

Wesleyan University

From the SelectedWorks of Charles A. Sanislow

February 2012

Long-Term Outcomes in Borderline Psychopathology: Old Assumptions, Current Findings, and New Directions

Contact
Author

Start Your Own
SelectedWorks

Notify Me
of New Work



Available at: http://works.bepress.com/charles_sanislow/106

Long-Term Outcomes in Borderline Psychopathology: Old Assumptions, Current Findings, and New Directions

Charles A. Sanislow · Katherine L. Marcus ·
Elizabeth M. Reagan

Published online: 3 December 2011
© Springer Science+Business Media, LLC 2011

Abstract Borderline personality disorder (BPD) and historical variants of the diagnosis were long held to represent an intractable syndrome of psychopathology consisting of interpersonal, intrapsychic, and affective disturbances. For years, patients labeled “borderline” were regarded pejoratively due at least in part to the lack of effective treatments. Prospective data from recent naturalistic follow-along studies along with the development of treatments with empirically demonstrated efficacy have changed how BPD is viewed. It is now less common to hide the diagnosis from the patient, and BPD has become a useful label to guide the treatment process and help the patient make sense of his or her suffering. Although it is now accepted that BPD is a treatment-responsive disorder and that remission is the norm, more work is needed to help patients achieve a higher level of functioning, and targeting persistent trait-like features suggests new directions for future efforts in treatment development.

Keywords Borderline · Borderline personality disorder · BPD · Long-term outcome · Functioning · Functional impairment · Psychosocial impairment · Occupational impairment

Introduction

For many years, the prevailing view on the outcome for borderline personality and borderline personality disorder

(BPD) was that there was little if any hope for improvement other than a “burnout” of the syndrome in which tumult associated with the suffering eventually waned, often accompanied by social isolation [1]. Even in the recent past, it was not unusual for clinicians to refer to the borderline diagnosis as a “wastebasket category,” presumably because it captured a heterogeneous array of varied symptoms and very difficult-to-deal-with problematic behaviors [2]. Such a label fostered negative connotations for the diagnosis and for the patient. One of the authors of the present article (Dr. Sanislow) recalls from the early-1990s a supervisor instructing on the art of diagnosis: “A schizoaffective is what you call a really bad borderline.” So axiomatic were these pejorative notions and the idea that those diagnosed as borderline were deemed untreatable. It was not until 1998 that the first integrated summary of treatment outcome findings was published [3].

Much has changed in the past 15 or so years. Rich, prospective data are now available on the natural course of BPD and empirical clarification of the varied patterns of diagnostic co-occurrence. Three major follow-along studies of personality disorders including borderline have been carried out: the Collaborative Longitudinal Study of Personality Disorders (CLPS) [4, 5], the Longitudinal Study of Personality Disorders (LSPD) [6, 7], and the McLean Study for Adult Development (MSAD) [8, 9]. The mid-1990s also marked a renewed interest in treatment development for BPD [10]. In the past two decades, a variety of treatment approaches have been developed, studied, and empirically validated. A variety of psychotherapies with empirically demonstrated efficacy have been developed (eg, dialectical behavioral therapy [DBT] [11–13], mentalization [14, 15], and transference-focused therapy [16], as well as cognitive behaviorally based group treatments (eg, Systems Training for Emotional Predictability and Problem Solving [STEPPS] [17, 18] and schema-focused approaches [19]). These studies are a testa-

C. A. Sanislow (✉) · K. L. Marcus · E. M. Reagan
Department of Psychology, Wesleyan University,
207 High Street/Judd Hall,
Middletown, CT 06459, USA
e-mail: csanislow@wesleyan.edu

ment to the interest in clarifying and treating BPD. Prospective data inform the natural course of the disorder in greater detail than has ever been demonstrated before, and the treatment studies provide a menu of treatment options for those suffering from BPD.

Once a diagnosis that clinicians were routinely instructed to hide from their patients, the current prevailing wisdom is that it is of therapeutic benefit to educate the borderline patient about his or her diagnosis to help him or her understand his or her experiences and better collaborate in the treatment process. One study found that when individuals are diagnosed with BPD and then educated about the disorder to the point of understanding the nature of it, and also given new coping strategies to deal with the behaviors related to BPD, severity of symptoms decreased over a short period (16 weeks). Thus, it appears that when people are presented with an understanding of their psychopathology and are better able to recognize and effectively manage their symptoms, they can thereby achieve more of a sense of control over their behaviors and thoughts [20].

Together, these treatment approaches provide strong support that BPD is a treatable condition, and that clinical improvement is possible, but what about the long-term outcome? First, we take a brief look at how the diagnosis has shaped our views of the disorder and its prognosis.

The Borderline Diagnosis

How the long-term outcome of BPD is understood is dependent on how the disorder is conceptualized and further suggests paths for how to improve treatment outcomes. Historically, the borderline diagnosis was seen as a wide-reaching syndrome, and outcomes were largely negative. As the diagnostic specificity of the diagnosis narrowed, outcomes appeared to become more favorable. It is important to understand those aspects of changing views of BPD outcome that might be related to changes in the diagnosis. On the other hand, improvements in diagnostic efficiency likely have enhanced treatment development and thereby improved outcomes.

The term *borderline* has undergone many changes from its introduction to its current definition, and more change is anticipated for the *DSM-5*. Of note, the *syndrome* has varied in diagnostic specificity (ie, the range of psychopathology encompassed), and these variations likely have influenced the view of the outcome of the disorder. Psychoanalyst Adolph Stern introduced the term *border line* in 1938 to describe a group of patients who fit neither the neurotic nor psychotic classification of psychopathology and were characterized by their resistance to psychoanalytic treatment [21]. It is interesting to note in passing that the lack of structure inherent in an analytic approach shares similarities with the experience of “invalidation” argued to be a key component

in the developmental history of those with the modern diagnosis [13].

The psychopathology encompassed by the borderline label pre-*DSM-III* is remarkable in its breadth. Several diagnostic terms were used through the years to capture where within a dimension of severity (from neurotic to psychotic) a patient fell, sometimes colloquially referred to as *north* or *south* of the border to suggest a greater or lesser degree of “borderline.” Diagnostic labels such as pseudo-neurotic schizophrenia and ambulatory schizophrenia captured the more extreme side of borderline, whereas borderline personality organization [22] perhaps best represented the centrality of the borderline concept for many years. With the introduction of the Feighner Criteria [23], later the Research Diagnostic Criteria [24], came some debate over whether borderline was a variant of an affective disorder or more related to the psychotic spectrum. Clarifications by Gunderson and colleagues [25, 26], along with work by Spitzer and colleagues [27] led to the “splitting” of borderline into borderline and schizotypal personality disorders with the *DSM-III*. Some debate lingered over whether or not brief episodes of thought disturbances or dissociative experiences were part of the post-*DSM-III* syndrome of borderline, and this feature was recaptured for the diagnosis with the addition of a ninth criterion in the *DSM-IV*.

Briefly, the BPD diagnosis (*DSM-IV-TR*) presently consists of nine criteria: “frantic efforts to avoid real or imagined abandonment,” “a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation,” “identity disturbance: markedly and persistently unstable self-image or sense of self,” “impulsivity in at least two areas that are potentially self-damaging (eg, spending, sex, substance abuse, reckless driving, binge eating),” “recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior,” “affective instability due to marked reactivity of mood (eg, intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days),” “chronic feelings of emptiness,” “inappropriate anger or difficulty controlling anger (eg, frequent displays of temper, constant anger, recurrent physical fights),” and finally “transient, stress-related paranoid ideation or severe dissociative symptoms.”

The presence of at least 5 of the above nine criteria is required for a patient to meet criteria for the current *DSM-IV-TR* BPD diagnosis. Thus, the present diagnosis continues to capture—by definition—the heterogeneous nature of the syndrome. According to the *DSM-IV-TR* diagnostic algorithm, two people may share the diagnosis but share only one criterion in common. Literally, there are 256 permutations of the criteria set for which the diagnosis can be achieved! It is clear that the diagnosis has been narrowed, but the heteroge-

neity of those who might meet criteria has not necessarily been reduced. This raises the question: what is the “core” of BPD? As is the case for all personality disorders, the *DSM-IV-TR* stipulates for a BPD diagnosis, the criteria must be: “... an enduring pattern of inner experience and behavior ...” that is “... inflexible and pervasive ...” and “... stable and of long duration ...” It is “... present since adolescence or early adulthood ...” and “... not better accounted for as a manifestation of another mental disorder ...” or “... the direct physiological effects of a substance.”

By definition, the *DSM-IV-TR* sounds as if remission would be all but impossible. Data from earlier studies that date back from before the *DSM-IV* provided much of the basis for this viewpoint. Overall, patients with BPD were generally functioning at or below average [28]. The only way that interpersonal stability was achieved was through social isolation and avoiding intimacy [1]. On average, patients frequently were rehospitalized, with studies suggesting upward of 50% or more [29, 30]. Although some studies suggested modest gains in social improvements for some patients [31, 32], the impairment appeared largely stable. In sum, symptomatic behaviors tended to remain stable or improve only slightly, and the diagnosis remained stable. However, this basic notion has been challenged by the recent wave of longitudinal studies. We now turn to notable findings from the most recent major prospective studies conducted in the past two decades.

What Has Been Learned from the New Prospective Studies?

When the CLPS began, there was no formal definition for remission of personality disorders or BPD, and one had to be derived [5]. Remission was modeled after prospective studies for major depression. A modified version of the Diagnostic Interview for *DSM-IV* Personality Disorders (DIPD-IV) [33] was developed to track personality disorder criteria on a month-to-month basis [34], and remission was defined as meeting two criteria below threshold for 2 consecutive months. More recent research has seen the growth of several “BPD-change measures” for use in clinical trials research [35••].

In an early report from CLPS, Gunderson and colleagues [36] examined a subset of BPD patients who had remitted and identified four plausible determinants: Axis I disorder remissions, situational change, baseline misdiagnosis of BPD, and treatment. The majority of those who remitted appeared to benefit largely from an improvement in an Axis I condition or the resolution of highly conflicted relationships (for only 2 of the 18 patients studied was there a question of misdiagnosis). Although results are not generalizable because of the case study design, this report was remarkable for making the case for a possibility of a sustained remission.

Later, Gunderson and colleagues [37••] reported on the 10-year course of BPD from the CLPS. Two definitions of remission and relapse were studied, a 2-month remission for comparison to the major depressive disorder control group, and a 12-month definition for comparison to an “other” personality disorder group. BPD was characterized by high rates of remission, with only 9% retaining the diagnosis over 10 years. Relapse rates were also low, with only 11% of BPD patients relapsing for a sustained period of 12 months (21% for a period of 2 months). Relative to BPD diagnostic status, however, severe impairments in social functioning and overall functioning remained stable. Compared with an “other personality disorders” group and the no personality disorder major depressive disorder group, the rate of improvement of overall functioning in BPD was much lower.

Zanarini and colleagues [38] reported on the 10-year course of BPD. In MSAD, BPD was assessed over five 2-year intervals. For remission to occur required not meeting criteria on both the Revised Diagnostic Interview for Borderlines [39] and the DIPD-IV [33]. Over the 10-year period, 242 of 275 (88%) patients who began the study diagnosed with BPD met this stringent criterion for remission. Among the predictors for remission, stable premorbid work or school history along with favorable personality traits (eg, agreeableness) was suggested to bolster the likelihood of remission. This suggests that those characteristics that index and/or facilitate positive psychosocial behaviors are helpful for BPD patient improvement. In the MSAD, only approximately 20% of the patients experienced a relapse during the course of the study [38].

Relative to the BPD patients in CLPS whose psychosocial functioning remained relatively stable without improvement, patients in MSAD showed modest psychosocial improvement over the long term, though relatively less improvement was seen in vocational functioning. In any event, for both CLPS and MSAD, general impairments in functioning lagged behind diagnostic remission, and several areas of impairment were identified in CLPS. Regardless of these variations, the disjunction between functional impairment and diagnostic remission raises questions about the meaningfulness of the diagnostic thresholds [39]. For instance, it has been shown that those below the *DSM-IV-TR* diagnostic threshold still exhibit impairments on par with those with the diagnosis [40].

When personality disorders are measured dimensionally, greater stability is evident. Although the LSPD was a nonclinical sample, dimensional analyses of personality disorder criteria suggested greater levels of stability [6, 7]. In CLPS, using the Schedule for Adaptive and Non-Adaptive Behavior [41], greater levels of personality disorder stability, including BPD, were demonstrated [42]. Even assessment of the *DSM-IV* BPD measured dimensionally indicates greater stability than threshold criteria for a diagnosis [43]. Personality traits that undergird

personality disorders are also more stable than *DSM-IV* diagnoses, and there has been an effort to incorporate dimensional trait assessments into the *DSM-5* [44]. Interestingly, CLPS has reported that changes in lower-level personality traits precede changes in higher-level personality disorder symptoms, but not the reverse [45].

It has been argued that personality disorders, including BPD, are at the extreme end of dimensions of personality traits. Among others [46], Widiger and Trull [44] have argued that personality disorders are best captured in dimensional terms, and suggested that the diagnostic system be revised accordingly. Although the final version of the BPD diagnosis has not yet been determined for *DSM-5*, it does appear that both trait ratings and functioning ratings will be integrated into the BPD diagnosis. Divisions on the Axis I/II boundary are still being debated at this time, though there is evidence that suggests broader psychopathological dimensions cut across Axis I/II [47].

Among BPD criteria, affective instability may be seen to have “trait-like” qualities and may be a risk factor for the development or maintenance of BPD. Linehan [13] has described a core component of BPD as emotional hyperreactivity and a slow return to baseline. It has been shown over 10 years that the BPD affective instability appears to be the most stable BPD criterion [37••] and also that it may be a feature of BPD that is most clearly linked to neurobiology [48–50]. Others have argued that a psychobiological dimension of affective instability cuts across Axis I/II disorders, including BPD and affective disorders or post-traumatic stress disorder. Childhood, adolescent, and early-adulthood risk factors for BPD include a family history of substance abuse, sexual abuse, and physical abuse [51]. Such stressful and traumatic experiences can have an adverse effect on neural and hormonal systems involved in the regulation of emotion and mood [52, 53]. More severe psychopathology is present at baseline (ie, greater number of BPD criteria met, functional impairment, and a history of childhood trauma), which suggests an earlier disruption of these systems that may worsen the prognosis for BPD. Affective instability is likely a more proximal link to neural systems; therefore, targeting this feature of BPD for research offers the possibility of benefitting from the many recent advances made in integrative neuroscience.

Curiously, MSAD found that if BPD developed at a young age, remission was more likely than in those patients in older age groups indexed for the study [9]. It was speculated that those patients recruited into MSAD at an early age had not yet had a chance to experience subsequent stressful experiences that might be compounded, as compared with an older patient who had lived with BPD for a longer period of time. Similarly, CLPS found that when looking at the longitudinal course of BPD over a 6-year time period in individuals of varying ages, overall improvement in BPD

criteria was not dependent on age [54]. However, consistent with the MSAD finding, receiving the diagnosis at an older age was associated with a decline in functioning, whereas a diagnosis at a younger age suggested a better chance for improvements in functioning levels. This is also consistent with the notion that repeated, stressful experiences can tax the stress–response system and lead to more enduring impairment.

Suicide and Self-Injurious Behaviors

The most tragic of long-term outcomes associated with BPD is suicide. A lifetime rate of 10% for completed suicides is generally the accepted rule of thumb, and multiple suicide attempts are not uncommon for patients suffering from BPD [3]. The average number of self-harm behaviors, including life-threatening behaviors (ie, suicide attempts, cutting one’s self, overdosing), among those with BPD rises between the ages of 18 and 24 years and is sustained through an individual’s 50s. Similar patterns have been shown in non-BPD patients, though at lower rates. Such data raise questions about the “burnout” theory of personality disorders and suggest that suicide risk remains important to assess throughout the life course of those who have received a BPD diagnosis. More important, the risk of completed suicide for those suffering from BPD is real and something to be taken seriously by the clinician, and not—as sometimes was the case in the past—something to be viewed as “manipulative.”

One of the most consistent findings on suicide to emerge from the CLPS is that affective instability robustly predicts suicidal behaviors. In initial analyses, disinhibition, negative affectivity, and aspects of impulsivity significantly predicted suicide attempts. However, when multiple variables were taken into account, only negative affectivity and lack of premeditation were shown to be significant [55]. Thus, it seems advisable to pay attention to affective instability in suicide risk assessment relative to impulsivity among patients with BPD. The stability of affective instability (discussed previously) raises the specter of the continued lifetime risk of suicide.

Controlling for self-injury criterion, meeting certain BPD criteria (ie, affective instability, impulsivity, and identity disturbance), and the occurrence of sexual abuse in childhood put one at a higher risk of suicidal behavior. Among these, the foremost predictor of suicidality is affective instability [55]. Risk of suicide attempts appears to be amplified when conditions such as substance abuse and major depressive disorder are comorbid with BPD [56]. Suicide attempts were found to be more likely immediately following the worsening of major depressive disorder or substance abuse. It seems that negative life events, especially those relating to relationships

and criminality, play a significant role as precipitants of suicidal behavior [57].

Comorbidity: A Proxy for Stability

Perhaps more so than with any other *DSM*-based disorder, with BPD, comorbidity is the rule, not the exception [58–61]. Comorbidity is relevant to long-term outcome because greater ranges of psychopathology appear to be an indicator of BPD stability [62•]. As noted previously, onset of comorbid Axis I conditions may signal increased risk of suicide, and both Axis I and Axis II comorbidities may account for the intractable suffering associated with a more chronic course of BPD. Findings from the MSAD suggest that higher levels of neuroticism and avoidant, dependent, obsessive-compulsive, and self-defeating traits accompany a range of disorders and impeded recovery [9, 63•].

Similarly, in CLPS, it was found that the most stable levels of BPD pathology that persisted over 10 years tended to be those that were most highly correlated with other personality disorders indexed in the CLPS (schizotypal personality disorder, avoidant personality disorder, and obsessive-compulsive personality disorder) [62•]. These other pathological features that often accompany BPD offer a potential explanation for the treatment-resistant aspects of BPD. In other words, it may be this amalgam of pathology, the layers of pathological defenses and personality traits marked by the presence of these multiple co-occurring disorders, that interfere with the treatment process, and not necessarily *DSM-IV-TR* BPD per se.

For some time, the negative impact of personality disorders, including BPD, on the treatment outcome of Axis I disorders such as major depressive disorder have been accepted [64–67]. In CLPS, Gunderson and colleagues [68] studied the interface of BPD and major depressive disorder, and results suggest that remissions in BPD precede those in major depressive disorder, but not the reverse. This work underscores the need to develop treatments that target more stable psychopathological conditions in the service of addressing syndromes such as major depressive disorder that, though often chronic, tend toward periods or relapse and remission.

Comorbidities, be they Axis I, Axis II, or trait-level markers of psychopathology, present complications for conducting BPD treatment research. If treatment trials for BPD are highly selective to reduce comorbidity, resulting study groups are likely to be healthier than the typical patients presenting with BPD in the clinic. Thus, such research will be less generalizable to real world applications. Conversely, treatment trials that include patients with usual rates of comorbid conditions will increase the heterogeneity of the diagnostic sample. Although outcomes in the latter case may

be more generalizable, the possibility of clarifying treatment mechanisms will be less likely because of the amalgam of symptoms. For a more detailed discussion of comorbidity issues in sample selection for BPD research, see a report by Zanarini and colleagues [35••].

Conclusions and New Directions

Current research indicates that remission from BPD is a reasonable outcome and potentially more enduring in nature than previously assumed. Prospective data from large-scale clinical studies (eg, CLPS and MSAD) along with the development of empirically supported treatments have facilitated a shift in the view of BPD from a pejorative label for an intractant patient to a useful diagnosis that informs an effective treatment plan. Moreover, with this acceptance comes the utility of sharing the diagnosis with the patient and his or her significant others to help educate them about what to expect with the disorder, and also to facilitate a collaborative treatment process. This is very different from an earlier era of treatment during which the borderline diagnosis was often kept from the patient in the service of “protection.” It has also ushered in a new generation of clinicians and clinical researchers who do not shun working with the diagnosis. Support networks, including the NEA-BPD (National Educational Alliance for BPD) [69] and TARA (Treatment and Research Advancements National Association for Personality Disorders) [70], are among the valuable resources for those coping with the disorder. Together, this suggests increasing hope for those suffering from BPD.

The optimism with the changing view of BPD notwithstanding, some cautions may serve to direct future efforts to help ensure continued progress. When viewed dimensionally, long-term outcome studies suggest more stability for borderline pathology compared with categorical approaches to diagnosis and remission. Is there a “sub-diagnostic” syndrome that exists, conferring a continued risk for those who have technically remitted from the *DSM-IV-TR* diagnosis? Dimensional models also demonstrate that personality traits related to BPD are more stable. This raises questions about a set of core pathological traits that appear to be less amenable to change. In both cases, it seems there is a new direction for treatment development research. It may be that proactive intervention or maintenance treatments for individuals subclinical for BPD, or for those suffering related features of psychopathology outside the BPD diagnosis, could be studied for specific targeted problem areas (eg, for patients remitted from BPD, prevention efforts aimed at reducing the likelihood of an onset or relapse of an Axis I condition that could reduce suicide risk).

Chief among the more trait-like elements to the *DSM-IV-TR* definition of the disorder is affective instability criterion. It is plausible that even in the absence of any other BPD criteria, this feature of psychopathology could exert a cost for an individual's functioning. As noted previously, this confers risk for suicide and is thus a critical pathological element to be addressed even in the absence of a BPD diagnosis. Such emotional reactivity may be the product of temperamental systems that are constitutional in nature and may become more entrenched during psychological development by extreme or traumatic stress. It is also clear that emotional reactivity is not specific to BPD and is present in other conditions, such as post-traumatic stress disorder. Clarifying the pathophysiology of emotional reactivity is one pathway to identify treatment targets that might bolster treatments for BPD and other anxiety or distress disorders. For example, treatments designed to extinguish an overly generalized fear response could become an important treatment “add-on” component to psychotherapy.

The prospective, longitudinal studies have made clear a second area toward which future treatment research efforts might be expended. Although remission from the current BPD diagnosis is more the rule than the exception, substantial functional impairment remains. Though improvement in functioning is possible in some areas for some patients, overall findings across CLPS and MSAD provide convergent evidence of significant and persistent functional impairment in several areas. Continued troubles in occupational and psychosocial functioning suggest a couple of possibilities. First, it may be that the impairment conferred by BPD is related to those features of the diagnosis that are trait-like in nature. A second possibility is that the sustained impaired function stems from comorbid psychopathology associated with the more stable cases of BPD. Both of these possibilities raise questions about the structure of the current diagnosis. To at least some extent, these will be addressed by the planned inclusion of functional components to the revised *DSM-5* diagnosis [71]. A potential parallel exists in schizophrenia research in which after many years of focusing on positive symptoms, recent efforts include cognitive remediation for negative symptoms and approaches to enhance social functioning [72].

Focal treatment interventions have been tried, with promising success [73, 74]. Arguably the most significant advance for BPD treatment development has come from the development of DBT, and there is a lesson about progress for BPD outcomes. Linehan began her career as a suicide researcher, and the development of DBT was targeted to address suicidal behaviors and not specifically aimed at BPD. Given the frequency of suicidal behavior among those suffering from BPD, many of the patients whom she studied met criteria for the diagnosis. More recent leading research focuses on suicidal and self-injurious behaviors across

diagnoses [75] and is not limited to “focal” areas within BPD groups, but rather studying the construct across patient groups. Such approaches illustrate the benefit of targeting components of the diagnosis that are more broadly cut across other disorders, addressing to a certain extent the problems of comorbidity, but also by not unduly restricting variance by limiting it to a particular diagnosis. This approach may better isolate the full range of a dimensional mechanisms related to a particular dysfunction. Might the same approach be applied to what the prospective studies suggest is the most intractable aspect of BPD, namely functional impairment? In other words, consider the possibility of developing treatments and treatment trials based on functional impairments that are seen across disorders. For example, independent study variables might include specific areas of functional impairment in which levels of impairment in that area, not psychiatric diagnosis, would be used to define the treatment groups. The evidence is strong that functional impairments cut across related psychopathology (comorbidity) and below threshold of the BPD diagnosis (traits).

To be clear, this is not to challenge the clinical utility of the BPD diagnosis. Rather, the aim is merely to advance progress on specific problems that, while identifiable with BPD, clearly relate to other conditions as well, or may not apply to all those suffering from BPD. Such an approach could leverage specificity of behavioral, psychological, and biological mechanisms that might underlie various types of functional impairments. Furthermore, it would be consistent with other research diagnostic approaches aimed at clarifying psychopathological processes that are more proximal to their mechanisms [76]. Any resulting clarification of mechanisms or treatment technologies could be translated back to the treatment setting for those with the BPD diagnosis.

Acknowledgments The second and third authors made equal contributions to this paper and are alphabetically listed in authorship order. This work was supported by National Institutes of Health grant no. MH073708.

Disclosure No potential conflicts of interest relevant to this article were reported.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. McGlashan TH. The chestnut lodge follow-up study: Part III. Long-term outcome of borderline personalities. *Arch Gen Psychiatry*. 1986;43:20–30.
2. Akiskal HS, Chen SE, Davis GC, et al. Borderline: an adjective in search of a noun. *J Clin Psychiatry*. 1985;46:41–8.

3. Sanislow CA, McGlashan TH. Treatment outcome of personality disorders. *Can J Psychiatry*. 1998;43:237–50.
4. Skodol AE, Gunderson JG, Shea MT, et al. The Collaborative Longitudinal Personality Disorders Study (CLPS): overview and implications. *J Personal Disord*. 2005;19:487–504.
5. Gunderson JG, Shea MT, Skodol AE, et al. The Collaborative Longitudinal Personality Disorders Study: development, aims, design, and sample characteristics. *J Personal Disord*. 2000;14:300–15.
6. Lenzenweger MF. Stability and change in personality disorder features: the longitudinal study of personality disorders. *Arch Gen Psychiatry*. 1999;56:1009–15.
7. Lenzenweger MF. The Longitudinal Study of Personality Disorders: history, design, considerations, and initial findings. *J Personal Disord*. 2006;20:645–70.
8. Zanarini MC, Frankenburg FR, Hennen R, et al. The McLean Study of Adult Development (MSAD): overview and implications of the first six years of prospective follow-up. *J Personal Disord*. 2005;19:505–23.
9. Zanarini MC, Frankenburg FR, Hennen J, et al. Prediction of the 10-year course of borderline personality disorder. *Am J Psychiatry*. 2006;163:827–32.
10. Koenigsberg HW. Psychotherapy of patients with borderline personality disorder. *Curr Opin Psychiatry*. 1995;8:157–60.
11. Linehan MM, Armstrong HE, Suarez A, et al. Cognitive-behavioral treatment of chronically parasuicidal borderline patients. *Arch Gen Psychiatry*. 1991;48:1060–4.
12. Linehan MM, Heard HL, Armstrong HE. Naturalistic follow-up of a behavioral treatment for chronically parasuicidal borderline patients. *Arch Gen Psychiatry*. 1993;50:971–4.
13. Linehan M. Cognitive-behavioral treatment of borderline personality disorder. New York: Guilford; 1993.
14. Fonagy P, Luyten P. A developmental, mentalization-based approach to the understanding and treatment of borderline personality disorder. *Dev Psychopathol*. 2009;21:1355–81.
15. Bateman A, Fonagy P. Randomized controlled trial of outpatient mentalization-based treatment versus structured clinical management for borderline personality disorder. *Am J Psychiatry*. 2009;166:1355–64.
16. Clarkin JF, Levy KN, Lenzenweger MF, Kernberg OF. Evaluating three treatments for borderline personality disorder: a multiwave study. *Am J Psychiatry*. 2007;164:922–8.
17. Black DW, Blum N, Pfohl B, St. John D. The STEPPS group treatment program for outpatients with borderline personality disorder. *J Contemp Psychother*. 2004;34:193–210.
18. Blum N, St. John D, Pfohl B, et al. Systems Training for Emotional Predictability and Problem Solving (STEPPS) for outpatients with borderline personality disorder: a randomized controlled trial and 1-year follow-up. *Am J Psychiatry*. 2008;165:468–78.
19. Farrell JM, Shaw IA, Webber MA. A schema-focused approach to group psychotherapy for outpatients with borderline personality disorder: a randomized controlled trial. *J Behav Ther Exp Psychiatry*. 2009;40:317–28.
20. Zanarini MC, Frankenburg FR. A preliminary, randomized trial of psychoeducation for women with borderline personality disorder. *J Personal Disord*. 2008;22:284–90.
21. Stern A. Psychoanalytic investigation of and therapy in the border line group of neuroses. *Psychoanal Q*. 1938;7:467–89.
22. Kernberg O. Borderline personality organization. *J Amer Psychoanal Assn*. 1967;5:641–85.
23. Feighner JP, Robins E, Guze SB, et al. Diagnostic criteria for use in psychiatric research. *Arch Gen Psychiatry*. 1972;26:57–63.
24. Spitzer RL, Robins E. Research diagnostic criteria: rationale and reliability. *Arch Gen Psychiatry*. 1978;35:773–82.
25. Gunderson JG, Singer MT. Defining borderline patients: an overview. *Am J Psychiatry*. 1975;132:1–10.
26. Gunderson JG, Carpenter WT, Strauss JS. Borderline and schizophrenic patients: a comparative study. *Am J Psychiatry*. 1975;132:1257–64.
27. Spitzer RL, Endicott J, Gibbon M. Crossing the border into borderline personality and borderline schizophrenia: the development of criteria. *Arch Gen Psychiatry*. 1979;36:17–24.
28. Skodol AE, Buckley P, Charles E. Is there a characteristic pattern to the treatment history of clinic outpatients with borderline personality? *J Nerv Ment Dis*. 1983;171:405–10.
29. Stone MH, Hurt SW, Stone DK. The PI 500: long-term follow-up of borderline inpatients meeting DSM-III criteria: I. Global outcome. *J Personal Disord*. 1987;1:291–8.
30. Werble B. Second follow-up study of borderline patients. *Arch Gen Psychiatry*. 1970;23:3–7.
31. Bardenstein KK, McGlashan TH. The natural history of a residentially treated borderline sample: gender differences. *J Personal Disord*. 1988;2:69–83.
32. Sabo AN, Gunderson JG, Najavits LM. Changes in self-destructiveness of borderline patients in psychotherapy: a prospective follow-up. *J Nerv Ment Dis*. 1995;183:370–3.
33. Zanarini MC, Skodol AE, Bender DS, et al. The Collaborative Longitudinal Personality Disorders study: reliability of Axis I and II diagnoses. *J Personal Disord*. 2000;14:291–9.
34. Shea MT, Stout R, Gunderson JG, et al. Short-term diagnostic stability of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *Am J Psychiatry*. 2002;159:2036–41.
35. •• Zanarini MC, Stanley B, Black DW, et al. Methodological considerations for treatment trials for persons with borderline personality disorder. *Ann Clin Psychiatry*. 2010;22:75–83. *This article summarizes treatment development and outcome studies for BPD and reviews diagnostic and change measures. Several issues in conducting treatment outcome research with BPD are identified.*
36. Gunderson JG, Bender D, Sanislow C, et al. Plausibility and possible determinants of sudden “remissions” in borderline patients. *Psychiatry*. 2003;66:111–9.
37. •• Gunderson JG, Stout RL, McGlashan TH, et al. Ten-year course of borderline personality disorder: psychopathology and function from the Collaborative Longitudinal Personality Disorders Study. *Arch Gen Psychiatry*. 2011;68:827–37. *This paper summarizes 10-year findings for the CLPS study. Both diagnostic remission and functional stability are examined.*
38. Zanarini MC, Frankenburg FR, Chauncey DL, et al. The diagnostic interview for borderlines revised: discriminating BPD from other Axis II disorders. *J Personal Disord*. 1989;3:10–8.
39. 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. *Psychol Med*. 2005;35:443–51.
40. Clifton A, Pilkonis PA. Evidence for a single latent class of Diagnostic and Statistical Manual of Mental Disorders borderline personality pathology. *Comp Psych*. 2007;48:70–8.
41. Clark LA. Manual for the Schedule for Nonadaptive and Adaptive Personality (SNAP). Minneapolis: University of Minnesota Press; 1993.
42. Morey LC, Warner MB, Shea MT, et al. The representation of four personality disorders by the schedule for nonadaptive and adaptive personality dimensional model of personality. *Psychol Assess*. 2003;15:326–32.
43. Skodol AE, Oldham JM, Bender DS, et al. Dimensional representations of DSM-IV personality disorders: relationships to functional impairment. *Am J Psychiatry*. 2005;162:1919–25.
44. Widiger TA, Trull TJ. Plate tectonics in the classification of personality disorder: shifting to a dimensional model. *Am Psychol*. 2007;62:71–83.

45. Warner MB, Morey LC, Finch JF, et al. The longitudinal relationship of personality traits and disorders. *J Abnorm Psychol.* 2004;113:217–27.
46. Livesley WJ, Jang KL, Vernon PA. Phenotypic and genetic structure of traits delineating personality disorder. *Arch Gen Psychiatry.* 1998;55:941–8.
47. Kotov R, Ruggero CJ, Krueger RF, et al. New dimensions in the quantitative classification of mental illness. *Arch Gen Psychiatry.* 2011;68:1003–11.
48. Donegan NH, Sanislow CA, Blumberg HP, et al. Amygdala hyperreactivity in borderline personality disorder: implications for emotional dysregulation. *Biol Psychiatry.* 2003;54:1284–93.
49. Koenigsberg HW. Affective instability: toward an integration of neuroscience and psychological perspectives. *J Pers Dis.* 2010;24:60–82.
50. Koenigsberg HW, Harvey PD, Mitropoulou V, et al. Characterizing affective instability in borderline personality disorder. *Am J Psychiatry.* 2002;159:784–8.
51. Zlotnick C, Johnson DM, Yen S, et al. Clinical features and impairment in women with borderline personality disorder (BPD) with posttraumatic stress disorder (PTSD), BPD without PTSD, and other personality disorders with PTSD. *J Nerv Ment Dis.* 2003;191:706–14.
52. Pine DS, Helfinstein SM, Bar-Haim Y. Challenges in developing novel treatments of childhood disorders: lessons from research on anxiety. *Neuropsychopharmacology Rev.* 2008;113:1–16.
53. Bales KL, Carter CS. Neuroendocrine mechanisms of social bonds and child–parent attachment, from the child’s perspective. In: de Hann M, Gunnar MR, editors. *Handbook of developmental social neuroscience.* New York, NY: Guilford Press; 2009. p. 246–264.
54. Shea MT, Edelen MO, Pinto A, et al. Improvement in borderline personality disorder in relationship to age. *Acta Psychiatr Scand.* 2009;119:143–8.
55. Yen S, Shea MT, Sanislow CA, et al. Borderline personality disorder criteria associated with prospectively observed suicidal behavior. *Am J Psychiatry.* 2004;161:1296–8.
56. Yen S, Pagano ME, Shea MT, et al. Recent life events preceding suicide attempts in a personality disorder sample: findings from the Collaborative Longitudinal Personality Disorders Study. *J Consult Clin Psychol.* 2005;73:99–105.
57. Yen S, Shea MT, Pagano M, et al. Axis I and Axis II disorders as predictors of prospective suicide attempts: findings from the collaborative longitudinal personality disorders study. *J Abnorm Psychol.* 2003;112:375–81.
58. McGlashan TH, Grilo CM, Skodol AE, et al. The Collaborative Longitudinal Personality Disorders Study: baseline Axis I/II and II/II diagnostic co-occurrence. *Acta Psychiatr Scand.* 2000;102:256–64.
59. Zanarini MC, Frankenburg FR, Hennen J, et al. Axis I comorbidity in patients with borderline personality disorder: 6-year follow-up and prediction of time to remission. *Am J Psychiatry.* 2004;161:2108–14.
60. Zanarini MC, Frankenburg FR, Vujanovic AA, et al. Axis II comorbidity of borderline personality disorder: description of 6-year course and prediction to time-to-remission. *Acta Psychiatr Scand.* 2004;110:416–20.
61. Grilo CM, Sanislow CA, McGlashan TH. Co-occurrence of DSM-IV personality disorders with borderline personality disorder. *J Nerv Ment Dis.* 2002;190:552–4.
62. • Sanislow CA, Little TD, Ansell EB, et al. Ten-year stability and latent structure of the DSM-IV schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *J Abnorm Psychol.* 2009;118:507–19. *This paper examines the 10-year stability of the four personality disorders studied in the CLPS. Questions are raised about the how the structure of borderline, schizotypal, avoidant, and obsessive-compulsive become less distinct, or more correlated, over time.*
63. • Hopwood CJ, Donnellan MB, Zanarini MC. Temperamental and acute symptoms of borderline personality disorder: associations with normal personality traits and dynamic relations over time. *Psychol Med.* 2010;40:1871–8. *This paper examines the relations among personality traits and BPD using data from the MSAD. Several personality traits are identified that suggest a vulnerability for personality disorders.*
64. Shea MT, Glass DR, Pilkonis PA, et al. Frequency and implications of personality disorders in sample of depressed outpatients. *J Personal Disord.* 1987;1:27–42.
65. Shea MT, Elkin I, Imber SD, et al. Course of depressive symptoms over follow-up: findings from the National Institute of Mental Health Treatment of Depression Collaborative Research Program. *Arch Gen Psychiatry.* 1992;49:782–7.
66. Ilardi SS, Craighead WE. The relationship between personality pathology and dysfunctional cognitions in previously depressed adults. *J Abnorm Psychol.* 1999;108:51–7.
67. Grilo CM, Sanislow CA, Shea ME, et al. Two-year prospective naturalistic study of remission from major depressive disorder as a function of personality disorder comorbidity. *J Consult Clin Psychol.* 2005;73:78–85.
68. Gunderson JG, Stout RL, Sanislow CA, et al. New episodes and new onsets of major depression in borderline and other personality disorders. *J Affect Disord.* 2008;111:40–5.
69. <http://www.borderlinepersonalitydisorder.com/> Accessed on 11/4/11.
70. <http://www.tara4bpd.org/dyn/index.php>. Accessed on 11/4/11.
71. <http://dsm5.org/proposedrevision/Pages/PersonalityDisorders.aspx>. Accessed on 11/4/11.
72. Kurtz MM, Mueser KT. A meta-analysis of controlled research on social skills training for schizophrenia. *J Consult Clin Psychol.* 2008;76:491–504.
73. Gratz KL, Gunderson JG. Preliminary data on an acceptance-based emotion regulation group therapy intervention for deliberate self-harm among women with borderline personality disorder. *Behav Ther.* 2006;37:25–35.
74. Weinberg I, Gunderson JG, Hennen J, et al. Manual assisted cognitive treatment of deliberate self-harm in borderline personality disorder patients. *J Pers Disord.* 2006;20:482–92.
75. Nock MK, Prinstein M. A functional approach to the assessment of self-mutilative behavior. *J Consult Clin Psychol.* 2004;72:885–90.
76. • Sanislow CA, Pine DS, Quinn KJ, et al. Developing constructs for psychopathology research: Research Domain Criteria. *J Abnorm Psychol.* 2010;119:631–9. *This paper describes the Research Domain Criteria project and suggests a new approach for research classification that is based more on mechanisms of psychopathology. The purpose of the Research Domain Criteria project is to provide a classification system to define study groups for psychopathology research.*