Borderline Personality Disorder

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Abstract: This review summarizes the current body of knowledge about borderline personality disorder (BPD). Each section describes the development of BPD’s knowledge base in the 33 years since the diagnosis became part of the American Psychiatric Association’s standard diagnostic classification system. The changes in our understanding are remarkable with respect to virtually every consideration of BPD’s etiology, course, and treatment. This expansion of knowledge has forced the authors to have to choose what we deem is most important, and thereby this review may overlook or underrepresent other significant developments. Nonetheless, we expect readers will share our excitement about what’s been learned. Throughout our review we will highlight very interesting and distinctly different questions that now surround this diagnosis.

Definition

Ontogeny

The first efforts to define the borderline condition were by clinicians who recognized a group of patients within their office practice (1) or inpatient settings (2) that didn’t fit into existing categories but seemed like atypical variants of other disorders. Specifically, like schizophrenia, they had lapses in their reality testing; like depression, they were often desperately unhappy; and like antisocial personality, they seemed impulsive and willfully noncompliant. Three more-or-less concurrent subsequent developments brought wider attention to this condition in the late 1960s. Grinker gave the condition an academic blessing by making the first empirical effort to define the “borderline syndrome” (3). Kety et al. (4), who were conducting a twin study of schizophrenia, needed to include atypical variants to gain statistical power. In the process, they adopted the concept “borderline schizophrenia” and this gave the condition importance as a possible key to the expanded classification of “bordering”—i.e., spectrum—disorders. Finally, Kernberg (5, 6) awoke the psychoanalytic community to the “borderline personality organization” construct. His descriptive effort highlighted the identity problems, primitive defenses, and transient failures of reality testing that still aptly describe these patients. More importantly, he fueled the hope that skilled intensive psychodynamic therapies could affect major changes. Thus descriptive psychiatry, biological psychiatry, and psychotherapeutic hopes dramatically increased attention to these patients.

Borrowing from these contributions and from the diverse literature that had otherwise accumulated about these patients, the senior author proposed a definition (7), developed a reliable structured interview (8, 9), and, using these, conducted a project that established the syndrome’s discriminating features and diagnostic threshold (10). Thus the criteria were developed and formed the basis for the disorder’s entry into the official classification system in 1980 (DSM-III). Those criteria have been shown to have internal coherence with a single latent structure (11–14). With only modest revisions those criteria still endure in DSM-5 (see Table 1).

Controversies

Despite the diagnosis’s relative descriptive stability since 1980, its integrity has been an almost continuous subject of much controversy. It was first...
A pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Frantic efforts to avoid real or imagined abandonment. **Note:** Do not include suicidal or self-mutilating behavior covered in criterion 5.
2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation.
3. Identity disturbance: markedly and persistently unstable self-image or sense of self.
4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating).
5. Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior.
6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).
7. Chronic feelings of emptiness.
8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).
9. Transient, stress-related paranoid ideation or severe dissociative symptoms.

**Note:** Do not include suicidal or self-mutilating behavior covered in criterion 5.

Considered a spectrum variant of schizophrenia (as noted above), then of MDD, then of PTSD, and then bipolar disorder. Of note, a considerable body of research has consistently validated the disorder’s internal coherence, its discriminability from other disorders and—as will be described in this review—its unique course, heritability, pathogenesis, and treatability with diagnosis-specific therapies. Thus, from the point of view of the usual standards, by which validity of a psychiatric disorder is established, it has met the tests. It appears in DSM-5 and as a diagnosis whose usage is now legitimate for adolescents.

The questions that currently surround the borderline diagnosis appear to supersede its established validity. Where it belongs in the classification system is one such issue. Unlike the other personality disorders it has empirically validated treatments, and, in the presence of other major psychiatric disorders its treatment often takes priority. These distinctions set it apart from other personality disorders. Moreover, within the nosological superstructure of internalizing versus externalizing dimensions it appears to occupy some space of its own, whereby it combines characteristics of both (16–18). Moreover, its genetic template overlaps with that for other personality disorders and also with the genetic template for axis I disorders (19, 20). Hence, it is even difficult to see it as a spectrum variant within the major metaschemes of classification.

In an effort to replace all of the existing and clinically traditional categories of personality disorder with a trait dimensional scheme, the ICD-12 will remove BPD—and all other PD categories—from its classification. Within the DSM-5 personality workgroup, there was debate about its redefinition (21). That debate concerned whether BPD should be redefined as a trait disorder representing a variant of normal personality. Trying to blend personality traits with personality psychopathology, Table 2 shows the redefinition that the DSM-5 Task Force proposed. External reviewers, including the APA Board of Trustees, judged it too radical a change, and to be insufficiently tested scientifically and clinically. It will appear in DSM-5’s Section 3 as an alternative definition in need of research.

### Epidemiology

#### Prevalence

In **community samples** borderline personality disorder (BPD) has a median prevalence of 1.7% (0.0%–2.7%) as estimated from all samples reviewed by Torgersen (22) and Trull et al. (23). The high prevalence rate (5.9%) found in the large epidemiologic study (24) was significantly reduced (to 2.7%) when a more conservative analysis of diagnostic data were used (23). Thus, only the latter study was used in prevalence estimation. The somewhat lower prevalence reported by some studies can be attributed to use of self-report assessments (25) or low response rate (26). BPD prevalence is comparable to prevalence of other major psychiatric disorders, such as schizophrenia or bipolar disorder, but lower than major depressive disorder or anxiety disorders (see Table 3). The lifetime prevalence of BPD is between 5.5 to 5.9% (27, 28). This disparity between cross-sectional prevalence and lifetime prevalence is consistent with...
Typical features of borderline personality disorder are instability of self-image, personal goals, interpersonal relationships, and affects, accompanied by impulsivity, risk-taking, and/or hostility. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, as described below, along with specific maladaptive traits in the domain of Negative Affectivity, and also Antagonism and/or Disinhibition.

A. Moderate or greater impairment in personality functioning, manifest by characteristic difficulties in two or more of the following four areas:
1. Identity: Markedly impoverished, poorly developed, or unstable self-image, often associated with excessive self-criticism; chronic feelings of emptiness; dissociative states under stress.
2. Self-direction: Instability in goals, aspirations, value, or career plans.
3. Empathy: Compromised ability to recognize the feelings and needs of others associated with interpersonal hypersensitivity (i.e., prone to feel slighted or insulted); perceptions of others selectively biased toward negative attributes or vulnerabilities.
4. Intimacy: Intense, unstable, and conflicted close relationships, marked by mistrust, neediness, and anxious preoccupation with real or imagined abandonment; close relationships often viewed in extremes of idealization and devaluation and alternating between over-involvement and withdrawal.

B. Four or more of the following seven pathological personality traits, at least one which must be #5 Impulsivity, #6 Risk taking, or #7 Hostility:
1. Emotional lability (an aspect of Negative Affectivity): Unstable emotional experiences and frequent mood changes; emotions that are easily aroused, intense, and/or out of proportion to events and circumstances.
2. Anxiousness (an aspect of Negative Affectivity): Intense feelings of nervousness, tenseness, or panic, often in reaction to interpersonal stresses; worry about the negative effects of past unpleasant experiences and future negative possibilities; feeling fearful, apprehensive, or threatened by uncertainty; fears of failing apart or losing control.
3. Separation insecurity (an aspect of Negative Affectivity): Fears of rejection by – and/or separation from – significant others, associated with fears of excessive dependency and complete loss of autonomy.
4. Depressivity (an aspect of Negative Affectivity): Frequent feelings of being down, miserable, and/or hopeless; difficulty recovering from such moods; pessimism about the future; pervasive shame; feelings of inferior self-worth; thoughts of suicide and suicidal behavior.
5. Impulsivity (as aspect of Disinhibition): Acting on the spur of the moment in response to immediate stimuli; acting on a momentary basis without a plan or consideration of outcomes; difficulty establishing or following plans; a sense of urgency and self-harming behavior under emotional distress.
6. Risk taking (as aspect of Disinhibition): Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard to consequences; lack of concern for one’s limitations and denial of the reality of personal danger.
7. Hostility (as aspect of Disinhibition): Persistent or frequent angry feelings; anger or irritability in response to minor slights and insults.

Within clinical settings BPD is the most common personality disorder at different levels of care. About 6.5%–42.7% (median = 20.3%) (29) of all psychiatric inpatients and about 8%–18% (median = 11.9) (30) of all psychiatric outpatients have BPD. Frequent psychiatric hospitalizations in BPD are associated with suicidality, psychotic symptoms, and anorexia (31) but, surprisingly, not with depression. Similarly, 56% of emergency room patients admitted for suicidal behaviors have BPD (32). The disproportionately high prevalence of BPD found in clinical settings versus general population can be explained by high level of health services use by BPD patients (33, 34). In general medical settings 6% of primary care patients have BPD (35), which at least in part can be attributed to increased prevalence of chronic medical illnesses in BPD patients (36). These elevated figures highlight importance of timely diagnosis and appropriate treatment of BPD patients in all clinical settings.

**SOCIODEMOGRAPHIC FACTORS**

While reports of gender in clinical samples typically reported that BPD is about three times more prevalent in women than in men (37), most epidemiological studies conducted in community samples show similar prevalence rates for both genders (26, 38), although one epidemiologic study confirmed the uneven gender distribution of BPD.

**Table 2. Proposed Redefinition of BPD for DSM-5**

| A. Identity (Markedly impoverished, poorly developed, unstable self-image) |
| B. Self-direction (Instability in goals, aspirations, value, or career plans) |
| C. Empathy (Compromised ability to recognize feelings and needs of others) |
| D. Intimacy (Intense, unstable, conflicted close relationships) |

**Table 3. Prevalence of Some Major Psychiatric Disorders**

<table>
<thead>
<tr>
<th>Psychiatric disorder</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenia</td>
<td>1.1</td>
</tr>
<tr>
<td>Borderline personality disorder</td>
<td>1.7</td>
</tr>
<tr>
<td>Bipolar I disorder</td>
<td>1.3</td>
</tr>
<tr>
<td>Major depressive disorder</td>
<td>6.7</td>
</tr>
<tr>
<td>All personality disorders</td>
<td>9.1</td>
</tr>
<tr>
<td>Anxiety disorders</td>
<td>18.1</td>
</tr>
</tbody>
</table>

Gender Estimates for BPD

1. Sampling sites. BPD men are more likely to be found in substance abuse clinics or correctional settings. Women are more likely to present with co-morbid PTSD or eating disorder (28, 39) and are thus more common in outpatient clinics (37). Epidemiological samples avoid this bias and provide more accurate estimates of gender distribution of BPD.

2. Effects of gender stereotypes on diagnostic practices (reviewed by Skodol & Bender [40]). For example, the same BPD traits (e.g., anger, impulsivity) are more likely to be judged pathological in women than in men (41, 42) and female clinicians are more likely to diagnose BPD in other women than are men (43), probably reflecting women’s heightened discomfort with certain traits (e.g., anger, self-harm).

More research is warranted to clarify these factors.

(23). Gender bias reported in clinical samples seems to be influenced by sampling and diagnostic biases (see Figure 1 Sidebar).

No epidemiologic studies have assessed age of onset of BPD. Extrapolating from onset of self-harm—the most predictive symptom of BPD (44)—the respective age of onset for BPD can be assigned to before age 12 (32.8%), adolescence (13–17 years of age; 30.2%), and early adulthood (18 or older; 37%) (45).

BPD’s prevalence seems to be related to lower education, income, and social class (22). These factors seem to contribute to adversity that may predispose to development of BPD (see subsequent “Biopsychosocial Underpinnings” section) although characteristic BPD symptoms are likely contributors to their poor educational achievement, lower income, and social class as well.

Reports on the marital status of BPD patients in epidemiologic samples indicate they are more likely to be married than Cluster A or C personality disorders patients (22). In clinical samples, probably due to the higher degree of impairment in all personality disorder, BPD patients are similar to other personality disorders in rates of cohabitation or marriage (15.5%) (46); 23% (47) are similar to other personality disorders. However, also consistent with their unstable relationships, BPD patients have higher marital dysfunction (22).

The tendency to be more prevalent in urban areas (i.e., urbanicity) was investigated in two studies, with one reporting somewhat increased prevalence of BPD in urban areas (26) and the other reporting no relationship (24).

**Fortunate and unfortunate situations:** “Fortunate” means high education (above high school), living together with a partner, and living in the outskirts of a city. “Unfortunate” means the opposite. BPD seems to be more prevalent in unfortunate situations (1.2%), than either in the moderately fortunate (0.8%) or fortunate situation (0.4%) (22).

**Culture**

BPD has been generally found in every culture where it’s been studied and negative findings were not reported, including the United States (24), China (48), Japan (49), Brazil (50), Norway (26), and India, Kenya, and Europe (51), although reported prevalence rates varies across different countries (e.g., Japan and China samples reported somewhat lower prevalence rates). Cultural factors seem to effect BPD’s presentation. For example, in Japan—where suicide is permissible, substance use is uncommon, and living with ones’ parents is more acceptable—BPD patients report more suicidal behaviors, less substance use, and more interpersonal instability (52). Studies in larger more representative samples are needed to test this conclusion.

**Natural history**

The course of BPD had been considered very gloomy until research shed light on it. One reason for this was the excessive attention given to the subgroup of BPD patients who were repeat visitors to emergency rooms and inpatient units, the so-called “frequent flyers”. This impression called into question by an original series of research studies that deployed follow-back strategies, i.e. getting in touch with patients who had previously been in treatment at a given institution (53–55). Figure 2 and 3 indicate what was found. Generally, the results showed gradual improvement in psychopathology and a better course than schizophrenia though worse than depression. Because these results were attained from high-income populations, were often from high-prestige institutions using different and sometimes unstandardized assessments and, above all, were retrospective, these results had uncertain credibility.

As noted above, we cannot yet identify a particular age of onset except to note that it has been found in children less than 12 and its incidence appears to grow up to early adulthood. Certainly BPD’s impulsive, aggressive, and self-destructive behaviors emerge during adolescence (56), and remain stable (57).
The methodological limitations of the earlier longitudinal studies were addressed in two prospective studies, the McLean Study for Adult Development (MSAD) and the Collaborative Longitudinal Personality Disorder Study (CLPS). Both were NIMH-funded and were initiated in the 1990s with many results that have been published in recent years. Table 4 summarizes some of their design and methodology characteristics. Of note, while the CLPS stopped after 10 years, the MSAD continues to collect data with the most recent reports having 16 years of follow-up. This review will first discuss the course of BPD psychopathology, then social functioning, and then mediators/moderators.

COURSE OF PSYCHOPATHOLOGY

Figure 4 shows the dramatic, unexpected and prevalent remission of BPD psychopathology as measured by both the decrease in the number of criteria and by an operationalized definition of remission. Of note, the course was similar in both studies despite differences in measures and patient sampling. Thus this overall pattern of change can be considered cross-validated. As noteworthy and unexpected was the finding from both studies that when borderline patients remitted, they were apt to remain so. In the CLPS data only about 12% relapsed (defined as returning to greater than 5 criteria for a year or more). More refined analyses by MSAD indicated, as expected, that a larger portion relapsed when shorter periods of time were used to define either remission or relapse.

Both studies examined the pattern of BPD symptom (criteria) reduction. A report on the 4 year outcome from CLPS suggested what McGlashan (58) labeled a “hybrid model” with some criteria (e.g., self-harm) diminishing more rapidly than others (e.g., affective instability). Similar results were reported from MSAD, where Zanarini (46) adopted the concept of positive (unstable, symptom-like) and negative (stable, trait-like) components. She found that impulsive characteristics and strong unstable relationships resolved earlier than loneliness/emptiness and intolerance of aloneness (59).

COURSE OF SOCIAL FUNCTIONING

A far more discouraging picture of BPD’s natural course emerged from the examination of social
adaptation. The GAF scores of BPD samples remain quite low (about 65) (60), and the rate of those receiving disability support remains quite stable (about 40%) (61). At 10 years follow-up about 30% are married or have stable partnerships and a similar fraction have full-time vocational activities (60). As grim as this seems, the group mean scores for the BPD sample rose significantly on subscales such as self-satisfaction, recreation, and friends. Moreover, these group means hide a considerable amount of variance within the BPD samples and within any particular individual BPD person. Zanarini found that the subsample on disability at 10 years was comprised mainly by a subsample who were not on disability originally (61)! In a longer-term 16-year follow-up, Zanarini has recently reported that 60% of her McLean Hospital-based sample has “recovered,” a term that combines both persisting remission with sustained partnerships (59). This report offers a more hopeful perspective about sustained and substantive changes than have prior reports.

**Mediators and Moderators of Course**

As described elsewhere (see the “Biopsychosocial Underpinnings” subsection), borderline patients have a neurophysiological system that is primed to make them stress responsive. The longitudinal data support this. Their remissions frequently occur when they leave highly stressful situations (62). This explains why they can regain their composure and sociability so quickly when placed in low stress asylum-like situations. Of note, they report having more stressful events, most particularly interpersonal stressors like rejection, that precede and predict self-harm, suicidality, dissociation, and relapses (63–65).

Predictors of outcome were identified as well. Higher degree of BPD psychopathology, low functioning, and history of childhood sexual abuse predicted slower symptomatic recovery in CLPS (66); older age, childhood sexual abuse, family history of substance use, and poor vocational functioning predicted slower symptomatic recovery in MSAD (67). Some of these predictors are probable moderators (e.g., childhood sexual abuse) whereas others are probable mediators (e.g., low functioning) of the course, though more research is needed to examine these hypotheses.

Longitudinal data cannot tell us much about the effects of treatment but they do document the high utilization of health care resources and the gradual reduction of expensive resources like emergency rooms and hospitals (68, 69). They also document the heavy utilization of medications and especially polypharmacy by borderline patients. The more
medications, the worse the course. This probably reflects both overrelaxation on medications despite their modest effects and that a downhill course evokes more prescription of medications.

Examinations of comorbidity are clinically instructive. Of particular importance is that while effects of MDD on BPD course seem to be negligent, BPD has a highly negative effect on the course of MDD (70), a disorder that co-occurs in about 75% of borderline patients. On the other hand, comorbid cluster B personality disorders, notably antisocial and narcissistic types, seems to slow BPD recovery (67). Results regarding effects of comorbid PTSD are inconclusive (66, 71).

**Biopsychosocial Underpinnings**

Our understanding of the etiology of BPD has become increasingly complicated, accommodating the influences of not only biology and environment separately, but also the interaction between the two. Earlier etiological accounts of BPD, influenced by prevailing psychoanalytic theory, emphasized early environment or experiences, such as trauma, in the development of BPD. As research on BPD has specified biological factors associated with phenotypic features of the disorder, modern etiological formulations have focused on constitutional factors, associated to distinct genetic and neurophysiological characteristics. This review will consider the early developmental, genetic, and neurobiological factors implicated in the development of BPD, as well as the incorporation of these underpinnings into clinical formulations that have organized its major treatments. In each area, the recent advancements from research are impressive, yet readers should also note that while the research often differentiates BPD from healthy controls, the more demanding designs that would demonstrate specificity, vis-a-vis BPD’s major border disorders (MDD, bipolar disorder, and PTSD) usually still need to be conducted.

**Early Development**

Early psychoanalytic conceptualizations of BPD identified difficulties with separation from caretakers as a central developmental failure in BPD (72). Both insecure attachment and traumatic separation experiences were theoretically implicated as pathogenic factors contributing to the clinical observation of abandonment fears in BPD (73). These clinical theories prompted empirical investigations that confirmed the association between early caretaking experiences, such as more frequent separations and parental over- or under-involvement, and the diagnosis of BPD (74–76). Subsequent studies specifically demonstrated higher prevalence of sexual abuse in subjects with BPD compared with subjects without BPD, leading to a theory that sexual abuse specifically—and childhood trauma more generally—was a main etiological factor in the development of the disorder and that BPD may be better conceptualized as a chronic or complex form of posttraumatic stress disorder (77, 78). While studies continued to confirm the increased prevalence of childhood trauma among individuals with BPD, they also showed that childhood trauma is neither “necessary or sufficient” for the development of BPD (79), nor accounts for much of the amount of variance in the etiology of the disorder (13, 80, 81).

Prospective longitudinal studies have now clarified that while abuse is significantly associated with personality disorders in general (82) and BPD in particular (83), other environmental and parental factors also contribute significantly to BPD’s development. These include low socioeconomic status, family disruption, life stress, maternal inconsistency in the presence of over-involvement, aversive or hostile parental behaviors, and low parental affection (82–84).

In addition to discrete environmental and parental factors contributing to the etiology of BPD, the nature of transactional process between individuals and caretakers, as codified in attachment styles, have been implicated in the development of BPD. Multiple cross-sectional studies of adult samples with BPD and at least one prospective study have documented the predominance of insecure attachment styles, particularly a combination of preoccupied, disorganized, and fearful styles (83, 85–87). The preoccupied aspects of this combination of styles confers the needy, over-involved, angry, and passive qualities to interactions between individuals and caretakers, whereas the disorganized or fearful aspect captures the contradictory, confused, mistrustful, and disoriented qualities of such interactions. Taken together, these two styles interact to both perpetuate attachment interactions through high levels of involvement as well as potentiate distress and emotional challenge. It is likely that the interaction between these styles heightens vulnerability to adverse caretaking interactions and impairs use of attachment for its regulatory effect.

**Genetics** - In establishing the genetic basis of BPD, a number of family studies of BPD demonstrated a degree of familiality of the disorder (88). They indicate that the mean prevalence of BPD in first-degree relatives is 11.5%, much higher than in the general population (see Epidemiology section). However, these family study findings could not in themselves differentiate the proportion of etiological variance contributed by genetic and
environmental factors (already outlined above). Torgersen and colleagues used twin samples to establish concordance of 35% in monozygotic (MZ) twin pairs and 7% in dizygotic (DZ) pairs, with genetic modeling suggesting additive genetic effects ranging from 0.57 to 0.69 and shared-in-family environmental effects ranging from 0–0.11 (89). Another twin study examined the heritability of component traits of BPD indicated heritability of 45% for affective lability, 49% for cognitive dysregulation, and 48% for insecure attachment (90, 91). Since then one large family and two twin studies assessed the heritability of the component sectors of BPD and used multivariate modeling to determine whether there is a single common latent genetic factor in BPD. They found a range of heritability from 44%–60%, with the remaining variance contributed by individual specific environmental factors (18, 60, 92). This level of genetic influence exceeds the heritability of anxiety disorders and depression, but is less than that of bipolar affective disorder or schizophrenia.

Findings from the limited research on molecular genetics of BPD indicate associations between polymorphisms in genes relevant to the serotonergic and dopaminergic system (see Chanen & Kaess [84] for review). Because polymorphisms in the serotonin transporter gene (5-HTTLPR) have previously established a relationship with affective lability (93), impulsivity (94), and suicide (95) it has been of most interest. However, there remains only limited evidence of the relationship between this polymorphism and BPD, and positive results are inconsistently replicated (84).

Specific predisposing early environmental factors in the etiology of BPD combine with genetic factors to confer varying degrees of vulnerability to the development of BPD. As described above, early childhood adversity in a number of forms, constituting “life stress” in general, contribute to the development of BPD. Recent genetic studies have considered the gene-environment interactions of sensitive genotypes and predisposing environments related to a number of disorders. Functional length polymorphisms of the serotonin transporter gene has been shown to moderate the depressogenic effects of stressful life events (96, 97). In studies of BPD, this short allele polymorphism appears to moderate the effects of stressful life events by decreasing impulsivity (98). Moreover, the genetic influences that contribute to the development of BPD also increase the likelihood of exposure to specific stressful life events (99). In addition, genetic influence effects stress reactivity (100), making some people more likely to develop symptoms following traumatic stress (101). Therefore, while important to distinguish the unique contributions of environmental and genetic forces contributing to the etiology of BPD, it is critical to consider interactional effects between the two.

**Neurobiology**

Neurobiological research has begun to elucidate specific brain and neurohormonal features that underpin the phenomenological characteristics of BPD. Neuroimaging studies of BPD have primarily focused on examining the neural basis of the distinct emotional dysregulation characteristic of the disorder. The early wave of these studies documented hyperreactivity in limbic structures, primarily the amygdala (102, 103), while later studies began to describe distinct dysfunction in prefrontal and frontolimbic activity as the larger mechanism behind emotional dysregulation in BPD (104–106). These data support the notion that emotional intensity and dysregulation in BPD is the manifestation of failures of top-down frontal control processes that should modulate the effects of the bottom-up hyperreactive limbic structures. Ruocco and colleagues meta-analyzed the existing neuroimaging literature, concluding that BPD subjects show: 1) activation of a diffuse network of structures in negative emotion processing extending from the amygdala, anterior cingulate cortex, medial and dorsolateral prefrontal cortex, superior temporal gyrus, posterior cingulated, and cerebellum; 2) decreased activation of regions extending from the amygdala to the anterior cingulate and dorsolateral prefrontal cortex; and 3) increased activation of greater insula and posterior cingulate cortex (107).

The specific finding of reduced activation of the anterior cingulate cortex in negative emotional processing distinguishes BPD from MDD (108), suggesting an important distinction between these disorders. Also, the activity of the insular cortex may underpin a range of symptom sectors in BPD, as this region of the brain has been shown to be involved in the processing of emotional and physical pain, self-awareness, and social cognitive processes involved in empathy and adherence to or violation of social norms. Differences in neural activity in the insular cortex have been demonstrated in the context of difficulties among BPD subjects to sustain and repair cooperation during social exchange (109). Further research is needed to clarify the overlap of dysfunction in the brain areas implicated in negative emotion processing and interpersonal vulnerabilities in BPD, as these two sectors are most defining of the disorder.

Neuropsychological tests that assess actual brain functioning complement the localization results
from imaging studies. They have confirmed dys

function in such areas as prefrontal (primarily) and 
additionally, in temporal and parietal cortex (110, 
111). Interestingly, brain lesions in these areas do 
not produce BPD symptoms (112), suggesting that 
brain abnormalities in BPD patients are more re-

lated to abnormal function of these areas.

While neuroimagining research has focused on 
mechanisms of abnormal emotion processing, re-

search on neuropeptides has shed some light on the 
stress and interpersonal sensitivity of BPD. As dis-

cussed above, individuals with BPD commonly 
experience more early and adult life stress, which 
may both interact with and be potentiated by genetic 

factors, in the context of unstable and insecure 

attachments. As noted earlier (see “Natural History” 
and “Treatment and Outcome” subsections) many 
features of BPD are stress reactive. Biologically, el-
vated cortisol responses to psychosocial stress and 
dysregulated feedback responses in BPD subjects 
suggest overall impaired physiological management 
of stress (113). Neuropeptide systems involving 

opioids and oxytocin also effect stress responses 
show altered functioning in BPD (see Table 5). 
Both opioids and oxytocin also facilitate prosocial 
tendencies. Oxytocin administration is associated 
with enhanced performance on “mind-reading” or 
interpreting mental states from social cues (115) and 
collaboration in social exchange tasks in normal 
subjects (116). Strikingly, the administration of 
oxotcin to subjects with BPD actually increased 
mistrust and decreased cooperation in a social ex-

change paradigm (116). This suggests that the 
BPD’s oxytocin system may produce a paradoxical 
tendency toward mistrust and interpersonal in-

stability in social activity rather than the kind of 
affiliation that normal oxytocin responses cause.

**BIOPSYCHOSOCIAL TREATMENT IMPLICATIONS**

Both environmental and endogenous processes 
contribute to the etiology and clinical presentation 
of BPD. While early childhood adversity, dysfunc-
tional caretaking, and general early life stress may 
function as robust etiological forces in the develop-
mament of BPD, genetics, in addition to disturbed 
regulatory functions of brain and neuropeptide 
systems, may prime individuals to be more prone to 
pathological responses to environmental life stress.

**ASSESSMENT & DIFFERENTIAL DIAGNOSIS**

As noted earlier, the presence of a reliable struc-
tured interview for establishing the BPD diagnosis

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**Table 5. Neuropeptide Modeling of BPD’s Stress and Interpersonal Hypersensitivity**

<table>
<thead>
<tr>
<th>Neuropeptide</th>
<th>Effects on Stress Regulation</th>
<th>Effects of Interpersonal Behaviors</th>
<th>Findings in BPD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Opioids</strong></td>
<td>Regulates responses to stressor which induce physical, emotional, or social pain</td>
<td>Mediates “social affect”</td>
<td>↓ basal opioid levels as explanation for chronic dysphoria and emptiness, as well as difficulty self-soothing and tendency to misuse opiates</td>
</tr>
<tr>
<td></td>
<td>Activates HPA axis</td>
<td>Modulates responses to distress related to separation or rejection and relief related to reunion</td>
<td>↓ basal opioid levels and ↓ pain perception associated with self-injury</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reinforces social interaction</td>
<td>Opioid receptor antagonists may ↓ self-injury</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Involved in self-soothing responses related to separation by dampening fear and stress responses</td>
<td>↓ opioid release in context of sadness in orbitofrontal cortex, caudate, and accumbens; probable relationship to emotional dysregulation in BPD</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Preliminary evidence that polymorphisms of particular opioid receptor genes are related to preoccupation with attachment and identity problems</td>
</tr>
<tr>
<td><strong>Oxytocin</strong></td>
<td>Inhibits HPA axis activity</td>
<td>Central to maternal, pair-bonding, and prosocial tendencies (e.g., licking, grooming, nursing in animals; monogamy, affection, maternal gaze in humans)</td>
<td>↓ urinary and CSF levels in individuals with history of childhood maltreatment and early separations</td>
</tr>
<tr>
<td></td>
<td>Diminishes stress response</td>
<td>Enhances trust and collaboration</td>
<td>While nonborderline subjects demonstrate enhanced collaboration in an economic exchange paradigm upon administration of oxytocin, subjects with BPD, defect more and collaborate less</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Implicated in reading facial expressions/mentalizing</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Involved in encoding of memories of positive social interactions</td>
<td></td>
</tr>
</tbody>
</table>

*a Adapted from Stanley and Siever (114).*
set the stage for it being adopted into DSM-III in 1980. This interview, the Diagnostic Interview for Borderlines (DIB) divides BPD psychopathology into sectors of affect, cognition, behavior, and interpersonal relationships and the diagnosis requires scoring in at least 3 of these 4 sectors (9). In the aftermath of DSM-III a number of reliable structured interviews were developed for assessing the specific criteria for BPD and for all of the other personality disorders (117–120). Structured interviews of the DSM criteria have yielded much of the acquired knowledge about the disorder. Nonetheless, reliance on a polythetic criteria count has been subject to criticism because there are 256 possible combinations by which to attain the BPD diagnosis and hence, there is the danger of great heterogeneity within samples. While this is statistically true, in clinical samples there is much variance in the frequency in which the criteria occur, so that it is rare for a patient to meet criteria for BPD without having both affective and interpersonal criteria—the criteria which represent the core of BPD (121). There are, however, advocates for changing the diagnostic assessment in several ways. One is to use a prototypic description, thereby capturing an overall clinical impression of “borderlineness” (122). The other, endorsed by many clinical and research leaders, is to return to a DIB-like requirement that patients score in multiple sectors rather than by criteria count (21).

Again, as noted earlier, the differential diagnostic issues that surround BPD have undergone changes. The original question as to its being an atypical form of schizophrenia were rather quickly dismissed by virtue of the modest overlap in phenomenology and the failure of BPD to show any familial or genetic connection to schizophrenia. The question of BPD being an atypical form of affective disorder (123, 124) has proven more difficult to dispel. The fact that most (approximately 75%) BPD patients have lifetime MDD and they all present with complaints of severe dysphoria is difficult to ignore. Phenomenologically the borderlines are clearly distinct from those with depression because of their interpersonal and behavioral characteristics. But could these be due to depression? The evidence that the familial relationship is weak (88) and that BPD’s depression doesn’t respond to antidepressants (125, 126); see Treatment & Outcome section below) has sustained the idea of their separateness. Without question, psychiatrists give many BPD patients a MDD diagnosis. There will be fewer questions about reimbursement and they feel better trained to offer medication treatment than psychosocial therapies.

With respect to bipolar disorder, the overlap in phenomenology is more evident; both disorders are emotionally and behaviorally dysregulated. The dramatic expansion in diagnosing bipolar “spectrum” disorders has meant that this has become a common way that borderline patients get mislabeled (127, 128) and for the same unfortunate reasons that the MDD diagnosis is given. Here, however, the co-occurrence of BPD with bipolar disorders is much less (~15%), they appear independent in their course (66), and the familial relationship is very weak (88). The recent evidence that mood stabilizers benefit—albeit modestly and inconsistently—borderline patients (129, 130; see “Treatment and Outcomes” section) will continue to fuel questions about the relationship between these disorders, and sadly, will continue to encourage psychiatrist to rely too much on medication treatments.

A final differential diagnostic question of importance involves PTSD (15). When the high prevalence of trauma in the childhoods of borderline patients (approximately 70%) was reported in the 1980s (see “Biopsychosocial Underpinnings” section), it was suggested that BPD may be an atypical and severe form of PTSD. Against this idea was the fact that a significant number of BPD patients don’t have such trauma, and that borderline patients, unlike PTSD, respond to treatments that focus on feelings, not trauma, and challenge them to take control of their lives. It is now recognized that severe early trauma may have sequelae that include a BPD-like syndrome, called “Complex PTSD,” where trauma is central and requires priority. These patients have trouble with trust and cognitive processing that make BPD treatments for them ineffective. For most BPD patients, as noted earlier, their trauma is superimposed on a genetically determined preexisting sensitivity, and while such children will have psychophysiological difficulties processing trauma and trouble communicating about such adversities, they can as adults benefit from BPD’s therapies.

**TREATMENT AND OUTCOME**

**BPD TREATMENT AND TREATMENT OUTCOME**

Individual psychotherapy has been always seen as the primary treatment for BPD. Initially, dominated by the psychoanalytic paradigm, BPD was conceptualized in terms of specific structural deficits in the personality. This approach prescribed long-term, individual, intensive treatment aimed to restructure personality and thus eliminate BPD symptoms. This fueled therapeutic enthusiasm, which unfortunately was not confirmed by clinical
Improvements were rare exceptions rather than the rule and most such treatments were, in retrospect, doomed to fail due to the toxic effects of the psychoanalytic model, e.g., being unstructured, inactive, and patient-led.

In the early 1990s a second generation of treatments began to emerge that were structured, of 12–18 months duration, and were goal/symptom-oriented. They were described in treatment manuals, often involved a team of therapists, and were tested in randomized control trials (RCTs). Four of these empirically-validated treatments that we believe have the most recognition and usage are featured in Table 6.

Comparison of the outcomes shows that all evidence-based treatments (EBTs) for BPD produce similar improvements in suicidality, deliberate self-harm, depression, and decreased use of emergency rooms, hospitalizations, and medications. Improvement of functioning, especially vocational functioning, and quality of life is modest (132–136). These results mirror the course of BPD reported in longitudinal studies (see “Natural History” section).

All EBTs have a number of salient commonalities (see Table 7) (47, 137–142).

Supportive therapies have often been used as comparison treatments when testing more constrained theory-driven therapies such as DBT, MBT, and TFP. Among these are General Psychiatric Management (143), Structured Clinical Management (144), Supportive Psychoanalytic Psychotherapy (145), and Supportive Group Therapy (136). All of these approaches reduced symptoms through focus on building coping skills, problem-solving, psychoeducation, and validation. These approaches have yielded surprisingly similar outcomes to specialized EBTs (e.g., DBT, TFP, MBT) and yet they were easier to learn and less intensive. Thus, they have emerged as a more practical treatment for clinicians who are not BPD specialists and for clinics where implementation of more specialized therapies is not feasible (146).

Other treatments for BPD. Today there are 13 psychosocial interventions with at least one RCT supporting their effectiveness (for a review see Stoffers et al. [147]). Most lack published treatment manuals, training mechanisms, or much clinical use outside of the research trials. Some of the more recognized and/or potentially useful of these treatments are Schema Focused Psychotherapy

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**Table 6. Evidence-Based Treatments for BPD**

<table>
<thead>
<tr>
<th>Treatment (supportive trials/total trials)</th>
<th>Description</th>
<th>Empirical support</th>
<th>Training mechanisms</th>
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</thead>
<tbody>
<tr>
<td>DBT (13/13)</td>
<td>Behavioral therapy that teaches distress tolerance, emotional regulation, interpersonal effectiveness, and mindfulness; therapist is available to manage crisis; therapy involves 1/week individual and 2/week group therapy; therapists meet 1/week group consultation</td>
<td>Reviewed in Gunderson et al. (146)</td>
<td>Workshop through Behavioral Tech (<a href="http://www.behavioraltech.org">www.behavioraltech.org</a>) and a year-long supervision</td>
</tr>
<tr>
<td>MBT (3/3)</td>
<td>Assuming “not knowing”, curious stance about the patient, therapist teaches the skill of thinking about oneself and others in terms of meaningful intentional states; therapy involves 1/week individual and 2/week group therapy; therapists meet 1/week group consultation</td>
<td>Bateman and Fonagy (144, 164); Jørgensen et al. (136)</td>
<td>Workshop with Dr. Bateman and year-long supervision</td>
</tr>
<tr>
<td>TFP (2/3)</td>
<td>Attachment-based therapy that promotes integrated thinking about self and others through use of interpretation of motives and distortions in perception of self and others, including the therapist; it involves 2/week individual therapy; therapists meet for weekly supervision</td>
<td>Clarkin et al. (145); Doering et al. (133); Giessen-Bloo et al. (149)</td>
<td>Workshop through Personality Disorders Institute (<a href="http://www.borderlinedisorders.com">www.borderlinedisorders.com</a>) and a year-long supervision</td>
</tr>
<tr>
<td>GPM (1/1)</td>
<td>Individual case management mixing dynamic and behavioral models; it focuses on interpersonal and situational stressors; therapy involves 1/week individual therapy with 2nd (group, family, mediation) modality encouraged; therapists meet for weekly supervision</td>
<td>McMain et al. (143)</td>
<td>Workshop with Dr. Gunderson; supervision is desired</td>
</tr>
</tbody>
</table>
MULTIMODEL TREATMENT

There are clinical, conceptual, and empirical reasons to view multimodal, or split-treatments as effective. First, different modalities are likely to complement and augment each other. Second, members of the team are a natural source of mutual support. Third, multimodal treatment provides BPD patients with outlets to express anger and disappointment without leaving treatment (151). Finally, as noted, involvement of the multiple treaters is supported by RCTs for DBT and MBT.

Family Treatment. There are multiple reasons to involve families of BPD patients. First, destructive family dynamics are a major contributor to the discontinuation of treatment by BPD patients (152). Second, families experience significant distress related to the BPD patient’s problems (153). Third, whether or not the families have a role in the BPD patient’s adversity, sometimes family members become entangled in dysfunctional relationships with each other and in such a way impede treatment. Effective family intervention provides psychoeducation regarding BPD, its origin, course, and treatment. It teaches problem solving skills to address difficult dynamics as well as validation and other communication skills to deal with the emotional reactivity of the BPD relative (154).

Group Treatment. Group therapy is an important component of some EBTs (i.e., DBT, MBT). Not only groups offer a valuable opportunity to learn how to share feelings, be empathic, as well as deal with conflict and share attention, but also provide feedback to each other. Group treatment for BPD were supported not only in trials for DBT and MBT (147), but also as a separate treatment modality for BPD (136, 155, 156).

Pharmacotherapy. Medications only have a modest effect on BPD and their role is adjunctive in BPD treatment. It is important to recognize that many symptoms, such as frantic efforts to avoid abandonment, emptiness, identity disturbance, and dissociation do not respond to pharmacotherapy (157). Prescription of medication can help build positive alliance with the BPD patient (151). Selection of medication types is complicated. Although initial reports demonstrated modest effectiveness on depression and impulsivity by selective serotonin reuptake inhibitors (SSRIs) (158), more recent meta-analyses suggests that their overall effects are similar to placebo (126, 157). Atypical neuroleptics showed slightly better effectiveness, particularly in improving mood instability, anxiety, anger, impulsivity, and cognitive symptoms (126, 157). Metabolic side-effects, especially, weight gain and risk of diabetes made them less desirable by patients and decrease compliance. Mood stabilizers have shown some effectiveness in improving mood instability, anger, and impulsivity (157), but such possible side effects as kidney toxicity and sedation (with lithium), cognitive side effects (with topamax) or skin rash (with lamotrigine) reduce compliance. Use of benzodiazepines have not received empirical support and are generally contraindicated for borderline patients because of risks of behavioral disinhibition (159), addiction, and overdose. Pharmacotherapy has a slightly stronger basis for

<table>
<thead>
<tr>
<th>Principle</th>
<th>Description</th>
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<tbody>
<tr>
<td>A primary clinician</td>
<td>Primary clinician assesses progress, monitors safety and oversees communications. Is active and sometimes directive.</td>
</tr>
<tr>
<td>An agreed-upon treatment structure</td>
<td>Goals and roles should be clearly defined, especially personal limits and guidelines to handle crises.</td>
</tr>
<tr>
<td>Connect acts and feelings to events.</td>
<td>Therapist explores problematic behaviors in context of precipitating events, thoughts and feelings.</td>
</tr>
<tr>
<td>Support</td>
<td>Treaters provide validation of the patient’s distress and express hope regarding patient’s ability to change.</td>
</tr>
<tr>
<td>Patient is actively involved</td>
<td>The patients need to know that their progress depends upon their active efforts to take control over their feelings, behaviors, and future. Therapy helps the patient to become more involved.</td>
</tr>
<tr>
<td>Therapist is active</td>
<td>Clinicians are active (interrupt silences, digressions), focus on here-and-now (including parents’ angry or dismissive responses), and help explores thoughts, feelings, and behaviors. Responds to safety issues with concern, but resists cautiously.</td>
</tr>
<tr>
<td>Therapist monitors countertransference, consults with colleagues</td>
<td>Idealization and devaluation are part of relational repertoire of the patient to which the therapist sometimes responds with inclinations to rescue or punish. These reactions (i.e., countertransference), though expectable, can interfere with treatment. Consulting a colleague can help in managing these feelings.</td>
</tr>
</tbody>
</table>
treatment of comorbid disorders such as bipolar disorder or substance dependence (e.g., suboxone for opiate dependence (160) or antabuse for alcohol dependence (161), though concurrent psychotherapy that addresses both BPD and either of these comorbid conditions is critical for therapeutic process (162). Major depressive disorder in BPD only modestly responds to medications (157, 163), and it usually improves only after improvement of BPD (70). These findings underscore the excessive and potentially overuse of this class of medications for borderline patients.

**Important principles** of psychopharmacological case management are 1) Create realistic expectations, namely, medications are not likely to produce curative change and their effects are difficult to assess. 2) Enlist active involvement of the patient in identifying treatment targets, maintaining compliance, ensuring safety, evaluating benefits regarding medications and side-effects, and reading about the prescribed medications. 3) Use independent judgment regarding the effectiveness of medications, since BPD patients are likely to value or devalue medications in deference to their perception of the relationship with the prescriber, e.g., fear of being controlled or of not being cared for, as well as their expectations of being cured (151).

**Levels of Care.** Determination of the appropriate level of care is an issue all psychiatrists need to address (see Table 8). The central principle is to keep treatment at the least restrictive level of care, i.e., provide enough structure to keep the patient safe but enough exposure to problems that they remain engaged in working on treatment targets. Higher levels of care provide safety and containment of crises, although responding to dramatized expressions of distress by unnecessarily placing a BPD patient in a higher level of care is an over-reaction that will reinforce avoidance and encourage the recurrence of crises (151). On the other hand, if the level of care is inappropriately low, the BPD patient will spiral into panic and desperation. Impulsive behaviors will escalate and the treatment will stall. Determination of the optimal level of care is based on clinical experience, rather than an empirically established algorithm, since research in this area is undercut by methodological and ethical limitations (Figure 5 Sidebar).

**Future directions**

At this time the issues that challenge the continued growth of knowledge about borderline personality disorder are very different than those that the diagnosis faced when it entered the DSM-III 33 years ago. At that time the issues were diagnostic validity and treatability. The first of these issues has been convincingly answered by research that has established the syndrome’s latent unifying psychometric coherence and its surprisingly unitary latent genetic structure. Moreover, its treatability has been repeatedly demonstrated by a variety of treatments that have basic and replicable overlapping characteristics. The major problem borderline patients face in the current era is not due to not knowing how to help them, it is the problem of untrained treaters who underdiagnose and overmedicate them.

The BPD diagnosis is now confronted by other and very complicated issues. This review has identified and highlighted some of these. Central to these

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Table 8. Level of Care for BPD Patients

<table>
<thead>
<tr>
<th>Level of Care</th>
<th>Goals</th>
<th>Length</th>
<th>Clinical Tasks</th>
<th>Treatment modalities</th>
<th>Empirical Support (quality)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital 24 h/d 7 d/wk</td>
<td>Making therapy possible</td>
<td>1–2 weeks</td>
<td>Safety/crisis stabilization, assessment, treatment planning</td>
<td>Case management, medications, psychoeducation</td>
<td>No RCTs, only pre-post studies (moderate)</td>
</tr>
<tr>
<td>Residential/partial hospital 10–20 h/wk</td>
<td>Basic socialization</td>
<td>1–6 weeks</td>
<td>Daily living skills, social skills, impulse control, assist with community living, alliance building</td>
<td>Case management, groups (CBT, self-assessment), psychoeducation</td>
<td>One RCT and some pre-post studies (weak)</td>
</tr>
<tr>
<td>Intensive outpatient 4–10 hr/wk</td>
<td>Behavioral change</td>
<td>3–12 months</td>
<td>Further socialization, impulse control, alliance building</td>
<td>Case management, groups (skills, interpersonal), individual psychotherapy</td>
<td>Nine RCTs (strong)</td>
</tr>
<tr>
<td>Outpatient &lt;4 h/wk</td>
<td>Interpersonal growth</td>
<td>As long as needed</td>
<td>Introspection, agency, skill generalization, intrapsychic change, alliance building</td>
<td>Case management, groups (interpersonal, mentalization), individual psychotherapy</td>
<td>20 RCTs (strong)</td>
</tr>
</tbody>
</table>

*a Adapted from Gunderson et al. (146) and Gunderson and Links (151).
The best quality of evidence exists for both outpatient levels of care. Most of the support for the Residential/Partial Program level of care and all support for Hospital level of care come from pre-post design studies (Table 7). Many of these studies were conducted in samples of mixed personality disorders (146), thus limiting the generalizability of the results to BPD patients. A pre-post design does not control for the natural course of the disorder as well as for such bias as “regression to the mean” – the tendency of extreme assessments to return to less extreme levels. Given that higher BPD symptom levels exist at a higher level of care, and that there is a more situational and mercurial nature to these symptoms, studies using a pre-post design are particularly vulnerable to this type of bias. More RCTs are needed to test the effectiveness of higher levels of care. However, conducting such studies is complicated since the random allocation of high risk patients to higher vs. lower level of care brings up ethical dilemmas. Creative solutions are needed.

is the persisting need to determine what is the core of BPD psychopathology. The two candidates are failed emotional regulation and interpersonal hypersensitivity. While both candidates are lending themselves to neurobiological discoveries there remains a conspicuous reluctance by government agencies and by pharmaceutical companies to invest in this research. This in turn is directly related to another persisting and complex issue, i.e., the stigma that continues to surround the diagnosis. Here is a frontier for which education, starting within the mental health professions can bring about change. A third major scientific frontier that becomes evident from this review is the identification of early markers of risk for BPDs development that will allow earlier interventions. Such interventions are probably most likely to be psychosocial in recognition of the pre-borderline child’s excessive sensitivity to stress. Public awareness and research funding are needed.

Other frontiers identified in this review involve treatment of BPD. There exists a major need for the next generation of treatments to become more usable by nonexperts and part of the basic training given by all mental health professionals. Another implication is that the next generation of treatments needs to affect changes in vocational and more generally in the social functioning of BPD patients. Social rehabilitation processes should be major components of treatment. A related implication is that future measures of outcome should assess BPD’s neurobiological and psychological handicaps. Treatments may be best organized around helping individuals manage stress through case management interventions as well as psychotherapeutic interventions designed to enhance prefrontal activity in processing emotions as well as those designed to enhance attachment by interrupting dysfunctional social tendencies which may be in part driven by altered neurobiological responses.

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