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ORIGINAL PAPER

Borderline Personality Disorder: Considerations for Inclusion in the Massachusetts Parity List of "Biologically-Based" Disorders

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Abstract Borderline Personality Disorder (BPD) is a common and severe mental illness that is infrequently included under state mental health parity statutes. This review considers BPD parity, using the Massachusetts mental health parity statute as a model. While BPD can co-occur with other disorders, studies of its heritability, diagnostic validity/reliability, and response to specific treatments indicate it is best considered an independent disorder, one that negatively impacts the patient's treatment response to comorbid disorders, particularly mood disorders. 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 improve. The findings from psychopharmacologic and other biologic treatment data, coupled with associated brain functioning findings, indicate BPD is a biologically-based disorder. Clinical data indicate that accurately diagnosing and treating BPD conserves resources and improves outcomes. Based on this analysis, insuring BPD in the same manner as other serious mental illnesses is well-founded and recommended.

Keywords Borderline personality disorder · Mental health parity · Biologically-based mental illness · Mental health policy

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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"?

Susan: "Yes, of course!" she screamed. "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 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 there alive. I explained I could not sleep, had no interest in doing anything, couldn't concentrate, and was binging 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 lbs. He did hospitalize me, and off I went with another diagnosis—type 2, whatever that means—and they gave me a whole new set of meds: antidepressants, anti-psychotics, stuff to sleep, PRN's. After 12 days I went home, but nothing had really changed. Two weeks later I cut myself again, this time more seriously, and I have been back to the ER five times since then."

"The last time I went 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 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) 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."

Introduction

As of December, 2008, the American Psychiatric Association reports that 42 states have enacted mental health parity legislation (Paula Johnson, personal communication, August

30, 2009). These statutes vary in terms of included diagnoses and requirements. Maine is the only state to specifically include personality disorders in the statute (ME Rev. Stat. Tit. 24-§2325A). In addition, several states, including Arkansas, Connecticut, Georgia, Kentucky, Missouri, Rhode Island, and South Carolina have statutory language reflecting inclusion of all DSM-IV diagnoses with various exclusions of mental retardation, learning disorders, motor and communication disorders, and caffeine-related disorders. None of these states specifically exclude personality disorders.

Massachusetts is representative of a group of states that have adopted a standard of parity for "biologically-based" disorders (MA Gen Laws Ch. 175:47B) including (1) schizophrenia; (2) schizoaffective disorder; (3) major depressive disorder; (4) bipolar disorder; (5) paranoia and other psychotic disorders; (6) obsessive–compulsive disorder; (7) panic disorder; (8) delirium and dementia; (9) affective disorders; (10) eating disorders; (11) post traumatic stress disorder; (12) substance abuse disorders; and (13) autism. The statute also allows for addition of biologically-based mental disorders appearing in the DSM that are scientifically recognized and when approved by the Commissioner of the Department of Mental Health in consultation with the Commissioner of the Division of Insurance. It was in 2009 that the legislature amended the statute to include eating disorders, post traumatic stress disorder, substance abuse disorders, and autism.

Borderline Personality Disorder (BPD) is the most commonly diagnosed personality disorder in clinical settings, and is associated with marked distress and impairment in social, occupational, and role functioning. As seen in the case study, effective diagnosis and treatment is often elusive, and the burden of suffering on patients and families can be severe. This review seeks to elaborate considerations relevant to inclusion of the disorder in the Massachusetts list of mental health parity diagnoses.

Methods

The authors collaborated to conduct searches of PubMed from 1966 to present, with a focus on the most current scientific literature. Reference lists of publications obtained were searched to identify relevant studies, and works known to the authors were included. Published literature was searched in order to give an accurate survey of the disorder's etiology and epidemiology, course, treatment, and relevant considerations for its inclusion in Massachusetts state mental health parity statutes.

Results

BPD: Diagnostic and Symptom Overview

As defined by the current Diagnostic and Statistical Manual of the American Psychiatric Association, BPD is a diagnosis given to individuals who meet five of the nine criteria listed in Table 1.

Different approaches to conceptualizing BPD have been advanced based on the clustering of symptoms [1, 2]. Whether the existing DSM criteria consistently cluster into a single syndrome or should be thought of as independent dimensions has been debated. Factor analyses [3, 4] 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),

Table 1 Borderline personality disorder criteria [7]

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

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 [5, 6].

Epidemiology

In clinical populations, BPD is found in roughly 20% of the patients in inpatient and outpatient settings [8]. 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 [9, 10]. Because BPD patients present with mood instability, they are often given a diagnosis of bipolar disorder—a diagnosis that is greatly over utilized [11]. Indeed, nearly 40% of patients who have borderline personality disorder have been previously, inaccurately diagnosed with bipolar disorder [12].

In nonclinical populations, one in every ten 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 [13]. The aggregation of eight epidemiological studies reveals that, the prevalence of BPD ranges from 1.2 to 5.9% of the general population [14]. In clinical systems, BPD is more prevalent among women (about 75%). In community samples, BPD is equally prevalent across genders [15, 16] with males more frequently found in forensic and substance abuse programs.

Specificity and Comorbidity

Findings from a prospective study of 668 patients (Collaborative Longitudinal Personality Disorders Study) indicate that posttraumatic stress disorder, substance abuse, and major depressive disorder are 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 converse is not true.

Various research studies report that BPD should not be conceptualized as a variant or extension of any other disorder (ex. depression, bipolar) [17–21]. 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 [22]. In samples of outpatients, BPD was associated most strongly with avoidant, paranoid and dependent personality disorders [23]. 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 employed, other comorbid conditions improve.

Course, Prognosis, Phenomenology, and Treatment Use

Although personality disorders are not officially diagnosed until adulthood, BPD symptoms are often observed during adolescence. Beginning with compromised psychosocial functioning, the disorder over time dramatically impairs the individual's ability to achieve normal adult developmental milestones such as academic/occupational success and mature partnering [24]. Additionally, severe psychosocial and academic performance below expectation (the outcomes of failures to modulate affect and impulse) in adolescence are associated with added risk for BPD as well as other forms of severe impairments in adulthood [24]. Among adults, severe baseline psychopathology, poor vocational functioning, a history of childhood trauma, and poor quality of relationships predict a negative prognosis [25, 26].

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 [27–30]. A core feature of BPD is self-destructive behavior, including binging and purging, substance abuse, risky sexual behavior, reckless driving and spending, and self-injury [31]. In the short term, these behaviors are attempts to regulate out-of-control emotions, but the interpersonal consequences further impair troubled relationships [32].

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 [33]. Similarly, the McLean Study of Adult Development showed that BPD patients were more intense utilizers of treatment than patients with other personality disorders [34]. In both studies, those with BPD had higher use of emergency departments. They spent more days in the hospital and in partial hospital programs—the more expensive forms of psychiatric treatments.

Patients with BPD often improve, though a subset remains chronically ill [34]. In the NIMH Collaborative Longitudinal Personality Disorders Study, 44% of patients diagnosed with BPD continued to meet diagnostic criteria two years later [35]. In a prospective 6-year follow-up of patients with BPD, 74% continued to meet the criteria during the follow-up period [36]. Among a sample of Canadian patients with BPD, after 27 years, only 8% continued to meet the diagnostic criteria [37]. Remission is multiply determined. Important contributing factors include: effective interventions; the changing severity of co-occurring psychiatric illnesses; diminished impulsivity across the lifespan; social learning; and avoidance of conflictual relationships [38–40]. Despite diagnostic remission, a significant degree of functional impairment in relationships often remains [41].

Etiology

BPD emerges from interactions between genetics and the environment.

Heritability and Familiarity

The major BPD twin study shows that genes accounted for 69% of the variance in BPD in a sample of 92 identical and 129 fraternal twins [42]. This is greater than the heritability of major depressive disorder or anxiety disorders and is similar to that for bipolar disorders [43]. Genetic risk factors for BPD have been identified but await replication [44, 45]. In nine 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 [46]. 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 [47, 48].

Environmental Factors

Childhood abuse [49], especially when severe [50] or prolonged [51], is associated with BPD. In the Collaborative Longitudinal Personality Disorders Study, the association between BPD and childhood abuse and neglect was the most robust [52]. 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 [53].

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 0.28 [54], which is moderate in magnitude. Nonetheless, a sizeable minority (20–45%) of individuals diagnosed with BPD report no history of sexual abuse [55], and most people (roughly 80%) with sexual abuse histories do not demonstrate personality disorders [56]. In a longitudinal study that followed children who experienced serious abuse, a substantial proportion showed little impairment in social, occupational, and interpersonal functioning [57].

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 [58]. Functional MRI studies using standardized tests have demonstrated differences in brain areas and functioning between people with BPD and controls [59]. Using evoked emotional response, fMRI differentiated BPD from controls with differences appearing in the amygdala, anterior cingulate and ventromedial prefrontal cortex [58]. Compared to controls, individuals with BPD have been reported to exhibit greater left amygdala activation in response to facial stimuli that involve expressions of emotion [60].

This research suggests that both the affective instability and the interpersonal hypersensitivity seen in BPD have their roots in the amygdala's sensitivity to negative emotions. In the face of this increased amygdala 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 BPD 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 [58]. Additional findings from PET research suggest individuals with comorbid BPD demonstrate (a) altered activity in the parietotemporal and anterior cingulate cortical areas of the brain [61]; (b) frontal and prefrontal hypermetabolism and hippocampus and cuneus hypometabolism compared to controls [62]; and (c) lower 5-HT synthesis capacity in corticostriatal neuro-pathways relative to healthy controls [63].

Basic cognitive testing such as visual perceptional speed and working memory also have been found to differentiate women with BPD from age- and education-matched controls [64]. Differences in neurotransmitter systems also seem to distinguish individuals with BPD from controls. Work in this area has focused on the serotonergic [65] and dopaminergic systems [66], with significant findings regarding both neurotransmitters. For example, research on the dominaregic system has uncovered specific gene variants in people with BPD [66].

Most recently, transcranial magnetic stimulation (TMS) has been used as a tool to study the pathophysiological underpinnings of BPD. In addition to parasympathetic nervous system differences [67], TMS research suggests that BPD is associated with cortical inhibition deficits [68].

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

Treatment

In October 2001, the American Psychiatric Association (APA) published practice guidelines for the treatment of patients with BPD [31]. In 2005, an update was published [69]. 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 psychotherapy. One such therapy is Dialectical Behavioral Therapy (DBT) [70]. 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 [71]. 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 [72]. In a recent study [73], researchers found that BPD individuals with co-occurring substance dependence disorders who received DBT were more likely to achieve full remission, spend more time in partial remission, and report 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 [74–76].

In addition to DBT, five 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 in suicide ideation, hopelessness, depression, number of borderline symptoms, and dysfunctional beliefs at termination [77]. STEPPS is a cognitive-behavioral systems-based intervention that appears to improve symptoms and global functioning, and diminish depression in a cost-effective way in BPD patients [78]. MBT [79] is aimed at helping patients increase their capacity to understand their own emotional states and that of 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 [80] and 6.5 years [81]. TFP [82, 83] was shown to have efficacy on par with DBT in one study [82].

Given the fact that these treatments are not universally available, especially in rural areas, a recent study comparing DBT with general psychiatric management (a combination of psychodynamically-informed therapy with medication management according to APA treatment guidelines) found both were effective [84]. Further, when MBT was compared with structured clinical management, both showed substantial improvements, although MBT showed greater improvement in clinically significant outcomes [85].

Psychopharmacology

There are many challenges involved in developing an evidence-based psychopharmacologic regimen to treat BPD. One must address comorbid disorders related to 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 (including PTSD and psychotic disorders) [86]. Untreated BPD appears to significantly worsen the course and prognosis of the Axis I disorders, such as major depressive disorder [87] and bipolar disorder [88]. BPD 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 [89]. When clinicians lack a clear understanding of the relationship amongst these factors, this can lead them to bypass practice guidelines [90, 91] and treat persons with BPD with psychopharmacologic abandon, chasing various symptoms without a clear formulation of the illness complex. This process increases prescribing and its attendant burden of multiple medications, worsens care, and increases costs—a process both harmful to patients and taxing to the finite resources in healthcare finances [92, 93].

While not always easy to achieve, a rational psychopharmacologic approach to BPD can improve outcomes; patients can respond to medications that target key symptoms of BPD such as affective dysregulation, impulsive behaviors, and cognitive/perceptual disturbances [94]. The relevant literature is organized by considering four classes of medications:

Antidepressants The antidepressants including tricyclics, MAO inhibitors and SSRIs have been researched in the psychopharmacologic treatment of BPD [95–98]. Findings from these studies indicate the evidence to support the effectiveness of tricyclics is poor. 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 [94]. But as Abraham and Calabrese [95] recently pointed out, the role of the SSRI's as first-line psychopharmacologic treatment for BPD may warrant re-evaluation in favor of antipsychotic and mood stabilizer medications, and a meta-analysis shows their effects are greatest with anxiety and anger but negligible with depressed mood and impulsivity [96].

Antipsychotics 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 [95]. Second generation antipsychotics have been studied more recently, and olanzapine has been investigated most extensively [95]. One study demonstrated olanzapine was superior to fluoxetine as a monotherapeutic intervention [100], and another study demonstrated the superiority of DBT with olanzapine as compared to DBT with placebo [101]. Open-label studies of quetiapine [102], aripiprazole [103] and IM risperidone [104] have shown clinical and functional improvement sufficient to warrant further study. Soloff [99] 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 atypical antipsychotic medications, their metabolic effects raise concern for their use in obese patients (and perhaps those with moderately elevated BMI's) who meet criteria for BPD [105–108].

Mood Stabilizers With the exception of lithium carbonate, which seems not to be efficacious in the treatment of BPD [95, 98, 109], conclusions about mood stabilizers remain uncertain. Based on the studies to date, Abraham and Calabrese [95] supported their use, Paris [98] failed to support their use, and Soloff [99] opined that positive outcomes may be more attributable to the mood stabilizers' effects on impulsivity than to changes in mood, a view supported through meta-analysis [96]. In double-blind studies with random assignment, divalproex [110] was shown to reduce impulsive aggression. Findings from a retrospective chart review suggested lamotrigine [111] was a "safe and effective" option for addressing affective instability in BPD.

Benzodiazepines Although benzodiazepine use is widespread in the treatment of BPD [93], there is no evidence to support benzodiazepine use [99]. Of the few placebo-controlled studies of a benzodiazepine, one (in this case alprazolam) actually showed symptomatic worsening with increased frequency of severe behavioral dyscontrol [95, 99]. This is an example where accurate diagnosis of BPD can help. If a patient fails to be appropriately diagnosed as having BPD, benzodiazepines (which can worsen affect regulation, increase behavioral disinhibition, and impair cognition) would most likely worsen the condition in many, if not most cases.

Polypharmacy

An important gap in clinical services for individuals with BPD is the failure to make this diagnosis, with a result of polypharmacy and all its attendant negative consequences [98, 112].

Is BPD a "Biologically-Based" Disorder?

The Massachusetts Department of Mental Health refers 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 [41]. The disorder demonstrates heritability on par with bipolar disorder and higher than major depression. MRI studies have identified structural brain abnormalities and Functional MRI studies have confirmed neurophysiological brain dysfunctions. Neuroendocrine correlates (including significant alterations in the hypothalamic-pituitary axis [113]) 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 Massachusetts 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 [114, 115]. 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 are explored next.

Implications of Including BPD as a "Biologically-Based Disorder" for Massachusetts Parity

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 treatment are already in the system of care, frequently under another diagnosis, such as bipolar disorder [12]. The problem with the current system of inaccurate diagnosis is that patients are frequently 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

Table 2 One year health care costs per patient

	DBT	TAU
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
Total	$8,607	$17,682

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 effective specialized 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 many comorbidities) would improve care, services should be more cost effective when patients can be engaged in evidence-based treatments. Take DBT as an example. Estimates are that DBT costs approximately 50% as much as treatment as usual (TAU) for BPD (see Table 2) [116].

Regarding MBT, data are clear that this treatment reduces hospitalization, self-injury, and leads to long-term patient stabilization. In a cost-comparison with TAU, during the course of treatment TAU showed cost equal to the more expensive (per unit of treatment) MBT, and overall healthcare costs decreased in the MBT group for hospitalizations and emergency department visits. More importantly, 18 months after the completion of treatment, costs for continued care in the MBT group were 20% of costs for those given treatment TAU [117].

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 other personality disorders (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 an 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 controlled through quality management practices (internal to an agency) and quality oversight (external to an agency). There are methods to set up cost disincentives for intentional misdiagnosis, such as fines, decreased reimbursement rates, and loss of CMS certification.

A related concern is that were BPD to be added to the Massachusetts 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 are 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 offer 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 [118]. One could argue that because of observed periods of sustained remission, BPD does not warrant specific identification in the Massachusetts parity law. This line of argument should be dismissed given the same course of symptom fluctuation apparent, for example, in individuals compliant with treatment with bipolar disorder. Moreover, the potential for treatments designed specifically for BPD to greatly diminish health care resource utilization and to create enduring remission offer strong reasons to upgrade the use of the diagnosis and thereby the quality of treatment persons with BPD receive.

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 [119]. A similar trend could occur in BPD where 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, given the robust evidence for their efficacy in contrast with the weaker evidence for psychopharmacologic efficacy.

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 as a reimbursable diagnosis. And with symptomatic improvement and better psychosocial performance, stigma around the diagnosis and the individuals who have the disorder could actually decrease. For example, that leonine roar so often and so long heard in ER's and hospitals [120], "that borderline is here *again*", could be altered to respectful greetings to a person rarely seen at the emergency department or hospital inpatient unit.

Conclusion

This review synthesizes the current scientific literature on Borderline Personality Disorder with an eye toward determining if the disorder should be included as a parity diagnosis in Massachusetts (and potentially other states). Findings are that 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 treatment responses indicate it is best considered an independent disorder that negatively affects the patient's treatment response to comorbid disorders, particularly mood disorders.

BPD 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 disproportionately high users of treatment, especially emergency departments and inpatient hospitalizations—the most expensive forms of psychiatric treatment. While some patients remain chronically symptomatic, the majority improve. Within six years of follow-up, a substantial majority no longer meet 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 concordance rates for depressive or anxiety disorders. Functional MRI studies of BPD patients show abnormalities in the amygdala and the prefrontal cortex consistent with the high emotional arousal and decreased impulse control seen in the disorder.

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 not only to worsen, but also to be worsened by inappropriate treatment.

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 improves outcomes and conserves resources. Based on this analysis, insuring BPD in the same manner as other serious mental illnesses is well founded and recommended.

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References

1. Skodol AE, Oldham JM, Bender DS, et al.: Dimensional representations of DSM-IV personality disorders: Relationships to functional impairment. American Journal of Psychiatry 162:1919–1925, 2005
2. Trull TJ, Widiger TA, Lynam DR, et al.: Borderline personality disorder from the perspective of general personality functioning. Journal of Abnormal Psychology 112:193–202, 2003
3. Sanislow CA, Grilo CM, McGlashan TH: Factor analysis of the DSM-III-R borderline personality disorder criteria in psychiatric inpatients. American Journal of Psychiatry 157:1629–1633, 2000
4. Sanislow CA, Grilo CM, Morey LC, et al.: Confirmatory factor analysis of DSM-IV criteria for borderline personality disorder: Findings from the collaborative longitudinal personality disorders study. American Journal of Psychiatry 159:284–290, 2002
5. Clifton A, Pilkonis PA: Evidence for a single latent class of diagnostic and statistical manual of mental disorders borderline personality pathology. Comprehensive Psychiatry 48:70–78, 2007
6. Fossati A, Madeddu F, Maffei C: Borderline personality disorder and childhood sexual abuse: A meta-analytic study. Journal of Personal Disorders 13:268–280, 1999

7. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th edn., text revision. Washington, DC, American Psychiatric Association, 2000
8. Gunderson JG, Links P: Borderline Personality Disorder. In: Gabbard GO (Ed) Treatment of Psychiatric Disorders, 4th edn. Washington, DC, American Psychiatric Press, 2007
9. Zimmerman M, Mattia JI: Differences between clinical and research practices in diagnosing borderline personality disorder. American Journal of Psychiatry 156:1570–1574, 1999
10. Zimmerman M, Mattia JI: Axis I diagnostic comorbidity and borderline personality disorder. Comprehensive Psychiatry 40:245–252, 1999
11. Goldberg JF, Garno JL, Callahan AM, et al.: Overdiagnosis of bipolar disorder among substance use disorder patients with mood instability. Journal of Clinical Psychiatry 8:e1–e7, 2008
12. Zimmerman M, Cuggero CJ, Chelminski I, et al.: Psychiatric diagnoses in patients previously overdiagnosed with bipolar disorder. Journal of Clinical Psychiatry 71:6–31, 2010
13. Lenzenweger MF: Epidemiology of personality disorders. Psychiatric Clinics of North America 31:395–403, 2008
14. Torgersen S: Epidemiology. In: Oldham JM, Skodol AE, Bender DS (Eds) Textbook of Personality Disorders. Washington, DC, American Psychiatric Publishing, 2005
15. Torgersen S, Kringlen E, Cramer V: The prevalence of personality disorders in a community sample. Archives of General Psychiatry 58:590–596, 2001
16. Zlotnick C, Rothschild L, Zimmerman M: The role of gender in the clinical presentation of patients with borderline personality disorder. Journal of Personality Disorders 16:277–282, 2002
17. Deltito J, Martin L, Riefkohl J, et al.: Do patients with borderline personality disorder belong to the bipolar spectrum? Journal of Affective Disorders 67:221–228, 2001
18. Smith DJ, Muir WJ, Blackwood DH: Is borderline personality disorder part of the bipolar spectrum? Harvard Review of Psychiatry 12:133–139, 2004
19. Koenigsberg HW, Harvey PD, Mitropoulou V, et al.: Characterizing affective instability in borderline personality disorder. American Journal of Psychiatry 159:784–788, 2002
20. Magill CA: The boundary between borderline personality disorder and bipolar disorder: Current concepts and challenges. Canadian Journal of Psychiatry 49:551–556, 2004
21. Paris J: Borderline or bipolar? Distinguishing borderline personality disorder from bipolar spectrum disorders. Harvard Review of Psychiatry 12:140–145, 2004
22. Zanarini MC, Frankenburg FR, Dubo ED, et al.: Axis II comorbidity of borderline personality disorder. Comprehensive Psychiatry 39:296–302, 1998
23. Conklin CZ, Westen D: Borderline personality disorder in clinical practice. American Journal of Psychiatry 162:867–875, 2005
24. Winograd G, Cohen P, Chen H: Adolescent borderline symptoms in the community: Prognosis for functioning over 20 years. Journal of Child Psychological Psychiatry 49:933–941, 2008
25. Zanarini MC, Frankenburg FR, Hennen J: Prediction of the 10-year course of borderline personality disorder. American Journal of Psychiatry 163:827–832, 2006
26. Gunderson JG, Lyons-Ruth K: BPD's interpersonal hypersensitivity phenotype: A gene-environment-developmental model. Journal of Personality Disorders 22:22–41, 2008
27. Bolton S, Gunderson JG: Distinguishing borderline personality disorder from bipolar disorder: Differential diagnosis and implications. American Journal of Psychiatry 153:1202–1207, 1996
28. Black DW, Blum N, Pfohl B, et al.: Suicidal behavior in borderline personality disorder: Prevalence, risk factors, prediction, and prevention. Journal of Personality Disorders 18:226–239, 2004
29. Oldham JM: Borderline personality disorder and suicidality. American Journal of Psychiatry 163:1, 2006
30. Skodol WE, Gunderson JG, Pfohl B, et al.: The borderline diagnosis I: Psychopathology, comorbidity, and personality structure. Biological Psychiatry 51:936–950, 2002
31. Oldham JM, Gabbard GO, Goin MK, et al.: Practice Guidelines for the Treatment of Patients with Borderline Personality Disorder. Washington, DC, American Psychiatric Association, 2001
32. Holm AL, Severinsson E: The emotional pain and distress of borderline personality disorder: A review of the literature. International Journal of Mental Health Nursing 17:27–35, 2008
33. Bender DS, Skodol AE, Pagano ME, et al.: Prospective assessment of treatment use by patients with personality disorders. Psychiatric Services 57:254–257, 2006
34. Zanarini MC, Frankenburg FR, Hennen J, et al.: The McLean study of adult development (MSAD): Overview and implications of the first six years of prospective follow-up. Journal of Personality Disorders 19:505–523, 2005
35. Grilo CM, Shea MT, Sanislow CA, et al.: Two-year stability and change of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. Journal of Consulting and Clinical Psychology (in press)

36. Zanarini MC, Frankenburg FR, Hennen J, et al.: The longitudinal course of borderline psychopathology: 6-Year prospective follow-up of the phenomenology of borderline personality disorder. American Journal of Psychiatry 160:274–283, 2003
37. Paris J, Zweig-Frank H: A 27-year follow-up of patients with borderline personality disorders. Psychiatric Quarterly 71:291–307, 2000
38. Paris J: Implications of long-term outcome research for the management of patients with borderline personality disorder. Harvard Review of Psychiatry 10:315–323, 2002
39. Stevenson J, Meares R, Comerford A: Diminished impulsivity in older patients with borderline personality disorder. American Journal of Psychiatry 160:165–166, 2003
40. Warner MB, Morey LC, Finch JF: The longitudinal relationship of personality traits and disorders. Journal of Abnormal Psychology 113:217–227, 2004
41. Skodol AE, Pagano ME, Bender DS, et al.: Stability of functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder over two years. Psychological Medicine 35:443–451, 2004
42. Torgersen S, Lygren S, Oien PA, et al.: A twin study of personality disorders. Comprehensive Psychiatry 41:416–425, 2000
43. Kendler KS, Greenspan RJ: The nature of genetic influence on behavior: Lessons from "simpler" organisms. American Journal of Psychiatry 163:1683–1694, 2006
44. Ni X, Chan K, Bulgin N, et al.: Association between serotonin transporter gene and borderline personality disorder. Journal of Psychiatric Research 40:448–453, 2006
45. Distel MA, Hottenga JJ, Trull TJ et al.: Chromosome 9: Linkage for borderline personality disorder features. Psychiatric Genetics 18:302–307, 2008
46. White CN, Gunderson JG, Zanarini MC, et al.: Family studies of borderline personality disorder: A review. Harvard Review of Psychiatry 11:8–19, 2003
47. Silverman JM, Pinkham L, Horvath TB, et al.: Affective and impulsive personality disorder traits in the relatives of patients with borderline personality disorder. American Journal of Psychiatry 148:1378–1385, 1991
48. Zanarini MC, Frankenburg FR, Yong L, et al.: Borderline psychopathology in the first-degree relatives of borderline and axis II comparison probands. Journal of Personality Disorders 18:439–447, 2004
49. Trull TJ: Structural relations between borderline personality disorder features and putative etiological correlates. Journal of Abnormal Psychology 110:471–481, 2001
50. Zanarini MC, Yong L, Frankenburg FR, et al.: Severity of reported childhood sexual abuse and its relationship to severity of borderline psychopathology and psychosocial impairment among borderline inpatients. Journal of Nervous and Mental Disease 190:381–387, 2002
51. Rinne T, de Kloet ER, Wouters L, et al.: Hyperresponsiveness of hypothalamic-pituitary-adrenal axis to combined dexamethasone/corticotrophin-releasing hormone challenge in female borderline personality disorder subject with a history of sustained childhood abuse. Biological Psychiatry 52:1102–1112, 2002
52. Battle CL, Shea MT, Johnson DM, et al.: Childhood maltreatment associated with adult personality disorders: Findings from the collaborative longitudinal personality disorders study. Journal of Personality Disorders 18:193–211, 2004
53. Bandelow B, Krause J, Wedekind D, et al.: Early traumatic life events, parental attitudes, family history, and birth risk factors in patients with borderline personality disorder and healthy controls. Psychiatry Research 134:169–179, 2005
54. Fossati A, Maffei C, Bagnato M, et al.: Latent structure analysis of DSM-IV borderline personality disorder criteria. Comprehensive Psychiatry 40:72–79, 1999
55. Goodman M, Yehuda R: The relationship between psychological trauma and borderline personality disorder. Psychiatric Annals 32:337–346, 2002
56. Paris J: Does childhood trauma cause personality disorders in adults? Canadian Journal of Psychiatry 43:148–153, 1998
57. McGloin JM, Widom CS: Resilience among abused and neglected children grown up. Development and Psychopathology 13:1021–1038, 2001
58. Schmahl C, Bremner JD: Neuroimaging in borderline personality disorder. Journal of Psychiatric Research 40:419–427, 2006
59. Völlm B, Richardson P, Stirling J, et al.: Neurobiological substrates of antisocial and borderline personality disorder: Preliminary results of a functional fMRI study. Criminal Behaviour and Mental Health 14:39–54, 2004
60. Donegan NH, Sanislow CA, Blumberg HP, et al.: Amygdala hyperreactivity in borderline personality disorder: Implications for emotional dysregulation. Biological Psychiatry 54:1284–1293, 2003

61. Oquendo MA, Krunic A, Parsey RV, et al.: Positron emission tomography of regional brain metabolic responses to a serotonergic challenge in major depressive disorder with and without borderline personality disorder. Neuropsychopharmacology 30:1163–1172, 2005
62. Juengling FD, Schmahl C, Hesslinger B, et al.: Positron emission tomography in female patients with borderline personality disorder. Journal of Psychiatric Research 37:109–115, 2003
63. Leyton M, Okazawa H, Diksic M, et al.: Brain regional [^{11}C] methyl-l-tryptophan trapping in impulsive subjects with borderline personality disorder. The American Journal of Psychiatry 158:775–782, 2001
64. Stevens A, Burkhardt M, Hautzinger M, et al.: Borderline personality disorder: An impaired visual perception and working memory. Psychiatry Research 125:257–267, 2003
65. Coccaro EF, Siever LJ, Klar HM, et al.: Serotonergic studies in patients with affective and personality disorders. Archives of General Psychiatry 46:587–599, 1989
66. Joyce PR, McHugh PC, McKenzie JM, et al.: A dopamine transporter polymorphism is a risk factor for borderline personality disorder in depressed patients. They Psychological Medicine 36:807–813, 2006
67. Austin MA, Riniolo TC, Porges SW: Borderline personality disorder and emotion regulation: Insights from the Polyvagal Theory. They Brain and Cognition 65:69–76, 2007
68. Barnow S, Völker KA, Möller B, et al.: The neurophysiological correlates of borderline personality disorder: A transcranial magnetic stimulation study. Biological Psychiatry 65:313–318, 2009
69. Oldham JM: Guideline Watch: A Practice Guideline for the Treatment of Patients with Borderline Personality Disorder. Washington, DC, American Psychiatric Association, 2005. http://www.psychiatryonline.com/pracGuide/pracGuideTopic_13.aspx
70. Linehan MM: Cognitive Behavioral Treatment of Borderline Personality Disorder. New York, Guilford Press, 1993
71. van den Bosch LM, Verheul R, Schippers GM, et al.: Dialectical behavior therapy of borderline patients with and without substance use problems: Implementation and longterm effects. Addictive Behaviors 27:911–923, 2002
72. Linehan MM, Dimeff LA, Reynolds SK, et al.: Dialectical behavior therapy versus comprehensive validation therapy plus 12-step for the treatment of opioid dependent women meeting criteria for borderline personality disorder. Drug and Alcohol Dependence 67:13–26, 2002
73. Harned MS, Chapman AL, Dexter-Mazza ET, et al.: Treating co-occurring Axis I disorders in recurrently suicidal women with borderline personality disorder: A 2-year randomized trial of dialectical behavior therapy versus community treatment by experts. Journal of Consulting and Clinical Psychology 76:1068–1075, 2008
74. Bohus M, Haaf B, Simms T, Limberger MF, et al.: Effectiveness of inpatient dialectical behavioral therapy for borderline personality disorder: A controlled trial. Behavior Research and Therapy 42:487–499, 2004
75. Koons C, Robins CJ, Tweed JL, et al.: Efficacy of dialectical behavior therapy in women veterans with borderline personality disorder. Behavior Therapy 32:371–390, 2001
76. Verheul R, van den Bosch LM, Koeter MW, et al.: Dialectical behaviour therapy for women with borderline personality disorder: 12-Month, randomised clinical trial in the Netherlands. British Journal of Psychiatry 182:135–140, 2003
77. Brown GK, Newman CF, Charlesworth SE: An open clinical trial of cognitive therapy for borderline personality disorder. Journal of Personality Disorder 18:257–271, 2004
78. Blum N, Pfohl B, John DS, et al.: STEPPS: A cognitive-behavioral systems–based group treatment for outpatients with borderline personality disorder—a preliminary report. Comprehensive Psychiatry 43:301–310, 2002
79. Bateman A, Fonagy P: Effectiveness of partial hospitalization in the treatment of borderline personality disorder: A randomized controlled trial. American Journal of Psychiatry 156:1563–1569, 1999
80. Bateman A, Fonagy P: Treatment of BPD with psychoanalytically oriented partial hospitalization: An 18-month follow-up. American Journal of Psychiatry 158:36–42, 2001
81. Bateman A, Fonagy P: Eight-year follow-up of patients treated for borderline personality disorder: Mentalization-based treatment versus treatment as usual. American Journal of Psychiatry 165:631–638, 2008
82. Clarkin JF, Levy KN, Lenzenweger MF, Kernberg OF: Evaluating three treatments for borderline personality disorder: A multiwave study. American Journal of Psychiatry 164:922–928, 2007
83. Giesen-Bloo J, van Dyck R, Spinhoven P, et al.: Outpatient psychotherapy for borderline personality disorder: Randomized trial of schema-focused therapy vs. transference-focused psychotherapy. Archives of General Psychiatry 63:649–658, 2006

84. McMain SF, Links PS, Gnam WH et al.: A randomized trial of dialectical behavior therapy versus general psychiatric management for borderline personality disorder. American Journal of Psychiatry 166:1365–1374, 2009
85. Bateman A, Fonagy P: Randomized controlled trial of outpatient mentalization-based treatment versus structured clinical management for borderline personality disorder. American Journal of Psychiatry 166:1355–1364, 2009
86. American Psychiatric Association: Practice Guidelines for the Treatment of Psychiatric Disorders: Compendium 2002. American Psychiatric Association, Washington, DC, 2002
87. Gunderson JG, Morey LC, Stout RL, et al.: Major depressive disorder and borderline personality disorder revisited: Longitudinal interactions. Journal of Clinical Psychiatry 65:1049–1056, 2004
88. Neves FS, Malloy-Diniz LF, Corrêa H: Suicidal behavior in bipolar disorder: What is the influence of psychiatric comorbidities? Journal of Clinical Psychiatry 70:13–18, 2009
89. Frankenburg FR, Zanarini MC: The association between borderline personality disorder and chronic medical illnesses, poor health-related lifestyle choices, and costly forms of health care utilization. Journal of Clinical Psychiatry 65:1660–1665, 2004
90. American Psychiatric Association: Practice Guideline for the Treatment of Patients with Borderline Personality Disorder. American Journal of Psychiatry 158(Suppl):1–52, 2001
91. Herpertz SC, Zanarini MC, Schulz CS, et al.: WFSBP task force on personality disorders: World federation of societies of biological psychiatry (WFSBP) guidelines for biological treatment of personality disorders. World Journal of Biological Psychiatry 8:212–244, 2007
92. Sansone RA, Rytwinski D, Gaither GA: Borderline personality and psychotropic medication prescription in an outpatient psychiatry clinic. Comprehensive Psychiatry 44:454–458, 2003
93. Pascual JC, Córcoles D, Castaño J, et al.: Hospitalization and pharmacotherapy for borderline personality disorder in a psychiatric emergency service. Psychiatric Services 58:1199–1204, 2007
94. Oldham JM, Bender DS, Skodol AE, et al.: Testing an APA practice guideline: Symptom-targeted medication utilization for patients with borderline personality disorder. Journal of Psychiatric Practice 10:156–161, 2004
95. Abraham PF, Calabrese JR: Evidenced-based pharmacologic treatment of borderline personality disorder: A shift from SSRIs to anticonvulsants and atypical antipsychotics? Journal of Affective Disorders 111:21–30, 2008
96. Ingenhoven T, Lafay P, Rinne T, et al.: Effectiveness of pharmacotherapy for severe personality disorders: Meta-analysis of randomized controlled trials. Journal of Clinical Psychiatry 71:14–25, 2010
97. Bellino S, Paradiso E, Bogetto F: Efficacy and tolerability of pharmacotherapies for borderline personality disorder. CNS Drugs 22:671–692, 2008
98. Paris, J: Clinical trials of treatment for personality disorders. Psychiatric Clinics of North America 31:517–526, 2008
99. Soloff PH: Psychopharmacology of borderline personality disorder. Psychiatric Clinics of North America 23:169–192, 2000
100. Zanarini MC, Frankenburg FR, Parachini, EA: A preliminary, randomized trial of fluoxetine, olanzapine, and the olanzapine-fluoxetine combination in women with borderline personality disorder. Journal of Clinical Psychiatry 65:903–907, 2004
101. Soler J, Pascual JC, Campins J, et al.: Double-blind, placebo-controlled study of dialectical behavior therapy plus olanzapine for borderline personality disorder. American Journal of Psychiatry 162:1221–1224, 2005
102. Roepke S, Merkl A, Dams A, et al.: Preliminary evidence of improvement of depressive symptoms but not impulsivity in cluster B personality disorder patients treated with quetiapine: An open label trial. Pharmacopsychiatry 41:176–181, 2008
103. Nickel MK, Muehlbacher M, Nickel C, et al.: Aripiprazole in the treatment of patients with borderline personality disorder: A double-blind, placebo-controlled study. American Journal of Psychiatry 163:833–838, 2006
104. Díaz-Marsá M, Galian M, Montes A, et al.: Long-acting injectable risperidone in treatment resistant borderline personality disorder. A small series report. Actas Espanolas de Psiquiatria 36:70–74, 2008
105. Sansone RA, Sansone LA, Wiederman MW: The comorbidity, relationship and treatment implications of borderline personality and obesity. Journal of Psychosomatic Research 43:541–543, 1997
106. Sansone RA, Schumacher D, Wiederman MW, et al.: The prevalence of binge eating disorder and borderline personality symptomatology among gastric surgery patients. Eating Behaviors 9:197–202, 2008
107. Sansone RA, Wiederman MW, Sansone LA: The prevalence of borderline personality disorder among individuals with obesity: A critical review of the literature. Eating Behaviors 1:93–104, 2000

108. Frankenburg FR, Zanarini MC: Obesity and obesity-related illnesses in borderline patients. Journal of Personality Disorders 20:71–80, 2006
109. Links PS, Steiner M, Boiago I, et al.: Lithium therapy for borderline patients: Preliminary findings. Journal of Personality Disorders 4:173–181, 1990
110. Hollander E, Swann AC, Coccaro EF, et al.: Impact of trait impulsivity and aggression on divalproex versus placebo response in borderline personality disorder. American Journal of Psychiatry 162:621–624, 2005
111. Weinstein W, Jamison KL: Retrospective case review of Lamotrigine use for affective instability of borderline personality disorder. CNS Spectrums 12:207–210, 2007
112. Zanarini MC: Update on pharmacotherapy of borderline personality disorder. Current Psychiatry Reports 6:66–70, 2004
113. Zimmerman DJ, Choi-Kain LW: The hypothalamic-pituitary-adrenal axis in borderline personality disorder: A review. Harvard Review of Psychiatry 17:167–183, 2009
114. New AS, Triebwasser J, Charney DS: Review: The case for shifting borderline personality disorder to Axis I. Biological Psychiatry 64:653–659, 2008
115. Gunderson JG: Borderline personality disorder: Ontogeny of a diagnosis. American Journal of Psychiatry 166:530–539, 2009
116. Linehan MM, Heard HL: Borderline Personality Disorder: Costs, Course and Treatment Outcomes In: Miller N, Magruder K (Eds) The Cost-Effectiveness of Psychotherapy: A Guide for Practitioners, Researchers and Policy Makers. New York, Oxford Press, 1999
117. Bateman A, Fonagy P: Health service utilization costs for borderline personality disorder patients treated with psychoanalytically oriented partial hospitalization versus general psychiatric care. American Journal of Psychiatry 160:169–171, 2003
118. Livesley WJ: Research trends and directions in the study of personality disorder. Psychiatric Clinics of North America 31:545–559, 2008
119. Olfson M, Marcus SC, Pincus HA: Trends in office-based psychiatric practice. American Journal of Psychiatry 156:451–457, 1999
120. Geller JL, Brandzel M: Addressing the borderline's repetitive misuse of the state hospital. Psychiatric Quarterly 55:275–278, 1983

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