

Formal Rebuttal

The case for retaining borderline personality disorder as a psychiatric diagnosis

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Introduction

The paper by Tyrer (2009) suggests that borderline personality disorder (BPD) is neither borderline nor a personality disorder. We agree—at least partially. The term ‘borderline’ has historical significance, but does not describe any meaningful border, and BPD differs from most personality disorders in being associated with a wide range of disabling symptoms (Paris, 2007, 2008a). However, it does not follow that BPD should be reclassified as a mood disorder or that it should be eliminated from the diagnostic system.

Why BPD is not a mood disorder

Affective instability (AI), or emotional dysregulation (or, to use the term suggested by Tyrer, ‘fluxithymia’), describes short-term mood swings in which affect is intense and returns only slowly to normal levels (Harvey, Greenberg, & Serper, 1989; Herpertz et al., 1997). The idea that AI is an essential feature of BPD has been suggested before (Akiskal, Chen, & Davis, 1985; Linehan, 1993; Livesley, Jang, & Vernon, 1998) and has been supported by research (Henry et al., 2001; Koenigsberg

et al., 2002). This clinical phenomenon has stimulated a large body of research (Putnam & Silk, 2005). However, it does not follow that AI explains all the symptoms in BPD patients or that it should be placed in the same category as classical mania and depression.

BPD is a complex and multidimensional syndrome whose symptoms are not confined to or accounted for by changes in mood (Paris, 2007). Several lines of evidence contradict this hypothesis that the symptoms of BPD can be accounted for by changes in mood. First, AI differs from classical depression in being a short-lived and exaggerated response to interpersonal stressors (Gunderson & Phillips, 1991; Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007) rather than being characterized by sustained lowered mood. Moreover, AI responds inconsistently, if at all, to antidepressant treatment (Paris, 2008a). For example, as we know from the Collaborative Longitudinal Personality Disorders Study, in only 20% of the BPD cases with comorbid major depressive disorder (MDD) did the depression remit after treatment with antidepressants. Even when the depression did remit, its remission had little effect on the course of BPD. In contrast, when the BPD remitted, this remission was followed by improve-

ments in the severity of the MDD (Gunderson et al., 2004).

Second, AI does not make BPD a bipolar spectrum disorder. Although there is some comorbidity with bipolar-II, most cases are distinct, and these disorders do not overlap when patients are followed over time (Gunderson et al., 2006; Paris, Gunderson, & Weinberg, 2007). BPD patients also respond inconsistently to treatment with mood stabilizers (Paris, 2008a), and most of the impact of mood stabilizers is limited to symptoms of anger/impulsivity (Mercer, Douglass, & Links, 2009).

Third, impulsivity is just as central to BPD as AI. Impulsivity is a core feature without which the diagnosis would rarely even be considered (Links, Heselgrave, & van Reekum, 1999). It is difficult to see how mood instability by itself can account for recurrent suicide attempts, self-harm, substance abuse, eating disorders, sexual promiscuity or antisocial behaviours. Impulsive behaviours often function to regulate dysphoria.

Patients with BPD score high on all dimensions of impulsivity (Links et al., 1999; Paris et al., 2004). Impulsive spectrum disorders (such as antisocial personality and substance abuse) are the most frequent disorders in the first-degree relatives of BPD probands, and are more common than mood disorders (Silverman et al., 1991; White, Gunderson, Zanarini, & Hudson, 2003). Moreover, high levels of impulsivity are the most consistent predictor of clinical outcome in BPD (Links et al., 1999). Finally, neurobiological studies show that impulsivity in BPD is the only trait that has a robust association with biological markers. This has been shown by abnormalities in neurotransmitter activity in challenge tests (Coccaro et al., 1989; Paris et al., 2004), and in neuroimaging (Leyton et al., 2001; Siever et al., 1999).

Fourth, problems in interpersonal relationships are equally characteristic of BPD. One sees a pattern of interpersonal hypersensitivity, often associated with disorganized or ambivalent attachment styles, intolerance of aloneness, abandonment fears and a self-image of being evil (Gunderson,

1984; Gunderson & Lyons-Ruth, 2008; Zanarini & Frankenburg, 1994). Again, these complex phenomena are not easily accounted for by mood alone, and in fact are thought of as very atypical or exceptional if they were found in mood disorders. The depression in BPD is frequently marked by loneliness and emptiness (Westen et al., 1992), and while major depression per se is usually not responsive to environmental factors, the depression in BPD is often acutely responsive to environmental (i.e. interpersonal) factors.

Fifth, BPD patients often have prominent cognitive symptoms (Zanarini, Gunderson, & Frankenburg, 1990). Chronic depersonalization, paranoid trends, and transient delusions and hallucinations are all commonly present, and these symptoms are not often seen in patients with non-psychotic mood disorders.

These observations have important clinical implications. Defining BPD as a mood disorder might suggest that these are patients who should be given pharmacological treatment for AI. However, there is no evidence from randomized clinical trials that these agents are particularly effective in BPD (Paris, 2008a). While antidepressants and mood stabilizing drugs show some therapeutic effects (primarily on impulsivity), they do not stabilize the unstable mood of BPD, and never produce remissions.

The essence of the BPD diagnosis is that it describes multiple domains of pathology, rooted in multiple diatheses and endophenotypes. This conclusion has been supported by cluster analyses of BPD symptoms (Hurt, Clarkin, Munroe-Blum, & Marziali, 1992; Sanislow et al., 2002), which have found that emotional dysregulation, impulsivity and disturbed relationships form partially separate domains. Each of these clusters could be associated with specific neurobiological mechanisms (Paris, 2008b; Siever & Davis, 1991; Silk, 2000; Skodol et al., 2002). While it is tempting to simplify a complex problem, reducing BPD to mood instability does not do it justice.

To dismiss the concept of BPD because it may describe multiple domains of pathology, rooted

in multiple diatheses and endophenotypes, seems quite selective because there are many psychiatric disorders that might involve multiple domains of pathology and rooted in multiple endophenotypes and diatheses. Post-traumatic stress disorder as well as schizophrenia could serve as examples of this 'multiplicity', but no one is suggesting that these diagnoses be eliminated. Further, we appreciate that schizophrenia is far from a disorder wherein the personality is split, the original concept of schizophrenia and upon which the name schizophrenia was devised.

The importance of the BPD diagnosis for treatment

A main purpose of any psychiatric classification is to provide a guide to treatment. That is the most important reason for retaining the BPD diagnosis.

In recent years, a large and important recent literature has emerged, demonstrating the efficacy of specific psychotherapies designed for the BPD patient population (e.g. Bateman & Fonagy, 2006; Clarkin, Levy, Lenzenweger, & Kernberg, 2007; Davidson et al., 2006; Giesen-Bloo et al., 2006; Linehan, 1993), though we cannot suggest that these treatments would be ineffective in other disorders. What is most striking about these findings is that standard psychological treatments (usually called 'treatment as usual') are not very effective in BPD, while therapies designed for this diagnostic category are much superior. In addition, recent empirical work has explicated the mechanisms of action of the therapies such as transference-focused psychotherapy and does not support the resolution of unstable mood as central to the changes related to the therapy (Levy et al., 2006). Given these advances, making a diagnosis of BPD makes a difference in practice because clinicians need to conduct treatment differently.

For these reasons, we are concerned that removing BPD from the classification will rebound negatively on patients. This population will not go away; however, the diagnosis is redefined. Because

BPD patients are clinically problematic, they have often been inadequately managed: in earlier times, by offering classical forms of psychotherapy that are not appropriate for them, and more recently, by offering polypharmacy regimes that are not appropriate (Zanarini, Frankenburg, Khera, & Bleichmar, 2001).

Future directions

While we recommend the retention of BPD in the Diagnostic and Statistical Manual of Mental Disorders (DSM)-V, we do not favour the retention of the current criteria, which badly need revision. Diagnostic algorithms need to be more specific as opposed to the current 'Chinese menu approach' in which any five out of nine unweighted criteria is considered sufficient. The introduction of symptoms, such as a high degree of subjective emotional pain, which have been found to be both common among and specific to BPD, should be considered for inclusion in DSM-V (Zanarini & Frankenburg, 1994; Zittel Conklin & Westen, 2005). In addition, the introduction of some kind of dimensional scoring might help (Krueger, Skodol, Livesley, Shrout, & Huang, 2007), although empirical evidence is needed to prove that such a procedure would actually increase validity. And diagnostic reform needs to be applied to all categories of disorders in DSM-V, not just BPD.

Finally, we agree that 'borderline personality' is a bad name. Much bias and stigma are attached to the name but there is no evidence that changing the name will prevent the stigma from migrating to the new diagnosis. Schizophrenia or cancer were diagnoses that, a few generations ago, were an anathema to utter. But the names were not eliminated; rather, the public was educated as to what these diseases or disorders really were. And now we have schizophrenia centres and cancer centres of excellence. So before we replace one diagnostic name with another, we need further understanding of the disorder. We are far from this goal for almost all categories in the DSM system.

To rename BPD as 'emotional regulation disorder' or 'fluxithymia' assumes that we understand psychopathology, when in fact we do not, and also assumes that the new name will provide better care for these patients. The most conservative position when aetiology and pathogenesis are unknown is to continue conducting research until we have solid findings on which to base a new classification.

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