Integrating Early Intervention for Borderline Personality Disorder and Mood Disorders

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Abstract: Borderline personality disorder (BPD) has been demonstrated to be a reliable and valid construct in young people (adolescents and young adults). Both borderline- and mood-related psychopathology become clinically apparent from puberty through to young adulthood, frequently co-occur, can reinforce one another, and can be difficult to differentiate clinically. This Gordian knot of overlapping clinical features, common risk factors, and precursors to both BPD and mood disorders complicates clinical assessment, prevention, and treatment. Regardless of whether an individual crosses an arbitrary diagnostic threshold, a considerable proportion of young people with borderline- and mood-related psychopathology will develop significant and persistent functional, vocational, and interpersonal impairment and disability during this critical risk and developmental period. There is a clear need for early intervention, but spurious diagnostic certainty risks stigma, misapplication of diagnostic labels, inappropriate treatment, and unfavorable outcomes. This article aims to integrate early intervention for BPD and mood disorders in the clinical context of developmental and phenomenological change and evolution. “Clinical staging,” similar to disease staging in general medicine, is presented as a pragmatic, heuristic, and trans-diagnostic framework to guide prevention and intervention. It acknowledges that the early stages of these disorders cannot be disentangled sufficiently to allow for disorder-specific preventive measures and early interventions. Clinical staging defines an individual's location along the continuum of the evolving temporal course of a disorder. Such staging aids differentiation of early or milder clinical phenomena from those that accompany illness progression and chronicity, and suggests the application of appropriate and proportionate intervention strategies.

Keywords: adolescence, bipolar disorder, borderline personality disorder, clinical staging, depression, early intervention, prevention, risk factor

Seventy-five percent of all mental disorders have their onset by the age of 25 years. The peak period of onset for depression, bipolar disorder, and borderline personality disorder (BPD) occurs from puberty through to young adulthood.\(^1\)\(^-\)\(^5\) Mental disorders often do not present fully formed, however, and young people frequently present with evolving mixtures of symptoms. Our limited understanding of the prospective relationships between these often nonspecific symptoms and the major mental disorder syndromes implies a more complicated picture. Diagnostic precision is frequently possible only in retrospect. Nevertheless, even when a clear diagnosis cannot be made, the existence of distress and psychopathology can have negative developmental consequences for education, work, and family and peer relationships. This is especially so when treatment is delayed,\(^2\)\(^,\)\(^6\) which can risk persistent functional deficits.

Concordant with the nosology of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), discussion about the relationship between BPD and mood disorders has principally been described in terms of their phenomenology and treatment, rather than their etiology or pathogenesis.\(^7\) Most phenomenological studies have been undertaken in adults, when the disorders are “formed” and have mostly “run their course.” These studies have often used retrospective reports, thereby introducing recall bias, which makes the timing of the onset of symptoms or disorders uncertain.\(^2\) Many other studies have used clinical samples,\(^8\)\(^,\)\(^9\) leading to inflated levels of co-occurrence of psychopathology.\(^10\)
Clinical assessment tends to rely upon eliciting phenomenology and risk factors in the context of the patient’s life narrative. This article highlights the task of early detection and intervention for BPD and mood disorders in the real-world clinical context of rapid developmental changes, phenomenological instability and evolution, and the multiple clinical outcomes that can result from the same risk factors, especially environmental ones (i.e., multifinality). It principally draws upon available prospective, longitudinal data concerning the development of BPD and mood disorders, and suggests a pragmatic and heuristic early-intervention framework.

ANTECEDENTS AND EARLY SIGNS OF PSYCHOPATHOLOGY IN YOUNG PEOPLE

Borderline Personality Disorder
Although the consensus has been for many decades that personality disorder has its origins in childhood and adolescence, diagnosing personality disorder prior to age 18 years has been more controversial than diagnosing personality disorder in adults. Recent data suggest, however, that borderline personality and mood disorders in youth can be diagnosed with similar levels of confidence. BPD has been shown to be recognizable early in life, evolves continuously across the lifespan, and is more plastic than previously believed. BPD has similar reliability and validity in adolescence or adulthood and can be identified in day-to-day clinical practice. Moreover, it has incremental validity (unique explanatory value) over and above other mental state (Axis I) disorders in regard to psychopathology, functioning, suicidal ideation, and self-harm.

In fact, BPD is arguably better considered as a disorder of younger people, with evolving risks in childhood, a rise in prevalence from puberty, and a steady decline with each decade from young adulthood. Limited data suggest that BPD occurs in up to 3% of community-dwelling adolescents and young adults. BPD (or dimensional representations of BPD) in young people defines a group with high morbidity and potentially poor outcomes. BPD independently predicts current psychopathology, poor general functioning, poor self-care, and poor relationships with family, peers, and significant others. BPD also predicts poor outcomes prospectively over two decades. These outcomes include a future BPD diagnosis, increased risk for mental state disorders (especially substance use and mood disorders), interpersonal problems, distress, and diminished quality of life.

It is now clear that dimensional representations of personality pathology, including borderline personality pathology, have similar stability in adolescence and adulthood. The underlying dimensions of BPD features (conceptualized as impulsivity, negative affectivity, and interpersonal aggression) also appear to be moderately stable in children. Only the Children in the Community study data has specifically measured childhood or adolescent personality disorder features as predictors of later personality disorder over multiple waves of assessment from childhood to adulthood. In that study, childhood or adolescent personality disorder symptoms were the strongest long-term predictors of later DSM-IV Cluster A, B, or C personality disorders, over and above disruptive behavior disorders and depressive symptoms. Overall, the Children in the Community study data support a normative increase in BPD traits following puberty. These traits decline in early adulthood, in part due to socialization or maturation processes, revealing a group that is progressively deviant when compared with their peers and that perhaps corresponds more to the “adult” BPD phenotype. These data, supported by other studies, suggest that the adult BPD phenotype principally arises from the group of young people presenting with BPD features.

The heritability of BPD is estimated to be .67, which is akin to or higher than heritability estimates for other major psychiatric disorders. Childhood abuse or neglect, difficult family environment, and low socioeconomic status are all additional significant risk factors for developing BPD, but they are also risk factors for other personality pathology, along with many other psychiatric disorders, including mood and substance use disorders. Prospective, longitudinal data show that certain temperamental characteristics and early-onset mental state or behavioral phenomena that are analogous to characteristics of BPD are precursors to the emergence of the BPD phenotype, but they do not predict its onset with certainty. These phenomena include attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder, conduct disorder, substance use, depression, and self-harm, along with the features of BPD.

Example precursor signs and symptoms include maternal reports of childhood temperament, which are prospectively related to increased risk for adolescent or adult BPD up to 30 years later. Substance use disorders during adolescence, especially alcohol use disorders, also predict risk for young adult BPD, and disturbances in attention, emotional regulation, and behavior—especially the disruptive behavior disorders (ADHD, conduct disorder, oppositional defiant disorder) in childhood or adolescence—individually predict young adult BPD. Furthermore, findings from one study suggest that emotion-regulation and relationship difficulties might precede impulse-control problems in the development of adolescent BPD.

Self-harm is a central feature of BPD, and in retrospective reports from adults with BPD, self-harm commenced in childhood in over 30% of individuals and in adolescence in a further 30%. Yet, self-harm is surprisingly under-researched as a potential precursor to BPD. Self-harm is relatively common among youth and is associated with several clinical syndromes. Repetitive self-harm is less frequent,
however, and appears to differ from occasional self-harm. BPD can be diagnosed in the majority of female adolescent inpatients with self-harm, and it is more likely that adolescents will be diagnosed with BPD if they endorse both self-harm and suicide attempts, compared with individuals reporting either self-harm or suicide attempts alone. Also, the greater the number of BPD criteria in adolescents, the more likely they are to have engaged in self-harm or attempted suicide. Self-harm also occurs, however, in people with bipolar disorder, especially mixed states, although the diagnostic boundaries between mixed states and BPD are ill defined.

The findings outlined above demonstrate that BPD features can be detected at least from puberty onward, with reliability and validity that is comparable to other disorders. Nonetheless, BPD features are often heralded by, accompany, or follow signs and symptoms that are also associated with other “comorbid” mental state disorders, such as mood, anxiety, disruptive behavior, eating, and substance use disorders. All told, these signs and symptoms appear from childhood through to adolescence, and many of these look like aspects of the BPD phenotype and herald its later appearance in adolescence or emerging adulthood. However, the specificity of these factors for BPD, other personality disorders, or mental state disorders is limited.

**Bipolar Disorder**

Research on precursor signs and symptoms of bipolar disorder also shows considerable overlap with BPD in regard to the patterns and types of symptoms that occur prior to the onset of the full disorder. However, research on the early signs and symptoms of mental disorders tends to be confined to discrete “silos,” meaning that studies in young people that focus on bipolar disorder rarely measure BPD, and vice versa.

Advocacy for prevention and early intervention for bipolar disorder arises from concern about the consequences of delay in diagnosis and treatment, especially in view of the potential and specific neuroprotective properties of agents such as lithium. Around 70% of individuals diagnosed with bipolar disorder will experience their first symptoms before age 25, with the peak onset at age 17, but a formal diagnosis is often preceded by a lengthy delay. More than half of patients are diagnosed in the first year of illness, but some studies report that a diagnosis can take up to eight years following the first episode. There is also often considerable delay between the onset of bipolar disorder and the introduction of mood-stabilizing medication. Delayed treatment has been linked with unfavorable outcomes, such as cognitive and neurostructural evidence of neuropreservation, poor psychosocial adjustment, increased rates of hospitalization and suicide, substance use, forensic problems, and failure to achieve developmental milestones. When considered along with the potential neuroprotective effects that mood stabilizers might exert—limiting or averting structural brain changes associated with bipolar disorder—the rationale for early intervention is strong.

Offspring of individuals with bipolar disorder are more liable to develop a variety of mental disorders, especially mood disorder. In the Dutch bipolar offspring study 12-year follow-up, participants meandered over time between asymptomatic, subthreshold anxiety, substance abuse, and depressive and bipolar-like phenotypes, with very poor temporal diagnostic stability. It is noteworthy that this study did not even measure personality disorder as an outcome. Yet, many of the risk factors associated with BPD are also risk factors for a later diagnosis of bipolar disorder. These factors include childhood or familial ADHD, traumatic or stressful life events and childhood abuse, and substance abuse. Certain personality traits, such as high harm avoidance and high novelty seeking, as well as impulsive aggression, are also associated with later bipolar disorder.

A review of 13 retrospective and 12 prospective studies covering the period prior to the onset of first-episode mania highlighted that depression is more commonly the initial polarity of first-presentation illness. Retrospective studies have pointed to subthreshold mania, irritability and anger, lack of sleep, grandiosity, periods of depression, and mood changes (to a smaller extent). Prospective studies highlight racing thoughts, irritability, anger, periods of depression, mood swings, anxiety, and, in some cases, psychotic symptoms. In these studies the prodromal period ranged from weeks to 15 years, in part due to study design, because studies that use “enriched” bipolar risk samples, compared with general samples, report a shorter time to transition. The symptoms of irritability, anger, depression, and mood changes are also present in BPD. The distinguishing symptoms for bipolar disorder were hypomania, grandiosity, and sleep disturbance.

The largest and most rigorous study to examine the incidence of hypomanic and depressive symptoms prospectively from adolescence through to adulthood drew a random representative sample of 14- to 24-year-olds living in Munich, Germany. The findings show that hypomanic and depressive symptoms were commonly experienced once (almost 40% of 1565 participants) over the follow-up period. Multiple experiences of these symptoms, however, were less likely. The persistence of symptoms was predictive of transition to clinically relevant outcomes (i.e., hypomanic episodes or accessing mental health care) in a dose-dependent manner. Clinical bipolar disorder is relatively rare and might be seen as the poorest outcome of these developmental processes. The study’s authors suggest that a nonclinical bipolar phenotype, usually transitory, might be developmentally common during adolescence. As with BPD, the period between adolescence and young adulthood thus sees the emergence of affective and behavioral psychopathology that is of uncertain clinical significance. It is the quantity, persistence, and qualities of this psychopathology that become important in distinguishing between syndromes.

The Course and Outcome of Bipolar Youth study followed 413 7- to 17-year-olds with “bipolar spectrum” (bipolar I, II, or not otherwise specified [NOS]) disorder prospectively over
a four-year period. This study was designed to measure affective symptoms (i.e., mania and depression) but not features of other disorders, such as BPD. The study found that the greatest proportion of symptomatology was due to mixed/cycling and depressive symptoms. The study frequently found rapid mood changes, and almost all of the chronic symptoms reported were subsyndromal depressive symptoms. The study’s authors stress that early onset gives rise to a greater likelihood of a chronic and fluctuating course. While they argue that their findings support the presence of clinically relevant, brief episodes of manic or hypomanic symptoms, the failure to measure BPD or other personality pathology as an outcome is a substantial limitation.

Although affective dysregulation is characteristic of both bipolar disorders and BPD, some evidence suggests differences in terms of goal regulation, threat sensitivity, and emotion-relevant impulsivity. In a sample of college students, elevated risk for mania was associated with greater reward sensitivity and intense goal pursuit. By contrast, risk for BPD was associated with impulsivity driven by negative affect and sensitivity to perceived threat.

Some authors have proposed that bipolar disorder in children differs from that in adults by its non-episodic nature and the far greater presence of irritability, rather than mania, which has led to changes in diagnostic practices in the United States, in particular, and to a substantial increase in diagnoses of child and adolescent bipolar disorder. In reaction to this increase, Leibenluft reviewed the link between mood dysregulation, irritability, and bipolar disorder in young people. Overall, she concluded that children presenting with non-episodic severe irritability do not have mania; in studies of youth with severe mood dysregulation, 84% also met criteria for lifetime oppositional defiant disorder, 86% for lifetime ADHD, 58% for lifetime anxiety disorder, and 16% for lifetime major depressive disorder (MDD). She also noted that young people with irritability do not have high rates of bipolar disorder in their families and that, compared with young people with bipolar disorder, they have a different pathophysiology. Finally, she concluded that severe mood dysregulation does not lead to bipolar disorder. Rather, irritability predicts adult unipolar depression and anxiety disorders. These syndromes have been previously noted to comprise precursor signs and symptoms of BPD. Moreover, mood dysregulation and irritability are also characteristic of BPD, which was not measured in the cited studies.

These studies highlight the problem that the symptoms which precede first-episode mania have poor sensitivity and specificity. Depressive symptoms have high sensitivity but low specificity for bipolar disorder, whereas low-grade elevation of mood is more specific but is not present in all young people who will go on to develop bipolar disorder. Bechdolf and colleagues developed a “close-in” strategy (combining identified risk factors to “close in” on the target population) in order to balance the need for sensitivity and specificity. They evaluated the validity of Bipolar At-Risk (BAR) Criteria (defined as subthreshold mania, depression plus cyclothymic features, or depression plus first-degree relative with bipolar disorder) in young people aged 15 to 24 years presenting for intake assessment at a psychiatric service with nonpsychotic disorders. In their sample, 22.7% of the BAR group developed bipolar disorder, compared with only 0.7% in the non-BAR group. BAR criteria appear to have some predictive validity in the proximal prodrome of bipolar disorder. The authors are currently testing these criteria in a new prospective study. A comparison of the “signature” of bipolar and unipolar depression suggested that features of psychomotor retardation, melancholia, and atypical depression were more common in bipolar disorder and had some predictive capacity.

The above studies suggest that BPD and bipolar disorder share numerous distal risk factors and precursor signs and symptoms. Even specific childhood and adolescent bipolar disorder symptoms, such as depression, irritability, mood changes, and an episode of hypomania, do not robustly predict the development of bipolar disorder per se. Some of these precursors for bipolar disorder overlap with the precursors for BPD. Strategies to identify young people at risk of developing bipolar disorder have had some success in closing in on the risk factors associated with the onset of bipolar disorder in young people, but these strategies require further investigation.

**Unipolar Depression**

There is a marked rise in the incidence of depressive symptoms during and after the pubertal period. Twenty percent of young people will have a diagnosable depressive episode prior to 18 years of age. Half of all first depressive episodes occur during adolescence, with an average onset of 15 years of age. Depression during this developmental period has also been associated with greater risk for subsequent episodes and a more chronic course. Similar to BPD and bipolar disorder, depression in adolescence is associated with unfavorable long-term functional and psychopathological outcomes that include impairments in education, vocation, interpersonal relationships, substance use, and suicide.

Various retrospective studies have investigated risk factors and other characteristics of individuals whose first depressive episode occurred during adolescence. These studies require cautious interpretation—first because of the potential for recall bias, and second because of the marked heterogeneity of the depressive construct. A study of 198 depressed women attending primary care practices found that those whose first episode of depression occurred prior to age 16 years were more likely to have had a suicide attempt, self-harm, a history of alcohol abuse, teenage pregnancy, pervasive personality dysfunction, inattention and hyperactivity, and poor relationships with their peers. This group was also more likely to have been exposed to poor parental care, physical abuse, childhood sexual abuse, and interpersonal violence. Another study compared 372 adult patients with depression according to whether their first depressive episode occurred in childhood,
adolescence, or adulthood. The group reporting adolescent-onset depression was significantly more likely to meet criteria for personality disorders. Avoidant personality disorder was the most common, followed by BPD.

There is notable overlap among risk factors for depression, BPD, and bipolar disorder, including substance use, social disadvantage, poverty, exposure to violence, childhood maltreatment, inter-parental conflict, parental overinvolvement, and parental aversiveness. The basis for claims of specificity for risk factors for youth depression (e.g., having a parent with a depressive illness, low self-esteem, female sex, negative body image, poor social support, and ineffective coping) seems to be weak; for example, parental depressive illness is also associated with later bipolar or anxiety disorders. Likewise, many protective factors, such as healthy diet and sleep, the presence of supportive adults, strong family and peer relationships, coping and emotion-regulation skills, and focusing on age-appropriate developmental tasks, appear to be nonspecific.

Vulnerability to depression is associated with individual differences in temperament, such as high negative emotionality, which prospectively predicts depression. Furthermore, personality disorder and depression have been shown to be mutually reinforcing throughout adolescence and young adulthood. For example, BPD features identified between ages 14 and 22 years were prospectively associated with dysthymic or MDD at mean age 33 years, even after adjusting for a history of unipolar depression or other psychiatric disorder.

The evidence above suggests that it is not surprising that unipolar depression shares many risk factors and precursor signs and symptoms with BPD and bipolar disorder, as well as other disorders, such as substance use. Depressive symptoms are common among adolescents and lead to outcomes that might include depression, BPD, or bipolar disorder but that might also include good mental health. Crucially, the initial polarity of first-presentation bipolar disorder is most commonly depressive, making treatment initiation (especially pharmacotherapy) for first-presentation depressive illness clinically challenging. Compounding the challenge is the heterogeneous nature of the current definition of depression, which incorporates diverse and, arguably, poorly specific forms of dysphoria.

**CLINICAL “COMORBIDITY” AND BORDERLINE PERSONALITY DISORDER**

Mental state disorder, personality traits, and personality disorder are significantly overlapping constructs. The frequency and extent of co-occurring mental state and personality pathology present challenges for clinical diagnosis. This is especially true for BPD, where the phenotypic expression of personality disorder commonly includes varying forms of dysphoria, such that co-occurring mental state and personality disorder are the norm. In a US, nationally representative survey of adults aged 18 years and over, 84.5% of those with BPD met criteria for at least one mental state disorder in the previous 12 months, with the mean number of mental state disorders being 3.2. Conversely, 25.2% of individuals who had a mental state disorder in the previous 12 months also met criteria for at least one personality disorder. In addition to being an artifact of a symptom-based diagnostic system, this co-occurrence might be a predictable consequence of the involvement of common risk and vulnerability factors for multiple disorders.

This pattern of co-occurrence of mental state and personality disorder has been found to be similar in young people in community and clinical settings. The Children in the Community study found that the long-term outcomes for mental state and personality disorders were of similar magnitude and often additive when they co-occurred. Both mental state (mood, anxiety, disruptive behavior, and substance use disorders) and personality disorders in adolescence showed risks for adverse outcomes for 20 years following first assessment. Co-occurring mental state and personality disorders consistently presented the greatest risk, which was at least the sum of the risks for both mental state and personality disorders but could also be several times the risk for either disorder alone.

In a comparison among adolescent outpatients with BPD, with other personality disorders, and with no personality disorder, those with BPD had the greatest burden of co-occurring mood (59%), anxiety (46%), disruptive behavior (70%), and substance use (35%) disorders. The same was true for female adolescent inpatients with BPD, where the most frequent co-occurring mental state disorders were mood (22%), eating (16%), dissociative/somatoform (13%), and substance use (10%). Thirty-nine percent of patients also had one or more co-occurring personality disorders, most commonly Cluster C (avoidant, dependent, obsessive-compulsive), followed by Cluster A (paranoid). These studies show that the normative clinical presentation of BPD is the presence of dysphoric anxiety, affective dysregulation symptoms, and other psychopathology.

Both the prominence of affective criteria and the co-occurrence of affective disorders (including bipolar I, bipolar II, MDD, and dysthymic disorder) in patients with BPD have driven debate about whether BPD should be viewed as a mood disorder. Relevant literature will be covered relating to this issue in young people.

**Borderline Personality Disorder and Bipolar Disorder**

One source of clinical controversy regarding BPD and bipolar disorder might be the diagnostic criteria themselves. Many of the criteria for BPD and bipolar disorder in DSM-5 and its forerunners are related to mood instability. A comprehensive exploration of the hypothesis that BPD is actually a bipolar spectrum disorder concluded that, more frequently than not, BPD remains both cross-sectionally and temporally distinct from bipolar disorders. Others have argued that the debate about overlap is scientifically false because mood lability and impulsivity are neither specific nor fundamental to either disorder. Proponents of this latter argument suggest that bipolar
disorder is principally a disorder of psychomotor activation and that the BPD criteria of abandonment, identity disturbance, recurrent suicidal or self-mutilating behavior, and dissociative symptoms distinguish BPD from bipolar disorder.

Others have highlighted that affective changes have a different time course and quality in BPD compared with bipolar disorder. In contrast to the presence of distinct and relatively persistent blocks of affective symptoms and a slower time course of affective change in bipolar disorder, BPD affect is subject to rapid and chaotic changes over minutes, hours, or days, more usually shifts between euthymia and anger, and is often triggered by environmental (especially interpersonal) factors.

Little research actually supports the hypothesis (largely based on the observation of unstable mood) that BPD is a bipolar spectrum disorder. One study of 87 depressed young people, recruited from consecutive referrals to a psychiatric clinic, aimed to compare BPD pathology during an index depressive episode in three diagnostic groups: bipolar disorder (n = 14), “bipolar spectrum disorder” (n = 27), and MDD (n = 46). None of the participants met full diagnostic criteria for a personality disorder. Compared with the MDD group, both of the bipolar-depressed groups reported significantly higher median levels of borderline characteristics. Three borderline characteristics were proposed as potentially useful for differentiating bipolar depression from unipolar depression: “I have tantrums or angry outbursts”, “I’ve never threatened suicide or injured myself on purpose”, “I have thoughts of suicide but no plan”, and “Giving in to some of my urges gets me into trouble.” The authors concluded that certain BPD screening questions—which they believe reflect cyclothymic characteristics or depressive mixed states—might be useful for differentiating bipolar from unipolar depression in young adults, and that BPD in early-onset depression is predictive of ultimate bipolar outcome. This study had significant limitations, however, including the reliance on the International Personality Disorders Examination screening questionnaire (which does not perform well in young outpatients), the absence of any case-level BPD, and the use of nonvalidated diagnostic criteria for “bipolar spectrum disorder.”

Another study that investigated 100 adolescents and young adults (aged 15–36 years) with early onset of bipolar disorder found that a co-occurring diagnosis of BPD significantly increased the risk for self-harm but not suicide attempts. The most important finding from this study was that, compared with later onset, early onset of bipolar disorder was associated with the highest risk of self-harm and suicide attempts. This finding is supported by data showing that anxiety, concentration difficulties, antisocial behavior, and substance use are present in the early stages of bipolar disorder and are associated with an unfavorable course.

Fundamental to the need to distinguish BPD and bipolar disorder is the ability of clinicians to disentangle symptoms and to actively investigate whether either disorder might be present. Failure to look for BPD in adults presenting with mood symptoms might be seen as a form of diagnostic myopia that risks favoring bipolar disorder diagnosis over BPD. This problem is potentially amplified by treatment-seeking patients’ greater awareness of bipolar disorder compared with BPD, and also by the greater stigma of BPD, especially among clinicians. The problem appears to be exacerbated in young people because of clinicians’ reluctance to make, or even antagonism toward making, the diagnosis of BPD among adolescents. Reasons cited in defense of this failure include the mistaken beliefs that personality pathology is transient or normative among young people, or that the diagnosis is not allowed in adolescence. While some of these concerns can be moderated by evidence, the diagnosis is undoubtedly stigmatizing, and stigma remains a key barrier to clinicians’ acceptance of the BPD diagnosis in young people.

These studies imply that assessment should consider all available clinical data; the particular character and context of mood-regulation difficulties are critical to distinguishing these disorders from one another and to initiating disorderspeciﬁc treatments. Certain characteristics might be especially salient, such as mood elevation, atypical depressive features, or alternating, persistent and stable blocks of psychomotor activation/retardation for bipolar disorder, or abandonment, identity disturbance, recurrent suicidal or self-mutilating behavior, and dissociative symptoms for BPD. Co-occurrence of bipolar disorder and BPD is also possible and appears to heighten the risk of self-harm and suicide attempts.

Borderline Personality Disorder and Unipolar Depression

DSM-5 includes suicidal ideation or attempts in the diagnostic criteria for both BPD and MDD. The high frequency of these and other depressive symptoms in young patients with BPD can contribute to a hasty diagnosis of depression or uncertainty about the presence of personality disorder.

A great deal of research has been conducted over many decades on the subject of BPD and depression in adults. When the two syndromes co-occur in adults, BPD negatively influences both the functioning of depressed patients and the risk for the relapse, recurrence, and new onset of depression. This subject has received comparatively little attention in young people, however, especially in clinical samples, and it is unclear how the findings in adults with BPD might apply to young people. A differential diagnosis of BPD needs to be entertained in any young person presenting with depression and vice versa, but diagnosing BPD is uncommon not only because of clinicians’ reluctance or antagonism concerning the diagnosis, as mentioned above, but also because of the lack of developmentally appropriate diagnostic criteria for BPD in the major diagnostic manuals. As with the comparison between bipolar disorder and BPD, it is possible that the nature of affective symptoms is different in young people with BPD, compared with their adult counterparts, and the relevant differences are likely to have significant consequences for both treatment and prognosis.

In a comparison of depressed adult patients with and without BPD, the Rhode Island Methods to Improve Diagnostic...
Assessment and Services study found that depressed patients with BPD had a younger age of onset, more depressive episodes, a greater likelihood of experiencing atypical symptoms, a higher prevalence of comorbid anxiety and substance use disorders, and more previous suicide attempts. This picture is similar to that for comparisons of bipolar depression to unipolar depression (e.g., Mitchell et al. and Akiskal et al.) and suggests that many of the features used to validate the bipolar spectrum are not specific to bipolar disorder.

Studies in adults with BPD also provide some insight into how the nature of depression in this group might differ from MDD. The McLean Study of Adult Development studied the time to remission of 24 BPD symptoms in a sample of 290 patients over a ten-year period. Symptoms reflecting affective instability, impulsivity, and some interpersonal difficulties, such as stormy relationships, showed patterns of sharp decline over time. By contrast, affective symptoms reflecting chronic dysphoria, intense anger, and interpersonal symptoms, such as intolerance of aloneness or abandonment fears, were more stable, albeit still showing substantial declines over the follow-up period. The more stable symptoms included chronic feelings of depression, helplessness, hopelessness, worthlessness, guilt, anger, anxiety, and loneliness, which overlap with the diagnostic criteria for MDD. The authors suggest that it is the chronic nature of these symptoms and their resistance to change that causes poor psychosocial adjustment in BPD. Importantly, this study showed that, unlike MDD, chronic dysphoria in BPD occurs in the context of symptoms reflecting abandonment and dependency issues, such as intolerance of aloneness, chronic anger, and dependency. Equally, depression in bipolar disorder has been shown to have subtle differences from unipolar depression, with the former including more atypical features (especially hypersomnia), a seasonal pattern, abrupt onset and offset, less tearfulness, and fewer somatic symptoms.

A qualitative study of seven individuals with BPD suggests that although chronic dysphoria in BPD appears to mimic depression, it might actually comprise an undifferentiated set of painful negative affects related to emotional suffering, rather than sadness. These affects occur in the context of aggression or the breaking off of a relationship (by another person), are accompanied by reactive aggressive impulses, but do not appear to involve an explicit articulation of loss.

It is unclear how these findings relate to young people with BPD. The link between dysphoria and aggression, however, is supported by a nonclinical study of 226 adolescent and young adult students. In the young adult students, maladaptive emotional coping significantly mediated the relationship between borderline pathology and reactive aggression, and maladaptive avoidance coping mediated the relationship between borderline pathology and proactive aggression. Another study of 196 girls (mean age = 10 years) found that borderline pathology is associated with aggression as a strategy to cope with the overwhelming experience of intense negative affect associated with stressful peer interactions.

In short, while there is considerable phenomenological overlap between BPD and MDD, understanding symptom relationships in the early stages of these disorders is hampered by a lack of evidence. Preliminary evidence from samples of adults with BPD suggests that the chronic dysphoria that characterizes BPD has a longer time course and occurs in a relational context; rather than sadness, it might actually be overwhelming negative affect that is externalized as irritability and aggression. This interpretation has received some support in community samples of young people.

**CLINICAL STAGING: A PRAGMATIC HEURISTIC FRAMEWORK FOR CLINICAL INTERVENTION**

In adult psychiatric practice, debate has been previously framed around the underrecognition of bipolar disorder or BPD in people presenting for treatment of depression. The reification of each DSM-5 syndrome leads, in turn, to situations that clinicians might be missing obvious cases or foolishly applying the wrong treatment or withholding much-needed treatments.

A crucial problem in youth is the disproportionate focus placed upon bipolar disorder, unipolar depression, and BPD as separate domains of risk, especially in retrospective studies. For example, in both bipolar disorder and BPD, patients might present as depressed, experience mood changes, have early age of onset and a history of abuse, engage in substance abuse, impulsive behaviors, and self-harm, and have other comorbid disorders. Youth frequently present clinically with blends of symptoms in the context of a dynamic, evolving, and unpredictable clinical picture. In that context, especially when symptoms are subthreshold, early application of a concrete and specific diagnosis might be inappropriate. The need for some form of treatment, however, often precedes reaching a diagnostic threshold, and intervention often cannot wait until the trajectory of these symptoms, with time, eventually becomes clear.

Another fundamental issue is that interventions (usually pharmacotherapeutic) for adult disorder phenotypes are often used as first-line interventions in youth, whereas psychosocial interventions are largely deemphasized or ignored. This focus on medication is implicit in discussions of potential interventions for mood dysregulation and irritability, and has been explicit in discussions about the use of mood-stabilizing medications for pediatric bipolar disorder. Timely use of mood-stabilizing medications is desirable because delay might diminish potential neuroprotective effects, and the presence of any active symptoms disrupts developmental trajectories. There is a risk, however, that premature use will medicate what might be a developmentally common, and usually short-lived, nonclinical bipolar phenotype. And whatever the reasons for introducing such medications, they might entail longer-term harms that might outweigh any benefits for young people, especially second-generation antipsychotics. What is absent from this calculus is a thorough evaluation of the potential role for less “toxic” interventions, especially psychosocial interventions.
Keeping the risks of treatment delay in mind, psychosocial interventions such as mental health literacy, family and individual psychoeducation, parenting skills and family interventions, substance use reduction, supportive counseling, and problem solving provide valid, but generic, methods for moderating the risk factors for progression to diagnosable disorder (outlined earlier) that are common to BPD and mood disorders, and for potentially averting significant disruption to normal development caused by mental disorders in this age group.

An alternative approach to focusing on diagnostic categories to guide prevention and early intervention in youth is to develop a range of risk syndromes, or warning signs for particular types of disorders. Fundamental to this cross-diagnostic, “clinical staging” approach is eschewing diagnostic categories and arbitrary age restrictions in favor of a focus on the severity and persistence of symptoms, the need for care, and the proportionality of any intervention. Once a convincing clinical phenotype has evolved, appropriate specific therapy can be initiated.

Clinical staging in the assessment of, and intervention for, mood disorders and BPD is illustrated in Table 1. This model outlines a response to each stage of disorder that is proportionate to the presenting clinical picture. Proposed interventions are simpler and more benign during early stages of disorder (Stages 0 and 1), and increase in intensity (and potential adverse effects) with disorder progression. In later-stage disorder (Stages 3 and 4), the risk of more severe adverse effects potentially outweighs the risk of not treating the disorder. Interventions can also be combined when syndromes are found to genuinely co-occur.

In this model, Stage 0 comprises asymptomatic individuals with increased risk of disorder. Appropriate interventions might include health education campaigns or self-help interventions regarding substance use, diet and healthy weight, negative coping strategies, and sleep patterns. Stage 1 comprises nonspecific or subthreshold BPD, potentially with hypomanic or depressive symptoms. Later stages would be characterized by the first onset of a disorder, by subsequent relapses and remissions, or by co-occurrence of other disorders (e.g., BPD or MDD).

While generic psychosocial interventions apply to the early stages of this model, more specific psychosocial interventions are proposed for early stages of disorder, including the Helping Young People Early (HYPE) program for BPD and psychosocial interventions for bipolar disorder and unipolar depression. Low-toxicity, novel pharmacotherapies might also be appropriate for Stages 1b and 2. Examples include N-acetylcysteine for bipolar disorder, substance abuse, and

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### Table 1

<table>
<thead>
<tr>
<th>Clinical stage</th>
<th>Definition</th>
<th>Potential interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Increased risk of severe mood disorder or borderline personality disorder (e.g., family history, exposure to abuse or neglect, substance use). No specific current symptoms</td>
<td>Mental health literacy Self-help</td>
</tr>
<tr>
<td>1a</td>
<td>Mild or nonspecific symptoms of mood disorder or borderline personality disorder (e.g., disturbances in attention, emotion regulation, and behavior)</td>
<td>Formal mental health literacy; family psychoeducation, parenting skills; substance abuse reduction; supportive counseling/problem solving</td>
</tr>
<tr>
<td>1b</td>
<td>Subthreshold features of mood disorder or borderline personality disorder</td>
<td>Stage 1a interventions plus phase-specific psychosocial intervention (e.g., cognitive-behavioral therapy, HYPE early intervention for borderline personality disorder)</td>
</tr>
<tr>
<td>2</td>
<td>First episode of threshold mood disorder or borderline personality disorder</td>
<td>Stage 1b interventions plus case management, educational/vocational intervention/rehabilitation, family psychoeducation and support, specific time-limited psychotherapy, specific and targeted pharmacotherapy (e.g., mood stabilizer)</td>
</tr>
<tr>
<td>3a</td>
<td>Recurrence of subthreshold mood or borderline personality disorder symptoms</td>
<td>Stage 2 interventions plus emphasis on maintenance medication and psychosocial strategies for full remission</td>
</tr>
<tr>
<td>3b</td>
<td>First threshold relapse of mood disorder or borderline personality disorder</td>
<td>Stage 3a interventions plus relapse-prevention strategies</td>
</tr>
<tr>
<td>3c</td>
<td>Multiple relapses of mood disorder or borderline personality disorder</td>
<td>Stage 3b interventions plus combination of mood stabilizers and intensive psychosocial interventions (e.g., dialectical behavior therapy)</td>
</tr>
<tr>
<td>4</td>
<td>Persistent, unremitting disorder</td>
<td>Stage 3c interventions plus clozapine and other tertiary therapies, and social participation despite disability</td>
</tr>
</tbody>
</table>

Adapted from Berk et al. (2013) and McGorry et al. (2006).
depression,133–135 and omega-3 fatty acids, which evidence suggests might be useful in both mood disorders and BPD from Stage 2 onward.136–138

This clinical-staging model for mood disorders and BPD will necessarily evolve and become more sophisticated with increased knowledge about the disorders’ developmental pathways (including likely biological and endophenotypic markers), potentially also leading to novel interventions. The model provides a starting point for developing more nuanced diagnoses and treatments, and it constitutes, in effect, an algorithm for determining how to intervene early in the course of these disorders in young people.

INTEGRATING BPD INTO PREVENTION AND EARLY-INTERVENTION STRATEGIES FOR MOOD DISORDERS

BPD in adolescents and young adults appears to be as reliable and valid a disorder as many other major psychiatric disorders. Both borderline and mood-related psychopathology become clinically prominent from puberty through to young adulthood, overlap phenomenologically, and frequently co-occur. BPD and mood disorders share numerous common risk factors and precursors, complicating this aspect of clinical assessment. Although the outcomes for individuals presenting with such psychopathology are extremely variable, borderline- and mood-related psychopathology can intensify or mutually reinforce one another across this developmental period, possibly crossing the threshold to become a “case” of either or both disorders. Regardless of whether an individual crosses such an arbitrary threshold for caseness, a significant proportion of individuals will develop clinically important and persistent functional, vocational, and interpersonal impairment and disability.

There is a clear need for intervention early in the course of these disorders. Balancing the sensitivity and specificity of early-detection programs presents challenges, however, especially given that such symptoms overlap with what might be developmentally normal and that they often resolve without clinical intervention. Effective early intervention has the potential to improve developmental and functional outcomes for those affected, but this provisional benefit must be balanced against the risk of premature diagnosis at an age when diagnostic instability is normative—a situation that can lead to stigma, mistaken diagnostic labels, inappropriate treatment, and other adverse outcomes.

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