# Chapter 14
# Conclusion: Integration and Synthesis

**Lois W. Choi-Kain and John G. Gunderson**

The relationship between mood disorders and borderline personality disorder (BPD) has long been controversial, fueling fierce debates about psychiatric diagnosis and treatment [1–3]. This controversy has spurred the development of a significant body of research, which allows us to ground our hypotheses and claims primarily in evidence rather than polemics. This book is an effort to review, synthesize, and evaluate the current evidence on the relationship between mood and borderline personality disorders. We hope to promote more objective and tentative conclusions that inform more effective clinical care of and continued research on the interaction between these commonly encountered disorders.

The adversarial nature of the original debates between the mood and personality disorder worlds arose from territorial agendas, revolving around efforts to establish the legitimacy of these respective disorders in an era where the criteria for most psychiatric diagnoses known today were in early stages of empirical validation. In the context of these diagnostic turf wars, much of the language and tone of the debate between mood and personality disorder experts was competitive and undercutting. The chapters contributed by Paris as well as Ghaemi and Barroilhet represent the evolution of this debate. Paris argues that the trend towards biological reductionism has caused neurobiological understandings and psychopharmacologic treatments to edge out psychoanalytic concepts and psychotherapeutic interventions.

L.W. Choi-Kain, MD, MEd (✉)
Department of Psychiatry, Harvard Medical School, 115 Mill Street, Belmont, MA 02478, USA

McLean Hospital, 115 Mill Street, M/S 312, Belmont, MA 02478, USA
e-mail: lchoikain@partners.org

J.G. Gunderson, MD
Department of Psychiatry, Harvard Medical School, 115 Mill Street, Belmont, MA 02478, USA

McLean Hospital, 115 Mill Street, M/S 312, Belmont, MA 02478, USA
e-mail: jggunderson@mclean.harvard.edu



L.W. Choi-Kain, J.G. Gunderson (eds.), *Borderline Personality and Mood Disorders: Comorbidity and Controversy*, DOI 10.1007/978-1-4939-1314-5_14

This shift has pushed mental health clinicians to prefer simpler conceptualizations of psychiatric presentations in terms of mood disorders as opposed to more complex formulations in terms of personality disorders. Ghaemi and Barroilhet make a similar distinction with different implications. They argue that bipolar disorder is more of an illness or disease than BPD because it is almost completely genetic in etiology. Both Paris and Ghaemi and Barroilhet distinguish BPD as a complex clinical picture which develops primarily from psychosocial influences. Both chapters focus their criticism more at the way in which the diagnostic concepts are applied and less at the legitimacy of mood or borderline diagnoses. At the same time, these chapters still embody dichotomizing tendencies that falsely or simplistically separate biologically and psychosocially based disorders and interventions. This dichotomy provides clinicians and researchers with hard edges around which to draw lines between categories of psychiatric illness, providing clarity in the face of clinical complexity. The problem arises when these dichotomizing tendencies position disorders as competitive, suggesting, as Ghaemi and Barroilhet indicate, that the overlap between psychiatric syndromes simply means one represents the other.

This collection of chapters reviews and synthesizes the existing literature to enable mental health professionals to develop a more nuanced and realistic way of interpreting and managing the overlaps and differences between these disorders. While residue of this historic hostility still exists, the current conversation between the mood and personality disorder camps accommodates both recognition that (1) unipolar depression, bipolar disorder, and BPD are valid diagnostic constructs and (2) when considered as part of a mutually exclusive differential diagnosis, the use of these diagnostic categories tends to oversimplify the relationships between the underlying vulnerabilities, phenotypic features, and indicated treatments for patients presenting with complex comorbidity and/or atypical variations of these illnesses. As the chapters by Paris and Ghaemi/Barroilhet suggest, our current use of diagnostic constructs is limited and leads to reductive and simplified clinical management. This tendency contributes to misdiagnosis or ineffective prioritization of one diagnosis over another.

In an attempt to clarify what we now know about the overlaps and distinctions between mood disorders and BPD, the authors in this volume have reviewed the current literature on the clinical and neurobiological profiles, development, and course, as well as psychopharmacologic and psychotherapeutic interventions for these disorders. This review has traced five general conclusions from the current status of knowledge about the relationship between these disorders:

1. Depression and BPD phenotypically diverge yet are highly comorbid, suggesting overlapping underlying liabilities. Depression and BPD also interact significantly in longitudinal course.
2. Bipolar disorder and BPD phenotypically overlap yet are infrequently comorbid, suggesting more disparate etiologies. This leads to increased diagnostic confusion and misdiagnosis.
3. Depression, bipolar disorder, and BPD all involve the interaction between temperamental or trait-like features and acute episodic symptoms or state-like features.

4. Real-world clinical settings involve patients who may present with symptoms, precursors, and risk factors shared among mood disorders and BPD. Premature diagnostic certainty results in therapeutic overkill with overly specialized intensive treatment mismatched to presentations. Clinical interventions scaled with clinical staging considerations may reduce the tendency towards misdiagnosis and iatrogenic interventions.
5. Psychopharmacologic treatment is primary for depression and bipolar disorder and adjunctive for BPD. Psychotherapeutic approaches are primary for BPD and adjunctive for depression and bipolar disorder. A combination of approaches is indicated with comorbid or diagnostically unclear presentations, but further research is needed to determine the effectiveness of combined treatments and step-wise approaches to care.

The remainder of this summary will review and consider the evidence presented in this text supporting each of these conclusions.

## Depression and BPD: Superficially Divergent, Fundamentally Overlapping

Depression and BPD are clinically distinct disorders with divergent treatment strategies but appear to stem from shared underlying vulnerabilities. Depression is one of psychiatry's most prevalent disorders with heterogeneous variations that respond to a diversity of treatments. BPD is a specific and severe clinical syndrome, which was distinguished initially, per Choi-Kain and Rodriguez-Villa's historical review, by its lack of or negative response to typical treatments which were generally effective for a range of common mental disorders. A number of chapters (Goodman et al.; Yalch, Hopwood, and Zanarini; Silk) in this book highlight differences in clinical features of MDD and BPD. Silk and Goodman et al. characterize depressive features of individuals with BPD in terms of dysphoria, emptiness, loneliness, and fears of abandonment. In addition, core features of impulsivity and interpersonal sensitivity in BPD distinguish it from MDD. The severity of mood symptoms and degree of functional impairment in individuals with BPD exceeds that found in those with MDD. The limited response of BPD symptoms to antidepressants also suggests a significant clinical difference between MDD and BPD. These differences suggest that these disorders are distinct and not just reflections of each other.

At the same time, comorbidity between MDD and BPD is significant. A vast majority of BPD samples, that is, 70–80 % [4, 5], report comorbidity with MDD. Conversely 50–85 % of outpatients with MDD have personality disorders, of which BPD is the most prevalent [6]. Furthermore, family studies have established a significant risk for MDD in relatives of probands with BPD [7–9]. Although there are clear differences in clinical phenomenology and biological features which can differentiate these two diagnostic entities as noted in Goodman and collaborators' chapter (i.e., brain region involvement, neurohormonal indices, and sleep architecture), the

high rate of co-occurrence and familiality implies the existence of shared underlying liabilities between the two disorders. These underlying liabilities increase the likelihood of not only developing either MDD or BPD but also of developing both disorders comorbidly. In the last decade, research on broader underlying familial internalizing and externalizing dimensions of psychopathology has confirmed overlaps in latent liability factors that explained the co-occurrence of disorders [10–12]. Studies have indicated that BPD is associated with both internalizing and externalizing factors, which explains its complex comorbidity pattern [13, 14].

Skodol summarizes the literature on the longitudinal interactions between MDD and BPD in Chap. 10. His summary illustrates that the co-occurrence of BPD and MDD is associated with both slowed remission and increased relapse in both disorders, thereby contributing to a greater chronicity in each. The reciprocal interactions between the two disorders suggest there may be shared underlying factors that contribute to the persistence and recurrence of acute symptoms in both disorders. Additionally, Yalch, Hopwood, and Zanarini report that as MDD severity increases, its overlap with BPD increases.

While many possible explanations for the interaction between MDD and BPD have been proposed (see chapter by Goodman et al.), the current state of knowledge seems to support the following hypotheses: (1) MDD and BPD are manifestations of the same phenomenon, (2) MDD and BPD share common vulnerabilities, and (3) MDD and BPD share common biological features which foster each other's development [15, 16]. These hypotheses are not mutually exclusive. In order to refine our understanding of the complex relationship between the two disorders, it is necessary to first identify the shared liabilities and biological features.

Throughout this book, several authors have reported on the role of personality traits or temperamental endowments, such as negative affectivity, emotional dysregulation, and interpersonal hypersensitivity in the development of both mood and borderline personality disorders. Goodman et al. describe several biological characteristics including amygdala hyperreactivity, subgenual ACC volume changes, and deficient serotinergic function that are thought to underpin the emotional dysregulation seen in both MDD and BPD. Genetic findings point to serotonin, tryptophan hydroxylase, and monoamine oxidase systems as potential sources of the shared vulnerability towards altered processing of social and emotional information. Future research is needed to clarify the biological factors that may underlie the relationship between emotional and relational characteristics contributing to liability for both disorders.

In both the depression and BPD research literatures, interpersonal factors have been implicated as central vulnerabilities contributing to risk for developing psychopathology in the context of life stress [17–19]. Interpersonal features, such as attachment insecurity and rejection sensitivity, which have distinguished those with BPD from those without, appear to also be associated with MDD but at lesser degrees [20, 21]. Attachment insecurity and rejection sensitivity may be nonspecific features which confer risk to a number of disorders, but may be more severe and prevalent in individuals with BPD. The degree to which these interpersonal factors contribute to risk for BPD and its comorbidity as well as their relevant underlying biological mechanisms requires further study.

## Bipolar Disorder and BPD: Superficially Overlapping, Fundamentally Divergent

Several chapters in this text describe the overlap between bipolar and borderline personality disorders as more limited than the overlap between depression and BPD. All authors in this book agree that bipolar disorder and BPD are distinct disorders which both tend to be delayed in diagnosis and thereby delayed in being adequately treated. Impulsivity is a key feature shared by both disorders, but not considered to be at the core of either. Mood fluctuations are also shared by both disorders, but, as explicated in the chapter by Reich, the affective instability seen in BPD involves more shifts between anger, depression, and anxiety, whereas those seen in bipolar disorder involve more euphoria. Ghaemi and Barroilhet assert that a comparison of these disorders is akin to one of red skies and red apples, suggesting a relationship at a superficial level. Their superficial similarities lead to significant underdiagnosis of BPD with overdiagnosis of bipolar disorder as described by Zimmerman and Morgan in their chapter.

Chapters contributed by Ghaemi and Barroilhet as well as Reich outline important clinical and biological differences between BPD and bipolar disorder. Symptoms of dissociation, parasuicidal behavior, and recurrent deliberate self-harm distinguish borderline patients from bipolar patients. Bipolar patients are more likely to describe euphoric mood, increased goal-directed activity, and psychomotor agitation. Reich reports that the affective instability seen in both disorders stems from different neurobiological bases. Ghaemi and Barroilhet also emphasize the high rates of trauma history in BPD, arguing that environmental factors have a more significant effect on the development of BPD, whereas genetics contribute more strongly to the development of bipolar disorder. The low rate of co-occurrence and lack of influence on each other's course longitudinally further supports the notion that these are two distinct, unrelated disorders.

Taken together, the authors contributing to this text suggest that borderline personality and bipolar disorder are distinct and unrelated, but their overlaps in symptoms lead to problems of misdiagnosis rather than co-occurrence. Morgan and Zimmerman as well as Ghaemi and Barroilhet suggest using family history and trauma history as clinical indicators. These clinical features may lean practitioners towards either a bipolar or borderline diagnosis. In reality, when clinicians base their diagnostic impressions on self-report, diagnostic clarity remains at times elusive despite the current understanding of differences between these diagnoses.

A specific area of more murky differentiation exists between bipolar type II and borderline personality disorders. As Skodol proposes in his chapter, the overlaps between these disorders in the realm of interpersonal sensitivity, childhood trauma, and recurrent suicidality combined with the relatively weaker associations with family history of bipolar I and more variable treatment response to mood stabilizers point to the possibility that these two disorders may be more related than bipolar type I and borderline personality. The only longitudinal interaction between

bipolar disorder and BPD is that type II bipolar disorder slows time to remission of BPD. Skodol suggests that the combination of BPD and bipolar type II may represent a more severe variant of BPD. More research is needed to assess the relative relationship of bipolar II to both BPD and bipolar I.

## Temperament, Mood, and Personality: Models for Overlapping and Interactive Concepts

In both the mood and personality disorder literatures, researchers have been investigating the relationship between temperamental endowments, personality features, stressful life events (e.g., trauma), and psychopathology. As described by Lara et al., temperament is conceptualized as an innate disposition that influences basic emotional, behavioral, and cognitive responses. Mood is then expressed from a temperamental basis in response to external or internal stimuli. The position of personality in relation to temperament and mood is variable. Personality traits are similar to temperamental characteristics that are enduring and biologically based. However, like mood, personality is expressed in terms of the interface between temperamental characteristics and environmental exposures. Chapters by Lara et al. as well as Yalch, Hopwood, and Zanarini represent the dimensional and categorical approaches developed to assess and explain the relevance of temperament, mood, and personality to etiology and symptomatic manifestations of these illnesses.

A vast number of assessments and models of temperament and personality have been proposed and validated. Lara et al. present a complex framework combining dimensional and categorical models of temperament and personality, organized in a similar way to the proposed (and rejected) revisions to personality disorder diagnosis for the DSM-V. Lara's Affective and Emotional Composite Temperament (AFECTS) model integrates a number of emotional traits (e.g., volition/energy, drive, anger, fear, caution, emotional sensitivity, anxiety, control, coping, and stability) which represent neurobehavioral subsystems with four general categories of affective temperaments, which is divided into twelve global configurations. This complex AFECTS system allows clinicians and researchers to assess underlying emotional and temperamental factors associated with specific disorders in a finer grained fashion. Using this system, Lara and his collaborators are able to identify both the similarities and differences between depression, bipolar disorder, and BPD. All three disorders interface with characteristics of low volition, low coping, and high anxiety. BPD subjects maintain a profile of very high anger and desire as well as low coping and stability. Depressed subjects show lower anger and desire and higher coping and stability than those with BPD, but higher anger and desire and lower coping and stability than controls. These findings suggest that depression involves mild or moderate variations of features related to anger and coping, while BPD involves more severe variations. Similarly, BPD and bipolar subjects shared the same profile, but with higher anger scores distinguishing those with BPD from those with bipolar. In comparison to both mood disorders, BPD involves greater severity of dysfunctional traits.

These simple profiles derived from a complex system analyzing a wide number of temperamental and personality features allow clinicians to focus on key qualities, like high externalizing emotions and low self-regulating traits, as the organizing principles in treatment. However, this approach is limited by its lack of conceptual differentiation of disorders beyond an assessment of superficial description of traits. As noted, the differentiation of bipolar disorder from BPD is only by severity of anger, which may only perpetuate misdiagnosis and confusion between the two diagnoses. Treatment approaches tailored towards dimensional assessments of diagnosis have not been adequately proven, so the effectiveness of this approach in treatment of comorbid disorders is unclear. The clinical utility of dimensional and complex models such as Lara's requires further study.

Yalch, Hopwood, and Zanarini present a model of hyperbolic temperament in BPD, in which the tendency towards intense emotional responses is combined with heightened interpersonal sensitivity. They note that negative affectivity, or a heightened tendency to experience negative emotions, is a heritable, stable trait associated with both depression and BPD and might explain the high level of co-occurrence and familial co-aggregation of these disorders. They also assert that the impulsivity, emotional dysregulation, and interpersonal hypersensitivity characteristics of BPD distinguish it from depression. These three characteristics importantly interact, resulting in what Zanarini and Frankenburg have called "emotional hypochondriasis" defined as "the transformation of unbearable feelings of rage, sorrow, shame, and/or terror into unremitting attempts to get others to pay attention to the enormity of emotional pain that one feels" [22]. Impulsive behaviors function as a way to remedy intense emotional pain as well as communicate interpersonally a bid for help, engaging another person to help regulate emotions. This model, for which Yalch, Hopwood, and Zanarini offer some empirical support, identifies more than a set of characteristics in BPD. It provides a model for interactions and functions between elements of the BPD syndrome as well as a model of transactions between an individual's innate vulnerabilities and environment.

The model of hyperbolic temperament in BPD specifies both the overlaps and distinctions between BPD and depression as well as between acute and chronic symptoms of BPD. As Yalch, Hopwood, and Zanarini explain, the negative affectivity in depression confers a general vulnerability to develop negative emotions in response to stress, while in hyperbolic temperament, the vulnerability to intense negative emotion is developmentally rooted in and activated by interpersonal stress. Acute symptoms of BPD – that is, impulsive, self-destructive, and interpersonally focused behaviors – emerge episodically and remit, while temperamental symptoms persist, leading to chronic dysphoria and psychosocial dysfunction [23]. Negative affectivity, according to Yalch, Hopwood, and Zanarini, is a common factor driving vulnerability for and chronic features of both MDD and BPD, while more specific behavioral and interpersonal factors may differentiate manifestation of acute symptoms in the two disorders.

Both chapters represent different frameworks for understanding the interplay of dimensional temperamental and personality features in the development of the clinical presentations that are classified categorically as disorders. While Lara's model

provides a broadly applicable system of analysis used to understand a range of mood disorders in terms of personality features, it lacks a more theoretical formulation for the coexistence and interplay of these features. His model may help clinicians to identify specific features which can be targeted in diagnostically nonspecific therapeutic interventions (i.e., medications and cognitive behavioral therapy), but is largely empirical and descriptive. In contrast, the model described by Yalch, Hopwood, and Zanarini is more specific to BPD, a single disorder, but provides a formulation for how the symptoms of the disorder interact, thereby allowing clinicians to base their interaction with patients in treatment around not only a description of their problems but a theory about the nature and source of those problems. This transactional formulation of BPD is organized much like that of Linehan's biosocial theory [24] and Bateman and Fonagy's developmental theory of BPD [25], which explain how symptoms and underlying vulnerabilities interact. These theoretical understandings of BPD have been useful in developing organized psychosocial treatments.

Both approaches are necessary and limited. The more descriptive approach used in the mood disorder literature allows researchers and clinicians to identify stable temperamental and personality characteristics influencing vulnerability towards mood states and disorders, but does not provide a clear theory to organize therapeutic interventions. Importantly, these models and assessments appear to be effective in differentiating depression from bipolar disorder but less effective in differentiating bipolar disorder from BPD, leading the proponents of the bipolar spectrum to assume this means BPD represents a form of bipolar disorder. Transactional models, as represented by Yalch, Hopwood, and Zanarini, provide more elaborated theory of the interface between personality or temperamental features and symptomatic clinical features. However, these are far more specific to BPD as a single disorder and therefore limited in their utility for the generalist practitioner. In order to bridge the differences between the frameworks used in both realms of psychiatry, it will be important to standardize instruments and methodologies to relate research findings and test clinical applications. Further research is needed to understand the broad implications of temperament and personality in terms of liability for both mood disorders and BPD with emphasis on identifying systems of assessment which can be reliably and practically implemented in clinical settings. Special attention is needed to ensure that efforts to dimensionalize diagnostic assessments improve rather than undermine established treatment guidelines.

## Clinical Evaluation and Staging for Prescribing Interventions: Mitigating Premature Diagnostic Certainty and Therapeutic Overkill

The emergence of identifiable risk factors, precursors, and early symptoms of both mood and borderline personality disorders commonly occurs during the developmental period between adolescence and early adulthood. Chanen and Thompson

highlight the difficulty attaining diagnostic clarity in the face of evolving symptomatology that may be sub-syndromal and nonspecific. In their review of the literature, Chanen and Thompson report that childhood adversity – specifically childhood maltreatment, trauma or stressful life events, and socioeconomic disadvantage – increases risk for various psychiatric diagnoses. These factors in themselves are not differentiating in diagnosis. The early signs and precursors to mood and borderline personality disorders overlap significantly, which is consistent with what has been noted throughout this book about later stage and fully developed variants of these disorders. Bipolar disorder and BPD in younger patients present with risk factors and comorbidity such as childhood disruptive behavioral disorders (e.g., ADHD) and substance abuse as well as personality traits such as impulsivity and emotional dysregulation. Early-onset depression is common in both BPD and bipolar disorder; therefore, depression is not specific to either diagnostic entity. Hypomanic and depressive symptoms are common in this developmental period. Specifically, recurrence or persistence of symptoms, rather than single episodes of mood symptoms, is predictive of the development of psychiatric syndromes at clinically significant levels warranting diagnosis and intervention.

Chanen and Thompson acknowledge the need for early intervention in all diagnostic scenarios as delays in making a diagnosis of bipolar disorder or BPD necessarily delay the access to appropriate treatment. However, in the face of the usual clinical ambiguity commonly encountered in general practice, clinicians may be pressed to err on the side of either premature diagnostic certainty or delay in making proper diagnoses. Chanen and Thompson propose a clinical staging approach to accommodate the possibility of starting with an uncertain stance towards diagnosis which can be carefully refined with longitudinal clinical observation. Furthermore, Chanen and Thompson criticize the tendency in both child and adult clinical psychiatry settings to select the most intensive interventions as first line rather than those scaled towards clinical presentation. Their clinical staging approach provides lower intensity, broad interventions with specific indications for more intensive treatments which may be otherwise unclear in their indications, problematic in terms of side effect burden, or too resource intensive to be widely available to the public.

Chanen and Thompson's model of clinical staging can potentially mitigate premature diagnostic certainty and therapeutic overkill in both child and adult settings. As they note in their chapter, "[t]he reification of each separate syndrome leads to the implication that one clinician or another is missing an 'obvious case' and has foolishly applied the 'wrong' treatment or is denying much needed specific treatment" (166). This tendency leads to defensiveness and mistrust among clinicians as well as among patients and their families and presents added challenges to effective treatment regardless of ultimate diagnosis. Even when a proper diagnosis of BPD is made, the most intensive therapeutic approaches are often prematurely recommended, leading to misallocation of scarce treatment resources to those who can access it, rather than to those for whom intensive treatments are clinically indicated.

## Psychopharmacologic and Psychotherapeutic Interventions: Priorities and Compromises

Current psychiatric evidence and practice guidelines suggest the following: (1) bipolar disorder responds primarily to psychopharmacologic treatment, and psychotherapy is adjunctive; (2) BPD responds primarily to psychotherapeutic interventions, and psychopharmacology is adjunctive; and (3) depression responds to both psychopharmacology and psychotherapy. Our discussion in this book has helped us to arrive at some paradoxes in the relationships between these disorders. The first paradox is that while depression and BPD are highly comorbid and overlap in underlying liabilities and biological processes, BPD does not consistently respond to antidepressant medication. The second paradox is that while bipolar disorder and BPD are superficially similar and fundamentally different, many elements of their standard treatments (i.e., mood stabilizers and psychotherapy) overlap. Understanding the complexities of the relationships between these disorders in particular and between mood, personality, and temperament more broadly will enable clinicians to effectively map the shared territories among these clinical concepts and fashion an organized and flexible treatment plan.

Major depression is a heterogeneous disorder with multiple subtypes and responds to a number of interventions comparably, including placebo, St. John's wort, psychotherapy, and antidepressant medication [26, 27]. While depression by itself typically remits with a variety of treatments, it does not remit in cases of comorbid BPD until BPD improves [28]. Comorbidity with BPD may in itself be a marker of more severe and chronic psychopathology which confers increased risk for chronicity and recurrence of mood problems. As noted by Silk in his chapter, antidepressant medications may ameliorate typical symptoms of depressive episodes as a distal outcome of shared vulnerabilities between depression and BPD, but do not target underlying vulnerabilities towards negative affectivity, emotional dysregulation, and interpersonal sensitivity, which are liabilities increasing risk for both disorders. Careful assessment of what is meant by depression, as recommended by Silk in his chapter, must be assessed to guide the decision of whether or not medication is indicated. Psychosocial approaches target these underlying vulnerabilities more specifically, whereas antidepressants appear to relieve more superficial and episodic symptoms. Additionally, some evidence exists suggesting psychotherapy is more effective than antidepressant medication in the treatment of patients with depression and history of early life stress [29]. Underlying vulnerabilities and environmental stressors interact to increase risk for both depression and BPD; therefore, treatment should aim to address these factors, not just acute symptoms of depression or BPD.

A variety of intensive psychotherapeutic approaches designed specifically for BPD have been found effective in decreasing suicidality, self-harm, depressive symptoms, and utilization of acute medical and psychiatric services (see Gunderson et al. [33] for review). The most prominent of these – Dialectical Behavioral Therapy (DBT), Mentalization-Based Treatment (MBT), Transference-Focused

Psychotherapy (TFP), and Schema-Focused Psychotherapy (SFT) – involve at least three to five hours of treatment weekly in the formats they have been found effective. Currently, a number of less intensive psychosocial or clinical management interventions, which include cognitive behavioral therapy (CBT), supportive psychotherapy, structured clinical management (SCM), and General Psychiatric Management (GPM), have been found to be comparable in reducing symptomatology to the more intensive evidence-based modalities described above, but require less specialized training and are more generalizable to nonspecialist settings [30–33]. While some of these treatments may have less robust effects on reducing symptoms of BPD and depression, they are more practical as first-line interventions for patients with BPD and mood disorder comorbidity. There is also limited evidence that a generalist approach (i.e., GPM) may lead to lower rates of drop out in cases of axis I comorbidity compared to a more intensive treatment such as DBT [34]. More intensive treatments might be reserved for patients who fail to respond to these first-line interventions. Efforts to train mental health clinicians broadly in less intensive approaches for BPD are essential so that access to care is broadened. Lastly, research efforts are needed to clarify the effectiveness of step-wise approaches based on clinical staging as proposed by Chanen and Thompson that guide prescription and allocation of these forms of care.

The chapters on psychotherapeutic interventions for BPD and mood disorders included in this book focus on more generalizable flexible frameworks that can be widely disseminated to mental health clinicians of all disciplines and adjusted for a wide range of emotional problems. Jacob and Rodriguez-Villa describe the adaptation of CBT for a wide range of emotional problems. They identify interpersonal vulnerabilities and instabilities in self-awareness as key clinical features in BPD which limit the effectiveness of general CBT interventions aimed at specific anxiety or mood disorders. DBT and SFT provide important adaptations to the specific treatment challenges for clinicians working with patients with BPD. Attention to psychoeducation about the BPD diagnosis, strategies to stabilize and increase self-awareness (e.g., mindfulness and self-assessment), and a focus on interpersonal patterns are common features of evidence-based treatments (EBTs) for BPD which can be easily adapted into a more general CBT framework. More research is needed to test this adapted CBT approach in working with patients with mixed presentations of BPD and mood disorders.

Luyten and Fonagy describe the adaptation of psychodynamic approaches to patients with MDD and BPD, based on assessments of depressive features, mentalizing capacities, stability of attachment functioning, and capacities for epistemic trust. Like Jacob and Rodriguez-Villa, Luyten and Fonagy contend that reflective and relational capacities complicate general psychotherapeutic approaches. Luyten and Fonagy additionally note that treatments which presume a stable capacity for mentalization may be iatrogenic for patients with BPD. In their chapter, they present a spectrum of mentalizing approaches which can be flexibly applied to individuals with depression without BPD and those with both BPD and depression. For both categories of patients, mentalizing approaches ultimately focus on affective experiences in interpersonal contexts. However, intensive full-scale MBT is needed for

patients with BPD as special attention is needed for assessment and stabilization of mentalizing capacities and attachment activation. The MBT approach provides a generalizable treatment framework which can be adjusted for severity of reflective and interpersonal dysfunction; therefore, MBT may be an approach that can be adapted, like CBT, to a wider range of disorders.

The overlap between treatments for bipolar and borderline personality disorders is more superficial. Both disorders respond to mood stabilizers and atypical antipsychotics, but even in bipolar disorder where medications are primary interventions, a minority of patients achieve remission with these agents. While evidence exists for the efficacy of mood stabilizers in reducing a variety of symptoms relevant to BPD, the literature is both limited and inconclusive, lacking rationale to designate any indication for any specific medication. The underlying processes for these disorders are mostly divergent, although cyclothymic temperament may increase liabilities for both diagnoses, as noted by Choi-Kain and Rodriguez-Villa in their chapter. In general, the basic mechanism in bipolar disorder related to the development of manic states is most reliably responsive to mood stabilizers and not likely to respond to psychotherapy alone. Conversely, the core vulnerabilities to emotional dysregulation in the face of interpersonal hypersensitivity are more responsive to appropriate psychosocial intervention and unlikely to respond to medication alone. In both diagnoses, the standards for treatment of depressive states remain unclear. However, as several authors have noted throughout this book, depressive symptoms improve when BPD improves in comorbid states.

Psychosocial approaches are clearly indicated for BPD, but are adjunctive for bipolar disorder. Studies of psychotherapeutic approaches to bipolar disorder are limited but demonstrate a role for reducing relapse (particularly to depression) and improving functionality [35]. Jacob and Rodriguez-Villa identify psychoeducation, problem-solving, support, coping, and self-care skills as common features of validated psychotherapies for bipolar disorder and BPD. These features focus on enhancing self-awareness and interpersonal stability [35]. The techniques inherent in different psychosocial treatments for these various diagnoses appear to differ not in content but in organization around core vulnerabilities and symptomatic problems. This suggests that what may make treatments work is an integration of therapeutic technique with a clear theory of the essential nature of the patient's problems.

While psychopharmacologic interventions are necessary and critical to the management of mood disorders, their use is often accompanied by unrealistic expectations, regardless of the diagnosis. Studies on trends in management of psychiatric disorders demonstrate an increase in long-term use of antidepressant medications without adequate knowledge of the risks associated with more prolonged use [36, 37]. There is also evidence that antidepressant use without psychiatric diagnosis is also on the rise, despite controversy about their superiority over placebo for depression [38–42]. With more complex clinical presentations involving comorbidity, there is a tendency for polypharmacy, which is largely unguided by treatment algorithms or evidence. This pattern of increased polypharmacy without the constraints of clinical guidelines or evidence poses undue risk for side effects and drug

interactions in the face of unclear benefits [43]. Clinicians and patients alike would benefit from more tempered and realistic understanding of what pharmacologic treatments can offer, regardless of diagnosis.

Concomitantly, there has been a significant decline in the practice of psychotherapy by psychiatrists, likely due to changes in insurance reimbursement and the predominance of psychopharmacologic intervention in the field [44]. Problematically, studies suggest that psychiatrists specializing in psychotherapy primarily see patients who can self-pay, while those who primarily prescribe medications "shun delivery of psychotherapy altogether" [44]. Evidence suggests that patients prefer psychological treatments over pharmacologic treatments for a variety of diagnoses, including depression, bipolar disorder, and BPD [45]. Depression, bipolar disorder, and BPD all respond to psychotherapeutic interventions though access to specialized intensive treatment is limited. In this era of declining practice of psychotherapy by psychiatrists, structured clinical or general management approaches are needed to broaden access to care for patients with complex comorbidities, particularly those with BPD. More training is necessary to provide generalists with strategies to manage the complexities and comorbidities of patients with BPD.

## Current Status and Future Directions

In the last two decades, the scientific progress in understanding the boundaries and overlaps between mood and borderline personality disorders has been significant. This book represents an attempt to review that progress. What is clear is that the older strain of dialogue between the voices of the mood disorder and personality disorder camps has segued from a contentious debate to a parallel but marginally interactive inquiry about the relationship between personality, temperament, biological processes, diagnostic entities, and treatment. On both sides, the interaction between personality factors and depression is widely recognized. Significant investigations on how to model these relationship both empirically and theoretically have been pursued, but more effort for cross-pollination of these investigations is necessary, using more streamlined methodologies to link the findings in both arenas. In contrast, the efforts to incorporate BPD into the bipolar spectrum have been slowed by increasing evidence that these disorders are only superficially similar while they are etiologically and fundamentally distinct. While the overlap between bipolar type II and BPD needs to be clarified, the consensus in this book is that the current state of knowledge allows clinicians clear indices of differentiating these disorders.

However, despite the increasing clarity on the distinction between these disorders, clinicians routinely encounter evolving, atypical, subthreshold, comorbid cases which are inherently difficult to diagnose. Chanen's clinical staging approach provides a framework for guiding clinicians to scale their interventions for clinical severity, so that clinicians are not pressured into false diagnostic certainty for early stage or ambiguous cases. While the advance of research has established effective treatments for both mood and borderline personality disorders, many of these

treatments are heavy handed. Psychopharmacologic treatments are replete with side effects, risk teratogenicity, and can be lethal in overdose. Psychosocial treatments that are held to be the gold standard for BPD are both too intensive and specialized for most generalist mental health practitioners to administer, so clinicians and patients face a serious dearth of accessible treatment for this disorder. More effort is needed to develop and proliferate more flexible and less intensive treatments for BPD. Research on and training for more generalizable psychosocial approaches that clinicians can adapt to a variety of common and comorbid mood and personality problem, such as CBT and mentalizing treatments, is needed. These more generalizable approaches might focus on shared personality features such as emotional dysregulation and interpersonal sensitivity as broadly relevant factors that contribute to risk for developing psychiatric illness more generally and in its most severe form, BPD.

The controversy about the distinctions and overlaps between mood and borderline personality disorders has unfolded in the context of a greater landscape in psychiatry, where the limitations of descriptive approaches to diagnosis have been highlighted by the reality of pervasive comorbidity, atypical variants, and misdiagnosis. The DSM-V revision was organized initially with an ambitious move towards efforts to refine diagnostic systems based on etiological rather than descriptive factors; however, adequate scientific clarity could not be achieved to make that needed shift. The current status of this dilemma relevant to the subject of this book suggests that the effort to bridge and integrate the fields of scientific inquiry and treatment strategies between the mood and personality sectors of the field is a more immediate and practical possibility. This integration enables a synthesis rather than division of efforts to more properly and comprehensively understand and treat these disorders and the patients who have them.

## References

1. Stone MH. Assessing vulnerability to schizophrenia or manic-depression in borderline states. Schizophr Bull. 1979;5(1):105–10.
2. Akiskal H, Chen SE, Davis GC, Puzantian VR, Kashgarian MM, Bolinger JM. Borderline: an adjective in search of a noun. J Clin Psychiatry. 1985;46(2):41–8.
3. Gunderson JG, Elliott GR. The interface between borderline personality disorder and affective disorder. Am J Psychiatry. 1985;142(3):277–88.
4. Zanarini MC, Frankenburg FR, Dubo ED, Sickel AE, Trikha A, Levin A, et al. Axis I comorbidity of borderline personality disorder. Am J Psychiatry. 1998;155:1733–9.
5. McGlashan TH, Grilo CM, Skodol AE, Gunderson JG, Shea MT, Morey LC, et al. The Collaborative Longitudinal Personality Disorders Study: baseline Axis I/II and II/II diagnostic co-occurence. Acta Psychiatr Scand. 2000;102(4):256–64.
6. Sass H, Junemann K. Affective disorders, personality and personality disorders. Acta Psychiatr Scand Suppl. 2013;108(S418):34–40.
7. Links PS, Steiner M, Huxley G. The occurrence of borderline personality disorder in the families of borderline patients. J Pers Disord. 1988;2:14–20.
8. Zanarini MC, Gunderson JG, Marino MF, Schwartz EO, Frankenburg FR. DSM-III disorders in the families of borderline outpatients. J Pers Disord. 1988;2:292–302.

9. Silverman JM, Pinkham L, Horvath TB, Coccaro EF, Klar H, Schear S, et al. Affective and impulsive personality disorder traits in the relatives of patients with borderline personality disorder. Am J Psychiatry. 1991;148:1378–85.
10. Krueger RF, Caspi A, Moffitt TE, Silva PA. The structure and stability of common mental disorders (DSM-II-R): a longitudinal-epidemiological study. J Abnorm Psychol. 1988;107:216–27.
11. Kessler RC, Ormel J, Petukhova M, McLaughlin KA, Green JG, Russo LJ, et al. Development of lifetime comorbidity in the World Health Organization world mental health surveys. Arch Gen Psychiatry. 2011;68(1):90–100.
12. Kotov R, Ruggero CJ, Krueger RF, Watson D, Yuan Q, Zimmerman M. New dimensions in the quantitative classification of mental illness. Arch Gen Psychiatry. 2011;68(10):1003–11.
13. Roysamb E, Kendler KS, Tambs K, Orstavik RE, Neale MC, Aggen SH, et al. The joint structure of DSM-IV Axis I and Axis II disorders. J Abnorm Psychol. 2011;120(1):198–209.
14. Hudson JI, Javaras KN, Laird NM, VanderWeele TJ, Pope HG, Hernan MA. A structural approach to the familial coaggregation of disorders. Epidemiology. 2008;19(3):431–9.
15. Gunderson JG, Phillips KA. A current view of the interface between borderline personality disorder and depression. Am J Psychiatry. 1991;148:967–75.
16. Koenigsberg HW, Anwunah I, New AS, Mitropoulou V, Schopick F, Siever LJ. Relationship between depression and borderline personality disorder. Depress Anxiety. 1999;10(4):158–67.
17. Akiskal HS, McKinney Jr WT. Depressive disorders: toward a unified hypothesis. Science. 1973;182(4107):20–9.
18. Blatt SJ, Zuroff DC. Interpersonal relatedness and self-definition: two prototypes for depression. Clin Psychol Rev. 1992;12(5):527–62.
19. Gunderson JG, Lyons-Ruth K. BPD's interpersonal hypersensitivity phenotype: a gene-environment-developmental model. J Pers Disord. 2008;22.
20. Choi-Kain LW, Fitzmaurice GM, Zanarini MC, Laverdiere O, Gunderson JG. The relationships between self-reported attachment styles, interpersonal dysfunction, and borderline personality disorder. J Nerv Ment Dis. 2009;197(11):816–21.
21. Staebler K, Helbing E, Rosenbach C, Renneberg B. Rejection sensitivity and borderline personality disorder. Clin Psychol Psychother. 2011;18(4):275–83.
22. Zanarini MC, Frankenburg FR. Emotional hypochondriasis, hyperbole, and the borderline patient. J Psychother Pract Res. 1994;3:25–36.
23. Zanarini MC, Frankenburg FR, Reich DB, Silk KR, Hudson JI, McSweeney LB. The subsyndromal phenomenology of borderline personality disorder a 10-year follow-up study. Am J Psychiatry. 2007;164(6):929–35.
24. Linehan MM. Cognitive behavioral treatment of borderline personality disorder. New York: Guilford Press; 1993.
25. Bateman A, Fonagy P. Psychotherapy for borderline personality disorder: mentalization based treatment. Oxford: Oxford University Press; 2004.
26. Parker G, Manicavasgar V. Modelling and managing the depressive disorders: a clinical guide. New York: Cambridge University Press; 2005.
27. Gaynes BN, Warden D, Trivedi MH, Wisniewski SR, Fava M, Rush AJ. What Did STAR*D Teach Us? Results from a large-scale, practical, clinical trial for patients with depression. Psychiatr Serv. 2009;60(11):1439–45.
28. Gunderson JG, Morey LC, Stout RL, Skodol AE, Shea MT, McGlashan TH, et al. Major depressive disorder and borderline personality disorder revisited: longitudinal interactions. J Clin Psychiatry. 2004;65:1049–56.
29. Nemeroff CB, Heim CM, Thase ME, Klein DN, Rush AJ, Schatzberg AF, et al. Differential responses to psychotherapy versus pharmacotherapy in patients with chronis forms of major depression and childhood trauma. Proc Natl Acad Sci U S A. 2003;100(24):14293–6.
30. Davidson K, Norrie J, Tyrer P, Gumley A, Tata P, Murray H, et al. The effectiveness of cognitive behavior therapy for borderline personality disorder: results from the borderline personality disorder study of cognitive therapy (BOSCOT) trial. J Pers Disord. 2006;20(5):450–65.
31. McMain SF, Links PS, Gnam WH, Guimond T, Cardish RJ, Korman L, et al. A randomized trial of dialectical behavior therapy versus general psychiatric management for borderline personality disorder. Am J Psychiatry. 2009;166(12):1365–74.

32. Clarkin JF, Levy KN, Lenzenweger MF, Kernberg OF. Evaluating three treatments for borderline personality disorder: a multiwave study. Am J Psychiatry. 2007;164(6):922–8.
33. Gunderson JG, Weinberg I, Choi-Kain L. Borderline personality disorder. Focus. 2013;11: 129–45.
34. Wnuk S, McMain S, Links PS, Habinski L, Murray J, Guimond T. Factors related to dropout from treatment in two outpatient treatments for borderline personality disorder. J Pers Disord. 2013;27(6):716–26.
35. Lauder SD, Berk M, Castle DJ, Dodd S, Berk L. The role of psychotherapy in bipolar disorder. Med J Aust. 2010;193(4 Suppl):S31–5.
36. Mojtabai R, Olfson M. National trends in long-term use of antidepressant medications: results from the US National Health and Nutrition Examination Survey. J Clin Psychiatry. 2014;75: 169–77.
37. Fava GA, Offidani E. The mechanisms in antidepressant action. Prog Neuropsychopharmacol Biol Psychiatry. 2011;35(7):1593–602.
38. Mathew SJ, Charney DS. Publication bias and the efficacy of antidepressants. Am J Psychiatry. 2009;166(2):140–5.
39. Khan A, Bhat A, Kolts R, Thase ME, Brown W. Why has the antidepressant-placebo difference in antidepressant clinical trials diminished over the past three decades? CNS Neurosci Ther. 2010;16(4):217–26.
40. Kirsch I, Deacon BJ, Huedo-Medina TB, Scoboria A, Moore TJ, Johnson BT. Initial Severity and antidepressant benefits: a meta-analysis of data submitted to the Food and Drug Administration. PLoS Med. 2008;5(2):e45.
41. Fournier JC, DeRubeis RJ, Hollon SD, Dimidjian S, Amsterdam JD, Shelton RC, et al. Antidepressant drug effects and depression severity: a patient-level meta-analysis. JAMA. 2010;303:47–53.
42. Pagura J, Katz LY, Mojtabai R, Druss BG, Cox B, Sareen J. Antidepressant use in the absence of common mental disorders in the general population. J Clin Psychiatry. 2011;72(4): 494–501.
43. Mojtabai R, Olfson M. National trends in psychotropic medication polypharmacy in office-based psychiatry. Arch Gen Psychiatry. 2010;67(1):26–36.
44. Mojtabai R, Olfson M. National trends in psychotherapy by office-based psychiatrists. Arch Gen Psychiatry. 2008;65(8):962–70.
45. McHugh RK, Whitton SW, Peckham AD, Welge JA, Otto MW. Patient preference for psychological vs pharmacologic treatment of psychiatric disorders: a meta-analytic review. J Clin Psychiatry. 2013;74(6):595–602.