by definition; BP II depressive states may rarely include psychotic features	Transient psychotic features are common, especially under stress
Phenomenology of 'highs'	Grandiosity, euphoria (can be irritable); anxiety typically abates	Euphoria rare or brief (i.e. < 48 h); anxiety often distinctive
Depressive symptoms	More likely melancholic in nature	More likely non-melancholic in nature
Affective instability	Severity: May be present but generally not severe Quality: Shifts from euthymia to depression or elation Triggers: More likely autonomous	Severity: Commonly high Quality: Shifts from euthymia to anxiety or anger Triggers: More likely interpersonally driven
Emotional regulation	Maladaptive strategies less severe; adaptive strategies superior	Maladaptive strategies more severe; adaptive strategies impaired
Deliberate self-harm and suicide attempts	Less likely	More likely
Neuropsychological deficits	Both trait and mood-state dependent deficits; broad deficits in executive function and sustained attention	Executive function deficits less clear. Sustained attention intact
Impulsivity	More likely during a hypomanic episode; 'attentional impulsiveness'	Not necessarily related to mood state; 'non-planning impulsiveness'
Personality variables	No distinctive personality traits	Distinct sensitivity to criticism by others
Social cognition	Impaired theory of mind; likely moderated by mood state	Failure of 'mentalization'
Relationships	Generally stable relationships; less avoidance due to fear of rejection	Distinctive relationship difficulties; avoidance due to fear of rejection
Self-identity	May be impacted during mood episodes, but more stable when euthymic	'Painful incoherence'
Prognosis	Does not remit with age, and can often worsen	Tends to improve over time, and criteria for the disorder may not be met by middle age
Treatment response	Mood episodes likely to respond to mood stabilizers and atypical antipsychotic drugs	Poor response to mood stabilizers; non-specific response to medications (e.g. sedation)

often less clear, as hypomanic mood episodes are non-psychotic, of briefer duration and are usually less severe.

The aim is to review the literature differentiating BP II from BPD—with a focus on more recent studies. Where specific BP II studies were lacking, we examined studies of BP disorder in general. Our review proceeds by considering candidate differentiating parameters.

Depressive Symptoms

Phenomenological differences in depression include an over-representation of melancholic symptoms in BP II [11], as well as mixed and agitated symptoms [12], whereas BPD is more characterized by non-melancholic depressive episodes reactive to interpersonal life events [13] (see Table 1). Atypical

features of depression (e.g. hypersomnia and hyperphagia) are common to both BP II [14] and BPD and therefore offer little discriminatory value [15]. 'Typical' depressive features (e.g. decreased self-esteem and self-criticism) are associated with BP II, whereas BPD depressive states are more characterized by emptiness and 'painful incoherence' [16].

Hypomanic Symptoms

Hypomania is a necessary feature of BP II, with individuals describing feeling energized ('wired'), euphoric and with any anxiety having disappeared. Conversely, those with BPD rarely describe elevated mood and instead display affective instability (AI) in the form of rapid mood changes described as an 'emotional roller coaster' [17] and often with marked anxiety. The presence of 'irritable' highs [9], mixed mood states [18] or ultra-rapid cycling in some BP II individuals may be mistaken as BPD-specific AI. More recently, the 'four-day rule' for hypomania has been criticized as arbitrary and not supported by empirical data [19]. The risk of not imposing a minimum duration to the BP disorders is that of false positive diagnoses [20]. However, one recent study did not confirm increased misdiagnosis of BPD when 'soft' BP criteria were compared with strict DSM hypomania criteria [21], suggesting that a valid BP II diagnosis can still be made if other features are considered.

Affective Instability (AI)

Affective instability, also termed emotional dysregulation (ED), refers to brief mood changes characterized by temporal instability, high intensity and delayed recovery from dysphoria [17]. AI is considered a core feature of BPD but also occurs in BP even during periods of euthymia [22, 23]. Underlying mechanisms for AI appear to differ across conditions—in BP II, it appears more autonomous and internally driven, compared with being reactive to interpersonal stressors in those with BPD [24]—with differing neurobiological processes underlying each condition supported by functional neuroimaging studies as shortly considered. Self-report measures comparing BPD with BP II have found that those with BPD experience rapid mood shifts that are qualitatively different from those experienced by BP II individuals—involving changes from euthymia to anxiety or anger—as against shifts from euthymia to depression or elation in those with BP II [25]. Using ecological momentary assessment methods, Mneimne et al. [26] compared non-subtyped BP with BPD individuals and found the latter as characterized by high interpersonal reactivity to shame and guilt as well as inertia or 'getting stuck' in shame, with both conditions evidencing transdiagnostic features relating to irritability and anger. Smartphone self-report mood ratings have been shown to differentiate individuals with BP (non-subtyped) and BPD based on variability in responses (e.g. BPD individuals demonstrating higher ratings of distress compared with those with BP) [27], and using machine learning, Arribas et al. [28••] were also able to distinguish BP (non-subtyped), BPD and healthy controls, based on smartphone mood ratings with 75% accuracy. Overall, AI can be considered a transdiagnostic feature across disorders, more prominent in BPD and with phenomenological nuances offering differentiation now better captured by emergent technologies.

Emotional Regulation

Emotional regulation involves processes responsible for evaluating, modifying and monitoring emotional reactions [29, 30]. Those with BPD display impaired emotional 'granularity'—including difficulties in mood labelling [31], deficits in emotional awareness and 'clarity' [32] and employ maladaptive cognitive strategies such as rumination [33] and thought suppression [34]. Deficits have also been found in a combined BP I and II sample even during remission from mood episodes, including poor suppression of emotion-related neural hyperactivity, deficits in reappraisal capacity, elevated impulsivity in emotionally arousing situations and use of negative attentional strategies to regulate mood [35]. In a study directly comparing a predominantly BP II group with a BPD group, maladaptive emotional regulation strategies (e.g. catastrophizing and rumination) were more severe in the BPD group, and adaptive strategies superior in the BP group [36]. Differences in emotional regulation strategies may assist differential diagnosis and advance choice of psychological interventions.

Mentalization

Deficiencies in the ability to 'stand outside of oneself' and observe emotions has sometimes been termed a failure of 'mentalization' or theory of mind (ToM) and is a core deficit in those with BPD [37] impacting on their interpersonal relationships. However, lower ToM performance has also been found, compared with controls, in euthymic BP II participants [38].

Gender

For BPD, a female preponderance is general to most treatment settings [39]. A comparable gender ratio has been reported for BP; however, 'the rate of bipolar II disorder is more often higher among females…a result suggested by clinical studies' [40].

Heritability

Family history studies reveal BPD probands to be more likely to have first-degree relatives with either impulse control disorders (antisocial personality and substance abuse) or unipolar depression and with BP unlikely [41]. Additionally, borderline 'features' are over-represented in family members of those with BPD [42]. There is a greater probability of first-degree relatives with a BP or a major mood disorder in BP probands [4] with Benazzi [43] reporting that BP II individuals had many more first-degree relatives with BP II than those with a BP I disorder, suggesting a familial subtype pattern. Overall, a family history of BP disorder is likely to support a BP II as against a BPD diagnosis.

Illness Course

Onset of childhood BP is rare, with late adolescence or early adulthood providing the highest risk period [44, 45] and generally evidenced by a distinct 'trend break' [46]. For BPD, a distinct onset is generally lacking [8]. BP disorders tend not to remit with age and often worsen [40], whereas the majority of those with BPD recover or improve with time, no longer meeting disorder criteria by middle age [47].

Suicidality and Deliberate Self-Harm (DSH)

Suicidality is a core diagnostic risk for those with BPD, albeit being common in BP II [48], with a large study of BPD versus predominant BP II participants quantifying respective suicide attempt rates at 60% versus 30% [49]. DSH (e.g. wrist cutting) also occurs in both BPD and BP II (especially during mixed states) [50] though with Bayes et al. [49] establishing a greater prevalence in BPD compared with a predominantly BP II group (83% vs. 49%).

Mood State Context

Many symptoms of BPD are frequently reactive to interpersonal events such as perceived rejection or abandonment [8]. Individuals with BP II are more likely than those with BPD to have spontaneous mood episodes [24], although reactive moods to psychological stressors may occur.

Neuropsychological Deficits

Few studies have directly compared BPD with BP II participants, with most comparing either BP II or BPD with controls. Executive function deficits have been found in BPD patients [51], though there is variability in the literature regarding the extent of differences compared with healthy controls [52]. Broader deficits in executive function have been found in BP II patients [53]. For example, a meta-analysis of 263 patients with BP II and 415 controls quantified greater deficits in global cognition, as well as specific deficiencies in processing speed, visual memory, complex figure recall and verbal and working memory [54]. However, deficits have been reported as dependent on mood state and episode type [55]. Sustained attention deficits are typically seen in BP [56] though not in BPD subjects [51].

Structural Neuroimaging

Studies directly compare BPD and BP II subjects using the same imaging paradigms are lacking, making comparisons across conditions difficult. A meta-analysis of all voxel-based studies comparing BPD or BP with healthy controls on measures of grey matter (GM) volume and GM density found that those with BPD showed differences predominantly in the amygdala and parahippocampal gyrus versus the cortico-thalamic-striatal circuit in BP subjects [57•]. A structural magnetic resonance imaging study compared BPD versus BP (87% type I and 13% type II) and found considerable overlap of GM changes but with BP subjects having more severe and diffuse changes [58]. The BP group had additional specific GM changes in cortical and subcortical structures versus those with BPD where changes were mostly in the fronto-limbic region and with white matter changes involving completely different regions evident in both conditions [58].

Functional Neuroimaging

Similar to structural neuroimaging studies, there have been few direct comparison studies involving BP and BPD groups. Boen et al. [59••] examined 22 individuals with BPD, 22 with BP II and 21 controls using positron-emission tomography (PET), finding those with BPD demonstrated hypometabolism in the midbrain, hypothalamus and striatum whereas those with BP II showed hypometabolism in the cerebellum. Both disorder groups showed hypometabolism in the insula areas suggesting a component of shared pathophysiology. A small functional magnetic resonance imaging study of non-subtyped BP versus BPD subjects found differential engagement of fronto-limbic emotion processing distinguished BP from BPD individuals [60]. Increased dorsomedial prefrontal cortex activity was observed in the BP group and reduced amygdala activity in BPD individuals, pointing to differing neural processing possibly underpinning the AI observed in the two conditions [60]. Differences in resting state functional network connectivity was examined across networks involving social cognition, emotion regulation and the self-referential processing system in those with BP (non-subtyped), BPD and controls [61]. In BP subjects, impairment occurred primarily among networks involved in self-referential processing—with such networks thought to underpin emotional dysregulation in mood disorders. In BPD

subjects, functional network connectivity additionally involved the emotion regulatory network [61]. These limited studies are suggestive of certain shared neural mechanisms underpinning overlapping symptoms with additional unique processes suggesting group differentiation.

Electroencephalogram (EEG)

To date, EEG findings have not differentiated the two conditions. In one study [62], the resting EEG in non-subtyped BP, BPD and control groups showed greater power in slow and fast oscillations in both clinical groups only, with the lack of differences between BP and BPD subjects potentially pointing to a shared biology.

Childhood Trauma

History of childhood trauma (e.g. physical, emotional or sexual abuse) is not a clear differentiating feature, as high rates are associated with both disorders—approximately 50% in those with BP and 60–80% in BPD patients [4]. However, Bassett [4] has suggested that the specific form of abuse may vary across disorders, while a study involving a largely BP II (vs. BPD) sample found childhood sexual abuse was a strong predictor of BPD with an odds ratio of 9.38 and with multivariate analyses suggesting its greater developmental impact compared with negative parental characteristics and other developmental trauma [49].

Genetics and Epigenetics

A recent genome-wide association study found genetic overlap of BPD with BP disorder (non-subtyped) as well as with schizophrenia and major depressive disorder, suggesting a shared aetiological factor [63]. The serotonin 3A receptor (5-HT_{3A}R) has been found to be associated with differing psychiatric disorders and interacts with early life trauma [64]. A recent study examined methylation (i.e. epigenetic modification) of 5-HT_{3A}R in individuals with non-subtyped BP and those with BPD, finding it related to severity of illness across both disorders and thus a suggesting a common rather than a distinguishing factor [64].

Biorhythms

Diurnal rhythms have been compared across BP, BPD and controls using smartphone mood ratings, portable actigraphy and heart rate devices. For example, a desynchronization in HR (compared with activity) of 3 h was observed in those with BPD and 1 h in BP individuals, and sleep (compared with activity) was desynchronized by 2 h in BPD individuals but not in those with BP [65•]. Another study by Carr et al. [66] of non-subtyped BP, BPD and controls found a positive correlation between mood variability and variation in heart rate, sleep and activity—and greatest in the BPD group. A number of studies have found, somewhat surprisingly, more severe diurnal rhythm disturbance in BPD compared with BP individuals.

Impulsivity

Impulsivity observed in BPD typically manifests as a way of coping with negative emotions, such as by distraction [67]. In contrast, impulsivity in BP individuals is present as both a trait [68] and with additional mood-state impulsivity more frequently associated with hypomanic versus depressive episodes [69]. Direct comparison of impulsivity levels found higher scores on the Barratt Impulsiveness Scale in BPD relative to BP II individuals [25] with Boen et al. [70], finding BPD (versus BP II) participants showed higher scores on the urgency and (lack of) perseverance dimensions. Wilson et al. [69] found those with BP II demonstrated 'attentional impulsiveness' associated with cognitive disturbances (e.g. racing thoughts, distractibility and impaired concentration), whereas those with BPD showed motor and 'non-planning impulsiveness' (e.g. difficulty planning actions and thinking about consequences).

Self-Identity

Those with BPD more experience a 'noxious' and fragmented sense of self [16] compared with those with BP II who may experience self-deficits during depressive episodes, grandiosity when hypomanic, and stability of self-identity when euthymic [24]. In a study comparing a BPD group and a predominant BP II group across 113 self-report items of features considered integral to BPD—items relating to identity disturbance were found to be both intrinsic to BPD and offered specificity in differentiating from BP [71].

Relationships

Assessment of the capacity to sustain relationships may assist diagnosis—those with BPD experience ongoing discrepancies in their assessment of self and others, exhibit severe abandonment fears and have a tendency toward idealization and devaluation [8]. In a study of individuals with a BPD versus a predominantly BP II group, it was found the former reported a greater avoidance of relationships due to fear of rejection [49]. Individuals with BP (when euthymic) tend to be better able to maintain stable relationships [8].

Psychotic Features and Dissociation

Up to 75% of those with BPD experience transient dissociative and paranoid symptoms, which are often stress-related

[72]. Whereas in BP II disorder, hypomanic episodes by definition lack psychotic features, while such features are rare during depressive episodes (lifetime prevalence estimates ranging from 3 to 45%) [73]. Regarding dissociative symptoms, adult patients who reported depersonalization first occurring in childhood were found to have a higher probability of having BPD versus BP disorder [49].

Psychiatric Awareness

Key influences impacting accurate differential diagnosis of BPD include the effect of 'Big Pharma' promotion of drug therapies and many clinicians not being trained in the evidence-based psychotherapies (e.g. dialectical behaviour therapy) for BPD. Such factors may bias clinicians toward making a diagnosis, which have associated pharmacological treatment options—and what Zimmerman has described as the problem of 'diagnosing BPD in a bipolar world' [74].

Personality and Temperament

Personality factors have been examined in a predominantly BP II sample versus a BPD group, with multivariate analyse revealing sensitivity to criticism being a key differentiating factor favouring a BPD versus a BP II diagnosis [49]. Perugi et al. [15] suggested that interpersonal sensitivity traits and mood lability are related to a shared cyclothymic temperament linking BPD, BP II and atypical depression. The presence of a cyclothymic temperament in those with BP II may lead to misdiagnosis of BPD. Akiskal et al. [75] described this presentation as 'BP II 1/2'—a 'dark', unstable variant of 'sunny' BP II disorder—which compared 'classic' euphoria-driven hypomanic symptoms is more associated with irritable risk taking.

DSM Criteria

DSM-5 lists BP as an important differential diagnosis of BPD. Conversely, BPD is listed as a differential diagnosis for both BP conditions. However, DSM does not offer information on the relative specificity of the differing items to its definition of BPD and their delineation from BP. Diagnostic efficiency of BPD criteria were examined in a study comparing a BPD group with a predominant BP II group with results finding 'affective instability' to occur with a high prevalence in both conditions, whereas 'identity disturbance' and 'abandonment fears' offered superior diagnostic efficiency in distinguishing BPD from BP (unpublished data).

Screening tools

Structured measures screening for BP disorder offer variable capacity to differentiate those with a BPD or a BP disorder. For example, for those screening positive for BP on the Mood Disorder Questionnaire (MDQ), the frequency of BPD was 80% of the rate of BP disorder [76]. However, a study using the MDQ in a mood clinic found a BP diagnosis was strongly predicted by the presence of elevated mood, increased goal-directed activity and episodicity (sensitivity = 88.7; specificity = 81.4), though BPD was only predicted by female gender [77] a finding replicated in a sample of BP (BP I, BP II and BP NOS) and BPD patients [78]. Examining BPD symptoms, the Personality Inventory for DSM-5 found the BPD algorithm (risk taking, impulsivity, emotional lability, separation insecurity, hostility, depressivity and anxiousness) demonstrated moderate accuracy in differentiating BP 'spectrum' disorders from BPD [79]. Overall, screening instruments may be of some value in offering indicative differentiation of the two conditions.

Comorbidity

Both BP and BPD are associated with an increased risk of substance abuse and anxiety disorders [80, 81]. In a sample of those with BP II, there was a lifetime over-representation of anxiety disorders [82]. Comorbid attention deficit hyperactivity disorder (ADHD) appears to also be overrepresented in BP II [82] and BPD conditions [83]. It is unclear whether comorbidity is greater between BP and BPD relative to other personality disorders, with mixed results reported [84, 85].

Comorbid BP II and BPD

A review of studies examining BP and BPD comorbidity quantified 10% of BPD individuals having a BP II diagnosis and 20% of those with BP II having a BPD diagnosis [20] and with a similar rate of 24% lifetime prevalence of BPD in BP II individuals quantified by McDermid et al. [86]. Frias et al. [87] reviewed evidence of non-subtyped BP and BPD comorbidity and found that the presence of BPD increased the number of BP mood episodes. However, presence of BP did not appear to have a negative impact on BPD. Comparing a group of 294 BP I, 72 BP II and 9 BPNOS (BP not otherwise specified) individuals, more baseline borderline personality features were associated with higher BP episode frequency [88•]. An effect of BP II on BPD diagnosis has been observed in a study, which examined 223 BPD, 24 BP I and 28 BP II individuals, finding that, while BPD and BP disorders were largely independent, BP II (but not BP I) specifically lengthened time to remission of BPD [89]. BPD has been shown to be a risk factor for developing BP (though not the reverse) [87] with McDermid finding BPD was a strong predictor of incident BP II [86]. Parker et al. [90], in a study of BPD and predominant BP II individuals, found that, irrespective of whether BPD occurs alone or comorbid with BP, individuals were more likely to have experienced childhood sexual abuse

or other developmental trauma, report depersonalization in childhood, to have experienced deficient parenting, exhibit DSH and return higher borderline personality profile scores—thus explaining why some features viewed as specific to those with BPD are reported by those with a BP condition. Overall, while the literature suggests that the majority of those with BPD are not comorbid for BP II (and vice versa), their co-occurrence is not rare and likely contributes to the clinical differential diagnostic dilemma (which is generally a binary one of choosing between the two options rather than allowing that both may be present).

Response to Treatments

'Pharmacological dissection' can be used to differentiate conditions however non-specific effects of drugs (e.g. sedation) may limit interpretation of findings. BPD patients rarely improve when prescribed mood stabilizers, with lithium in particular, failing to show utility for personality disorders [91]. Antidepressants, anticonvulsants, mood stabilizers and atypical antipsychotics appear more beneficial for BP conditions relative to BPD [4]. Shared features of both conditions and the non-specific benefits of psychological treatments also limit the capacity for psychotherapeutic response to offer diagnostic differentiation. There is evidence for use of adjunctive cognitive behaviour therapy (CBT) in BP depression [92], with benefits of CBT also observed in BPD, including reduction of dysfunctional beliefs [93]. Dialectical behaviour therapy (DBT) and mentalization-based therapies are first-line treatments for BPD [94], yet preliminary evidence (from a combined BP I and II sample) suggests adjunctive DBT is also effective in reducing BP depressive symptoms [95].

Diagnostic Assessment

Overall, the literature supports a diagnostic model whereby BP II and BPD exist as independent conditions [49] while acknowledging they can also co-exist in some individuals [90]. However, an over-reliance on the dominant psychiatric diagnostic paradigm, which focusses on polythetic symptom-based criteria, may obscure accurate differential diagnosis due to shared features. Such limitations led the ICD-11 to shift to a dimensional approach to personality, and the latest iteration of DSM-5 contains an additional alternative personality model (in Section III), which emphasizes impairment in personality functioning, as well as personality traits, which map onto the categorical BPD diagnosis. Fowler et al. [96] recently used the Personality Inventory for DSM-5 (PID-5) BPD algorithm (based on Section III BPD traits) in a sample of those with BPD or BP (non-subtyped), finding that this showed moderate-to-excellent accuracy in differentiating the disorders. Dimensional approaches to BPD offer an alternative method of evaluation, though it is yet to be seen if such strategies are useful clinically.

Clinical acumen continues to be a critically important tool of psychiatric diagnosis, as it utilizes a broader set of clinical features as well as longitudinal history data. Examination beyond symptoms, with an additional focus on non-symptom features may improve diagnostic accuracy. For example, a large head-to-head comparison study of individuals with BPD or BP [49] found key differences across diagnostic validators, which favoured a BPD diagnosis—namely a history of childhood sexual abuse, depersonalization in childhood, personality features related to relationship difficulties and sensitivity to criticism and an absence of a family history of BP disorder—which yielded high diagnostic accuracy of between 92 and 95%. The related study by Parker et al. [90] extended these findings and demonstrated that, irrespective of whether the borderline condition occurred on its own or was comorbid with BP, the developmental antecedents, personality profile and deliberate self-harm rates were the same—thus arguing for the co-occurrence of two independent conditions.

Such an independence model also argues for the following approach to clinical assessment [97]: firstly, assessing whether a BP II condition is present or absent—with a weighting of features specific to BP disorder; and, secondly, assessing for the presence or absence of a borderline condition—with a weighting toward developmental trauma as well as borderline features. Management approaches then focus on treating either a BP II disorder or a BPD or both conditions as detailed below.

Management Approaches

Studies examining efficacy of treatments (see Response to Treatments section above) suggest an approach to BP II disorder, which is weighted to pharmacotherapy—with a mood stabilizer (e.g. lamotrigine and lithium) considered necessary in the majority of patients [98]. Treatment of depressive episodes with antidepressant medications may also be indicated though with mixed opinions regarding the risk of switching [98]. Atypical antipsychotic medications may also play a role in mixed states, rapid-cycling or breakthrough hypomania [98]. Psychological interventions (e.g. psychoeducation, CBT, interpersonal and social rhythm therapy), while they are considered adjunctive, are nonetheless important in the overall management of those with BP II [98].

By contrast, for BPD, the primary treatment approach is psychotherapy (as detailed above). Therapies such as DBT improve emotion regulation in those with BPD, whereas mood stabilizers offer little benefit or only non-specific effects (e.g. sedation secondary to atypical antipsychotics). It is important to diagnose any comorbid major depressive disorder in those with BPD as this may warrant antidepressant therapy.

The presence of both conditions warrants either treating each condition concurrently or using a sequenced approach, whereby the most significant condition is first brought under control (most likely BP) and then the second disorder managed. There are few described approaches to the management of comorbid BP and BPD [99] though attending to both disorders using a multimodal strategy is critical for the patient's overall recovery.

Conclusions

We sought to critically examine recent studies evaluating differentiation of BP II disorder from BPD. The literature continues to be limited by a lack of studies directly comparing BP disorder with BPD head-to-head, and, where there have been direct comparisons, many studies fail to specifically examine the BP II subtype separately from BP I. Overall, there is a trend for more recent studies to examine for biological differences between the conditions, relating to genetics, epigenetics, diurnal rhythms and structural and functional neuroimaging. These studies have found indicative differences across BP and BPD, though to date are not yet sufficient to guide differential diagnosis at a clinical level. Technological advancements have seen increasing use of ecological momentary assessment allowing the transdiagnostic feature of affective instability to be interrogated, with differences between the conditions elucidated. Future biological studies would benefit from examination of the BP II subtype to elucidate those transdiagnostic features common to BP II and BPD and those that are unique and offer differentiation at a biomarker level.

Clinically, being able to recognize hypomanic symptoms—which should alone be sufficient to diagnose a BP II disorder—as distinct from affective instability is a key issue in resolving the diagnosis. However, when the status of hypomania is not clear or readily distinguishable from AI, the literature supports use of a number of other symptom and non-symptom validators, which may clarify the diagnosis—including features related to family history, illness course, developmental antecedents (e.g. trauma history and parental relationships), phenomenological differences in mood states, personality style and relationship factors. Other differentiating factors of equivocal use include features related to comorbid conditions, impulsivity, neuropsychological profiles, gender distribution and treatment response. Resolving the diagnostic dilemma is important as treatment of individuals with BP II is typically weighted to pharmacotherapy (usually a mood stabilizer) with psychological interventions considered adjunctive, whereas those with BPD benefit most from psychotherapies with medications considered secondary. In the minority of those that truly exhibit co-occurring conditions each disorder should be treated either concurrently or sequentially—with the latter model involving stabilizing the most significant condition first (generally the BP disorder) and then focusing on the other disorder.

Compliance with Ethical Standards

Conflict of Interest Adam Bayes, Gordon Parker and Joel Paris each declare no potential conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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