

## **Should Borderline Personality Disorder be added to the MA Parity list of “biologically-based” disorders?**

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## Preface

Mental health disorders recognized by the Massachusetts Mental Health Parity Law “are biologically-based as described in the most recent edition of the Diagnostic and Statistical Manual of Mental Disorders published by the American Psychiatric Association.” The Acts of 2008 added eating disorders, post traumatic stress disorder, substance abuse disorders, and autism to the previously identified disorders in the Acts of 2004 (schizophrenia, schizoaffective disorder, major depressive disorder, bipolar disorder, paranoia and other psychotic disorders, obsessive-compulsive disorder, panic disorder, delirium and dementia, and affective disorders).

The Parity Law (2000 and 2008)\* authorizes the DMH Commissioner to approve for the diagnosis and medically necessary and active treatment *any mental disorder*, as described in the most recent edition of the DSM.

In December, 2004 the MA Department of Mental Health convened a group of psychiatric experts to construct a set of clinical criteria that could be utilized in the event that the DMH Commissioner was asked, or wished, to consider the addition of a specific mental disorder to the group already included in the law.

The criteria are: *A mental illness is biologically based if it is associated with an underlying **abnormality of brain** structure or function that results in **significant disability** due to disturbance in mood, thought cognition or behavior.*

Evidence of underlying **abnormalities in brain** structure or function may include, but is not limited to, evidence that the disorder demonstrates:

- a. genetic transmission;*
- b. association with abnormal neurophysiological function(s);*
- c. association with neuroendocrine or other physiologic disturbances;*
- d. responsiveness to biologically based treatment(s).*

**Significant disability** means clinically significant distress or impairment in social, occupational or other important areas of functioning.

\* Chapter 80 of the Acts of 2000 and Chapter 256 of the Acts of 2008

## **Executive Summary**

Borderline Personality Disorder (BPD) is a severe mental illness seen in approximately 20% of inpatient and outpatient clinical samples and between 1.2% and 5.9% of the general population. It can co-occur with other disorders, but studies of its heritability, diagnostic validity/reliability, and of specific treatments indicate that it is best considered an independent disorder that negatively affects the patient's treatment response to comorbid disorders, particularly mood disorders.

Borderline Personality Disorder is severe and can be lethal, with an estimated 65-70% of individuals making at least one suicide attempt and 10% dying by suicide. Persons with BPD are high utilizers of treatment, especially emergency departments and inpatient hospitalizations – the most expensive forms of psychiatric treatment. While some patients remain chronically symptomatic, the majority improves. Within six years of follow-up; 74% no longer meet the diagnostic criteria.

BPD is believed to emerge from an interaction between genes and environment. The major twin study showed that genes accounted for 69% of the variance in diagnostic concordance. This concordance rate is similar to that found in bipolar disorder and stronger than rates for depression or anxiety. Functional MRI studies of BPD patients show abnormalities in the amygdala (an almond-sized and shaped brain structure linked with a person's mental and emotional state) and the prefrontal cortex (a part of the brain associated with planning, reasoning, solving problems and regulating thoughts, feelings and behaviors).

A number of treatments for BPD have been empirically validated, and those whose cost effectiveness has been studied (Dialectical Behavioral Therapy and Metallization-Based Therapy) showed lower costs of treatment compared with treatment as usual. Medications, including antidepressants, mood stabilizers, and antipsychotics help treat emotional dysregulation, impulsivity, and cognitive symptoms. When BPD is not properly diagnosed and patients do not receive treatments known to help them, their condition is likely to worsen.

The findings from psychopharmacologic and other biologic treatment data, coupled with associated brain functioning findings, indicate that BPD is a biologically based disorder. Treatment data indicate that accurately diagnosing and treating BPD conserves resources and improves outcomes. Based on this analysis, including BPD in the Massachusetts Parity Law as a "biologically-based disorder" is well founded.

## Case Study

"Susan," a 23-year-old woman, was brought by ambulance to a Boston emergency room after cutting her wrist at home following an argument with her boyfriend.

*Scenario 1:* The evaluator asked the patient, "Do you think you're depressed?" "Yes, of course!" she screamed. Susan: "I had to convince the doctor in the ER to admit me. There I was... scared, covered in blood, and alone. The doctor told me that it didn't seem so serious, that with a few stitches I could go home and calm down. I tried to tell him that for me there was no such option. If I went home then, I would never leave home again alive. I explained that I could not sleep, had no interest in doing anything, couldn't concentrate, and was bingeing and purging. My boyfriend had dumped me after an out-of-control sex and drinking spree and I felt like a nothing. I had a ton of diagnoses: schizo, depression, anxiety, bipolar, split personality, you name it. No one ever really seemed interested in talking to me. I hated taking the drugs because of the side effects; losing my hair and gaining 44 pounds. He did hospitalize me, and off I went with another diagnosis (type 2?) and they gave me a whole new set of meds. Antidepressants, antipsychotics, stuff to sleep, prn's. After 2 weeks I went home, but nothing had really changed. Two weeks later I cut myself again, this time more seriously, and I have been back five times since then". The next time Susan had to go to the ER it was different.

*Scenario 2:* After understanding how quickly Susan's urge to cut had developed, the ER evaluator observed, "it's like sometimes your emotions get so intense you urgently need to do something to relieve them." "Yes, that's exactly how it feels," replied Susan. "This evaluator seemed calm and not in a rush the way they usually are. I told her about the argument that I had with my mother about her new relationship, and how I felt like she was leaving me behind. My sex and drug sprees were at a feverish pitch, because my boyfriend acted like he didn't know me and the pain was terrible. Somehow this evaluator got me talking about the hole in my chest and the lost and scared feelings I had all the time. She gave me a new diagnosis-"borderline personality disorder"-and instead of going inpatient I was sent to a day program. The staff there understood my pain and taught me techniques (like mindfulness) that I had never even heard of over the eight years I was admitted to psychiatric hospitals. They insisted that my mother attend a session with me every day, and the other patients there had problems like mine-so I didn't feel like such a weirdo. In six weeks, I learned new skills to help me through intense feelings. I started to feel more in control and better about myself. The medications were helpful, too, and there are only two of them.

## **I BPD - Diagnostic and Symptom Overview**

Borderline Personality Disorder (BPD) is the most commonly diagnosed personality disorder in clinical settings. BPD is associated with marked distress and impairment in social, occupational, and role functioning. As defined by the current Diagnostic and Statistical Manual of the American Psychiatric Association, it is a diagnosis given to individuals who meet 5 of the 9 criteria listed in Table 1 (see below).

Different approaches to conceptualizing BPD have been advanced <sup>[1, 2]</sup> based on the clustering of symptoms. Whether the existing DSM criteria consistently cluster into a single syndrome or should be thought of as independent dimensions has been debated. Factor analyses <sup>[3, 4]</sup> from research studies suggest the presence of three primary factors: a) disturbed relatedness (unstable relationships, identity disturbance, and chronic emptiness), b) behavioral dysregulation (impulsivity and suicidality/self-mutilatory behavior), and c) affective dysregulation (affective instability, inappropriate anger, and efforts to avoid abandonment). A thorough review of the literature supports high correlations between the primary factors and strongly argues against viewing them separately. Moreover, results of other structural analyses suggest BPD to be a unitary diagnostic construct. <sup>[5, 6]</sup>

Table 1. Borderline Personality Disorder Criteria <sup>[7]</sup>

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). Note: Do not include suicidal or self-mutilating behavior covered in Criterion 5.
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

## **II Epidemiology**

In clinical populations, BPD is found in roughly 20% of inpatients and outpatient settings. <sup>[8]</sup> Remarkably, it is often not diagnosed. For example, in Zimmerman's study, only two of 61 BPD patients were actually given the diagnosis, and patients with BPD received nearly twice as many diagnoses as those without BPD. <sup>[9, 10]</sup> Because BPD patients present with mood instability they are often given a diagnosis of bipolar disorder – a diagnosis that is greatly overutilized. <sup>[11]</sup>

In nonclinical populations, 1 in every 10 people meets diagnostic criteria for a personality disorder. As personality disorders are associated with high levels of behavioral health service use, they represent a major public health concern. <sup>[12]</sup> Aggregating eight epidemiological studies, the prevalence of BPD ranges from 1.2 to 5.9 percent of the general population <sup>[13]</sup>. In clinical service provision systems, BPD is more prevalent among women (about 75%) but community samples show that it is equally prevalent across genders <sup>[14,15]</sup> and that males with BPD are more frequently found in forensic and substance abuse programs.

## **III Specificity and Comorbidity**

Findings from a prospective study of 668 patients (Collaborative Longitudinal Personality Disorders Study) indicated that posttraumatic stress disorder, substance abuse, and major depressive disorder were frequently associated disorders in persons with BPD. Data from this study show that treating BPD improves the course of eating disorders, substance use disorders, major depressive disorder, and bipolar disorder, but the inverse is not true. Various research studies report that BPD should not be conceptualized as a variant or extension of any disorder (ex. depression, bipolar). <sup>[16-20]</sup> The disorder should stand alone, and with specialized treatment, other comorbid disorders typically improve.

In terms of the co-occurrence between BPD and other personality disorders, a wide range in rates and patterns of comorbidity across samples has been reported. For example, in a sample of inpatients with BPD, comorbid diagnoses from other personality disorders were observed. <sup>[21]</sup> In samples of outpatients, BPD was associated most strongly with avoidant, paranoid and dependent personality disorders. <sup>[22]</sup> The multiplicity of additional personality diagnoses that people with BPD are given does not detract from the fact that once specific treatment(s) for BPD are initiated, other comorbid conditions improve.

#### **IV Course, Prognosis, Phenomenology, and Treatment**

Though personality disorders are not officially diagnosed until adulthood, BPD symptoms can often be observed during adolescence. Beginning with impaired psychosocial functioning, the disorder over time dramatically impairs the individual's ability to achieve normal adult developmental milestones such as academic/occupational success and successful partnering.<sup>[23]</sup> Additionally, severe symptoms in adolescence are associated with added risk for BPD as well as other forms of severe impairments in adulthood.<sup>[23]</sup> Among adults, severe baseline psychopathology, poor vocational functioning, a history of childhood trauma, and poor quality of relationships predict a negative prognosis.<sup>[24, 25]</sup>

BPD is severe and can be lethal. Approximately 65-70% of individuals with BPD make at least one suicide attempt, and 10% die from suicide – a rate 50 times higher than that observed in the general population.<sup>[26-29]</sup> A core feature of BPD is self-destructive behavior, including bingeing and purging, substance abuse, risky sexual behavior, reckless driving and spending, and self-injury.<sup>[30]</sup> In the short term, these behaviors attempt to regulate out-of-control emotions, but the interpersonal consequences further impair troubled relationships.<sup>[31]</sup>

Longitudinal research shows BPD increases behavioral health service use. Among a sample of 633 patients, those diagnosed with BPD were significantly more likely than patients with major depressive disorder to use most types of treatment.<sup>[32]</sup> Similarly the McLean Study of Adult Development showed that borderline patients were far heavier utilizers of treatment than patients with other personality disorders.<sup>[33]</sup> In both studies, those with BPD had higher use of emergency department and psychiatric inpatient care. They spend more days in the hospital or in partial hospitals -- the most expensive forms of psychiatric treatments.

Patients with BPD often improve, though a subset remains chronically ill.<sup>[33]</sup> In the NIMH Collaborative Longitudinal Personality Studies Study, 44% of patients diagnosed with BPD continued to meet diagnostic criteria two years later.<sup>[34]</sup> In a prospective 6-year follow-up of patients with BPD, 74% continued to meet the criteria during the follow-up period.<sup>[35]</sup> Among a sample of Canadian patients with BPD, after 27 years, only 8% continued to meet the diagnostic criteria.<sup>[36]</sup> Remission is multiply determined. Important contributing factors include: effective interventions; the changing severity of other psychiatric illnesses; diminished impulsivity across the lifespan; social learning; and avoidance of conflictual relationships.<sup>[37-39]</sup> Despite diagnostic remission, a significant degree of functional impairment in relationships often remains.<sup>[40]</sup>



## V Etiology

BPD emerges from interactions between genetics and the environment.

### Heritability and Familiarity

The major BPD twin study showed that genes accounted for 69% of the variance in BPD in a sample of 92 identical and 129 fraternal twins.<sup>[41]</sup> This is greater than the heritability of major depressive disorder or anxiety disorders and is similar to that for bipolar disorders.<sup>[42]</sup> Genetic risk factors for BPD are identified but await replication.<sup>[43, 44]</sup> In 9 family history studies, 12.6% of first-degree relatives of BPD probands had the disorder, a percentage four times higher than probands with other psychiatric conditions.<sup>[45]</sup> Affective instability, impulsivity, and disturbed interpersonal relationships are more common in first-degree relatives of individuals with BPD compared to individuals with schizophrenia or other personality disorders.<sup>[46, 47]</sup>

### Environmental Factors

Childhood abuse<sup>[48]</sup>, especially severe<sup>[49]</sup> or prolonged<sup>[50]</sup>, is associated with BPD. In the Collaborative Longitudinal Personality Disorders Study, the association between BPD and childhood abuse and neglect was the most robust.<sup>[51]</sup> At the same time, childhood abuse is only one of several early independent predictors of BPD. Others include attachment-related factors such as parental separation or unfavorable parental rearing styles.<sup>[52]</sup>

Meta-analytic findings support sexual abuse as a contributing factor in BPD. Data aggregated across approximately 2,000 subjects reported in 21 studies yielded an overall correlation of .28<sup>[53]</sup>, which is moderate in magnitude. Nonetheless, a sizeable minority (20-45%) of individuals diagnosed with BPD report no history of sexual abuse<sup>[54]</sup>, and most people (roughly 80%) with sexual abuse histories do not demonstrate personality disorders<sup>[55]</sup>. In a longitudinal study that followed children who experienced serious abuse, a substantial proportion showed little impairment in social, occupational, and interpersonal functioning.<sup>[56]</sup>

### Biological Factors and Pathophysiology

Recent data link BPD to both structural and physiological brain abnormalities. Volumetric studies using MRI consistently show decreased volumes in the hippocampus and amygdala of persons with BPD.<sup>[57]</sup> Functional MRI studies using standardized tests have demonstrated differences in brain areas and functioning between people with BPD and controls.<sup>[58]</sup> Using evoked emotional response, fMRI differentiated BPD from controls with differences appearing in the amygdala, anterior cingulate and ventromedial prefrontal cortex.<sup>[57]</sup> Finally, compared to controls, individuals with BPD have been reported to exhibit greater left amygdala activation in response to facial stimuli that involved expressions of emotion.<sup>[59]</sup>

This research suggests that both the affective instability and the interpersonal hypersensitivity seen in BPD have their roots in the sensitivity of the brain's amygdala to negative emotions. In the face of this increased amygdalar activation, persons with BPD demonstrate impaired self-regulatory function in the prefrontal cortex. Clinically, this corresponds to the subjective and observable dysregulation of emotions and behaviors in the setting of the borderline patient's recurrent states of affective arousal.

Consistent with these conclusions, brain metabolism studies using positive emission tomography (PET) show significant differences between BPD and controls in glucose metabolism.<sup>[57]</sup> Additional findings from PET research suggest that a) individuals with comorbid BPD and major depressive disorder demonstrate altered activity in the parietotemporal and anterior cingulate cortical areas of the brain<sup>[60]</sup>; b) frontal and prefrontal hypermetabolism and hippocampus and cuneus hypometabolism have been observed in individuals with BPD compared to controls<sup>[61]</sup>; and c) lower 5-HT synthesis capacity in corticostriatal neuro-pathways has been observed in individuals with BPD relative to healthy controls.<sup>[62]</sup>

Basic cognitive testing such as visual-perceptual speed and working memory also have been found to differentiate women with BPD from age- and education-matched controls.<sup>[63]</sup>

Differences in neurotransmitter systems also seem to distinguish individuals with BPD from controls. Work in this area has focused on the serotonergic<sup>[64]</sup> and dopaminergic systems.<sup>[65]</sup> Significant findings have been published regarding both neurotransmitters. For example, research on the dopaminergic system has uncovered specific gene variants in people with BPD.<sup>[65]</sup>

Most recently, transcranial magnetic stimulation (TMS) has been used as a tool to study the pathophysiological underpinnings of BPD. In addition to the parasympathetic nervous system differences (discussed below; see<sup>[66]</sup>), TMS research suggests that BPD is associated with cortical inhibition deficits.<sup>[67]</sup>

Taken all together, a large emergent body of research-based evidence in multiple modalities strongly suggests that a biological basis underlies BPD. This assertion is underscored by consistent findings that there are similar differences among individuals with BPD versus controls with respect to neuroanatomy, functioning and pathology.

## **VI Treatment**

In October 2001, The American Psychiatric Association (APA) published practice guidelines<sup>[30]</sup> for the treatment of patients with BPD. In 2005, an update was published.<sup>[68]</sup> Both documents advance the position that psychotherapy represents the core treatment for BPD, and that adjunctive, symptom-targeted pharmacotherapy can be helpful.

### Psychotherapy

BPD is treatable with several types of therapy. The first therapy with demonstrated effectiveness for treating is Dialectical Behavioral Therapy (DBT).<sup>[69]</sup> DBT combines techniques from Cognitive Behavioral Therapy (CBT) and meditative practices. The intervention comprises four core modules covered in group and individual sessions: mindfulness, interpersonal effectiveness, emotion regulation, and distress tolerance. In a randomized, controlled trial (RCT), DBT was shown to be effective for symptoms of BPD among patients with co-morbid substance abuse.<sup>[70]</sup> In another RCT in which DBT was compared to Comprehensive Validation Therapy (CVT) in a sample of patients with both BPD and opiate abuse, both treatments were shown to be effective, but greater maintenance gains were realized among patients treated with DBT.<sup>[71]</sup> In a recent study<sup>[72]</sup>, researchers found that for substance dependence disorders, patients who received DBT were more likely to achieve full remission, spent more time in partial remission, and reported more drug- and alcohol-abstinent days than did patients who received community treatment by experts. Findings from additional outcome studies also provide evidence that DBT affects symptom reduction, including decreases in rates of self-harming behaviors.<sup>[73-75]</sup>

In addition to DBT, 5 other treatments have been empirically validated: Cognitive Behavioral Therapy (CBT), Systems Training for Emotional Predictability and Problem Solving group therapy (STEPPS), Mentalization Based Therapy (MBT), Transference Focused Psychotherapy (TFP), and Schema Focused Therapy (SFT). After one year of receiving weekly CBT sessions and at 18-month reassessments, patients with BPD who reported having thoughts about suicide or who engaged in self-injurious behavior showed significant decreases suicide ideation, hopelessness, depression, number of borderline symptoms, and dysfunctional beliefs at termination.<sup>[76]</sup> STEPPS is a cognitive-behavioral systems-based intervention that appears to improve symptoms, global functioning, and diminish depression in a cost-effective way.<sup>[77]</sup> MBT<sup>[78]</sup> is aimed at helping patients increase their capacity to understand the emotional states of themselves and others even at times of increasing distress. It has shown impressive effects at reducing suicidality in randomized, controlled trials of an inner city sample of patients with BPD – effects that were sustained over 1.5 years<sup>[79]</sup> and 6.5 years.<sup>[80]</sup> TFP<sup>[81, 82]</sup> has demonstrated efficacy on par to DBT in one study.<sup>[81]</sup>

## Psychopharmacology

There are many challenges involved in developing an evidence-based psychopharmacologic regimen to treat BPD. Comorbid disorders relate to the affective dysregulation (including mood and anxiety disorders), impulsivity (including substance use and eating disorders), intolerance of aloneness (including somatoform disorders and medical illnesses) and cognitive disturbances in BPD (including PTSD and psychotic disorders).<sup>[83]</sup> Untreated BPD appears to significantly worsen the course and prognosis of the Axis I disorders, such as major depressive disorder<sup>[84]</sup> and bipolar disorder.<sup>[85]</sup> It has been conceptualized as an engine that can fuel both psychiatric and medical comorbidities, such as chronic fatigue syndrome, fibromyalgia, temporomandibular joint syndrome, back pain, hypertension and urinary incontinence.<sup>[86]</sup> When clinicians lack a clear understanding of the relationship among these factors, this can lead them to bypass practice guidelines<sup>[87, 88]</sup> and treat persons with BPD with psychopharmacologic abandon, chasing various symptoms without a clear formulation of the illness complex. This process increases prescribing<sup>[89]</sup>, worsens care, and increases costs – a process harmful to both patients and taxing to the finite resources in the healthcare financing system.<sup>[90]</sup>

While complex, data indicate that a rational psychopharmacologic approach to BPD can improve outcomes and that patients can respond to medications that target key symptoms of BPD; namely, affective dysregulation, impulsive behaviors, and cognitive/perceptual disturbances.<sup>[91]</sup> The relevant literature is organized by considering four classes of medications:

1. Antidepressants including tricyclics, MAO inhibitors and SSRIs have been researched in the psychopharmacologic treatment of BPD.<sup>[92-95]</sup> Findings from these studies indicate that the evidence to support the effectiveness of tricyclics is poor, and while there is reasonable evidence to support the efficacy of MAO inhibitors, their high lethality in overdose argues against them as a first line treatment. SSRIs have been recommended as the treatment of choice for both affective dysregulation and impulsivity<sup>[91]</sup>, though as Abraham and Calabrese<sup>[92]</sup> recently pointed out, the role of the SSRI's as first-line psychopharmacologic treatment for BPD may warrant re-evaluation in favor of antipsychotics and mood stabilizer medications.
2. First generation antipsychotics have been studied in BPD -- including haloperidol, thiothixene, trifluoperazine, loxapine, and chlorpromazine; all have some evidence for symptom reduction in terms of affective, behavioral, and cognitive symptoms.<sup>[92]</sup> Second generation antipsychotics have been studied more recently, and olanzapine has been investigated most extensively.<sup>[92]</sup> One study demonstrated that olanzapine was superior to fluoxetine as a monotherapeutic intervention<sup>[96]</sup>, and another study demonstrated the superiority of DBT with olanzapine as compared to DBT with placebo.<sup>[97]</sup> Open-label studies of quetiapine<sup>[98]</sup>, aripiprazole<sup>[99]</sup> and IM risperidone<sup>[100]</sup> have

shown clinical and functional improvement sufficient to warrant further study. Soloff<sup>[95]</sup> cited a case study of clozapine in which dramatic improvement occurred in a patient with BPD and severe chronic self mutilation who had failed all other treatments. Despite some evidence of effectiveness with atypicals, their metabolic effects raise concern for their use in obese patients who meet criteria for BPD.<sup>[101-104]</sup>

3. With the exception of lithium carbonate, which seems *not* to be efficacious in the treatment of BPD<sup>[92, 94,105]</sup>, conclusions about mood stabilizers remain uncertain. Based on the studies to date, Abraham and Calabrese<sup>[92]</sup> supported their use, Paris<sup>[94]</sup> failed to support their use, and Soloff<sup>[95]</sup> opined that positive outcomes may be more attributable to the mood stabilizers' effects on impulsivity than to changes in mood. In double-blind studies with random assignment, divalproex<sup>[106]</sup> was shown to reduce impulsive aggression. Findings from a retrospective chart review suggested that lamotrigine<sup>[107]</sup> was a "safe and effective" option for addressing affective instability in BPD.

4. Although benzodiazepines are widespread in the treatment of BPD<sup>[90]</sup>, there is no evidence to support their use<sup>[95]</sup>. One of the few placebo-controlled studies of a benzodiazepine, in this case, prazolam, actually showed symptomatic worsening with increased frequency of severe behavioral dyscontrol.<sup>[92, 95]</sup> This is an example where accurate diagnosis of BPD can help. If a patient is not diagnosed as having BPD, benzodiazepines (which can worsen affect regulation, increase behavioral disinhibition, and impair cognition) would be expected to worsen the condition in many, if not most cases. An important gap in clinical services is the failure to make this diagnosis with a result of polypharmacy in persons with BPD.<sup>[94, 108]</sup>

## **VII Is BPD a "Biologically Based" Disorder**

The MA Department of Mental Health utilizes the following expert-consensus definition of "biologically-based disorder":

"A mental disorder is biologically based if it is associated with an underlying abnormality of the brain structure or function that results in significant disability due to disturbance in mood, thought, cognition, or behavior. Evidence of underlying abnormalities in brain structure or function include, but are not limited to, evidence that the disorder demonstrates:

1. genetic transmission;
2. association with abnormal neurophysiological function(s);
3. association with neuroendocrine or other physiologic disturbances;
4. responsiveness to biologically based treatments."

As previously described, BPD produces significant functional disability such that even when symptoms “remit” the functional impairment can persist.<sup>[40]</sup> The disorder demonstrates heritability on par with bipolar disorder and higher than major depression. Functional MRI studies have confirmed neurophysiological brain dysfunctions and MRI studies have identified structural brain abnormalities. The neuroendocrine correlates (including significant alterations in the hypothalamic-pituitary axis<sup>[109]</sup>) are established. When BPD is treated with an appropriate and well-informed combination of psychotherapy and medication, symptoms can be effectively diminished, but when the diagnosis is overlooked or when BPD patients are not given appropriate treatment, they usually get worse.

Taken together, the findings from psychopharmacologic and other biologic treatment data, coupled with associated brain functioning findings, support the position that BPD is more similar to psychiatric disorders considered to be biologically-based than not. In fact, the existing evidence that supports BPD as a distinct biologically-based disorder is far stronger than was such evidence for schizophrenia or bipolar disorder when included in the original MA parity law. It would be appropriate to approach BPD for characterization and classification purposes in a manner similar to that in effect currently for illnesses such as schizophrenia or bipolar disorder. Several research groups are now arguing to reclassify BPD onto Axis I in DSM V.<sup>[110, 111]</sup> Adopting such a perspective would be expected to impact determinations for reimbursement of ongoing services in several ways, which itself presents certain issues for consideration. Some of these implications will be explored in the next section of this report.

## **VIII Implications of Including BPD as a “biologically-based disorder”**

### **Fiscal Considerations**

Adding BPD to the list of “biologically-based disorders” would increase its insurability as a stand-alone diagnosis. Theoretically, the population of eligible individuals would comprise 1-6% of the general population, thus significantly increasing expenditures if such a change attracted persons to services who are currently receiving none. Given the high prevalence and the severity of the illness, there is good reason to believe that most persons with BPD wishing to receive care are already in the system of care. The problem is that they are frequently not diagnosed as such and are ineffectively (and expensively) treated.

Although the pool of insured service recipients could enlarge, individuals with BPD currently use a highly disproportionate amount of services. If insured under parity, the frequency that services would be offered in less appropriate (and potentially more costly) settings, such as emergency departments, inpatient facilities, police stations, and courtrooms could decrease; as specialized appropriate services would ideally be offered

in more clinically appropriate behavioral health settings. Most importantly, the “cap” for outpatient services would be replaced by “medical necessity” allowing people with BPD to get the care they need to improve over time.

In addition to the clinical rationale that treatment for BPD as the primary diagnosis (rather than one of the myriad comorbidities) would improve care, it can be expected that when patients can be engaged in evidence-based treatment, this would be much more cost-effective. For example, regarding DBT specifically, much is known.

Estimates are that DBT costs approximately 50% as much as treatment as usual (TAU).  
 [112]

<b>One Year Health Care Costs Per Patient</b>		
	<b>DBT</b>	<b>TAU treatment as usual</b>
Individual Psychotherapy	\$3,885	\$2,915
Group Psychotherapy	\$1,514	\$147
Day Treatment	\$10	\$876
Emergency Room Visits	\$226	\$569
Psychiatric Inpatient Days	\$2,612	\$12,079
Medical Inpatient Days	\$360	\$1,096
<b>Total</b>	<b>\$8,607</b>	<b>\$17,682</b>

Regarding MBT, data are clear that the treatment reduces hospitalization, self-injury, and leads to long-term patient stabilization. In a cost-comparison with treatment as usual, during the course of treatment MBT showed equal treatment costs, as the more expensive MBT costs were offset by savings in hospitalizations and emergency department visits. Importantly, 18 months after the completion of treatment, costs for continued care in the MBT group were one fifth of that for those given treatment as usual.  
 [113]

## Diagnostic Drift and Net Widening

Should BPD be eligible for parity, but other personality disorders remain ineligible, it is possible that “diagnostic drift” could occur. In other words, individuals who would be diagnosed with any other personality disorder, and more specifically with cluster B personality disorders, using clinical criteria alone, could receive a BPD diagnosis with greater frequency than occurs currently (thereby assigning a patient a diagnosis eligible for reimbursement rather than in ineligible one). Such a phenomenon has occurred throughout the last 50 years, with “drift” occurring in both directions (from more severe to less severe diagnoses and vice versa) as a function of sociocultural and fiscal variables.

Diagnostic drift can be managed through quality management practices (internal to an agency) and quality oversight (external to an agency). There are methods to set up cost disincentives to intentional misdiagnosis, such as imposition of fines for the practice and loss of CMS certification.

A related concern is that were BPD to be added to the MA parity list of “biologically based disorders”, there would be a movement to classify other personality disorders in the same manner. However, at this time there is insufficient data to support such a move and further evaluation would be warranted.

## Symptom Fluctuation

The McLean Study of Adult Development and the Collaborative Longitudinal Personality Disorders Study offers strong evidence that although the personality traits and social maladjustment associated with BPD are stable across time, the disorder is characterized by periods during which diagnostic criteria may not be met.<sup>[114]</sup> One could argue that because of observed periods of sustained remission, BPD does not warrant specific identification in the MA parity Law. Clearly, however, this line of argument should be dismissed given the same course of symptom fluctuation apparent in individuals who are compliant with treatment for, for example, bipolar disorder. Moreover, the potential for treatments designed specifically for BPD to greatly diminish health care resource utilization and to create enduring remissions offer strong reasons to upgrade the use of the diagnosis and the quality of treatment they are given.



### Pressures on Intervention Practices

Following the availability of pharmacological interventions for schizophrenia, schizoaffective disorder, bipolar disorder and major depressive disorder, there was a decrease in the use of non-medication treatments for these conditions. It is possible that a similar trend may occur in which pressures to use medication as the first-line (or only) psychiatric treatment could eclipse more cost-effective and clinically appropriate psychosocial care – thereby increasing costs and decreasing quality. That said, it appears more likely that a move to treat BPD as a primary diagnosis would increase, not decrease, effective psychosocial treatments.

### Labeling and Stigmatization

Persons with BPD could be expected to be mixed in their reaction to specific identification of the diagnosis in the Parity Law. Those more vociferous in the patients' rights movement might claim that BPD, which already is a stigmatizing diagnosis, would be stigmatized further by this change.

On the other hand, given the stressors and burdens with which many people with BPD live, they may experience a sense of "justification" that the disorder and its consequences are sufficiently serious to warrant its inclusion.

### Conclusion

Taken together, findings from the data reviewed herein, coupled with theoretical discussions of these data, support the position that BPD is more similar to psychiatric disorders considered to be biologically-based than not.

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