Borderline personality disorder (BPD) is a serious chronic condition which may lead to a poor quality of life and carries a considerable risk of comorbidity and suicide. The syndrome has been acknowledged and investigated in psychiatry for at least seventy years. The DSM-IV gives a panel of nine symptoms and manifestations for its diagnosis, but not taking into account neurobiological and objective issues.

The issue of whether BPD should be considered as a discrete condition, as the edge of a dimensional state ranging from ‘almost normal’ personalities to clearly pathological ones or as a hybrid of traits and symptomatic behaviors (McGlashan, 2005) seems to be important to correctly diagnose our patients and is a corner stone in the Research Agenda for DSM-V. The pathophysiology of BPD is very important because this is the progress which will probably lead to the development of effective treatments. Several investigators have shown some laboratory and objective clinical findings in BPD as normality in endocrinological dynamic tests (De La Fuente et al., 2002), alterations in sleep-EEG parameters (De la Fuente et al., 2004), disturbed regional brain glucose metabolism (De la Fuente et al., 1997), abnormal scalp EEGs (De la Fuente et al., 1998) and an extremely high prevalence of neurologic soft signs (De la Fuente et al., 2006). These facts probably reflect common underlying non focal nervous system failure and hypothalamic-pituitary-adrenal axis normality in BPD.

However, these findings are not pathognomonic of the condition and BPD is diagnosed according to DSM-IV when a patient shows at least five symptoms out of a list of nine. First & Zimmerman (2006) propose that biological variables can aid to identify the patients as compared to the criteria used now to define them. For this the objective signs and facts found in BPD could be included as diagnostic criteria in the DSM-V. Scalp wake EEG, the clinical exploration for neurologic soft signs, sleep EEG recordings and the two endocrine tests, dexamethasone suppression and TRH stimulation, which are cheap, not invasive and have shown to be linked to the diagnosis of BPD could be incorporated in a diagnostic weighting construct with these variables as additional score items. This would have at least three advantages over the present criteria: firstly, a part of the diagnosis of BPD would become objective, secondly, having these parameters would attach physiopathological information to the diagnosis and thirdly, the laboratory information and objective
neurologic signs would aid to better characterize BPD patients for further research and effective treatments.

Although BPD and major depression (MD) can coexist, the depressive state most frequently associated with BPD seems to be distinct from nonborderline depression in terms of quality and duration of symptoms. Emptiness, loneliness, labile affect, behavioral dysregulation, anger and tension, self-condemnation, abandonment fears, self-destructiveness and hopelessness appear to be specific clusters of borderline affective symptoms (Rogers et al., 1995). These symptoms typically last between two and four days and reappear several times a month. Pharmacological studies have shown differences in treatment response between BPD and MD, conventional antidepressants and ECT have exhibited poor response (Feske et al., 2004) and even worsening in BPD.

Diverse laboratory data registered in the last few years have guided to propose that the depressive symptoms in BPD have a distinct biological substrate than those in the nonborderline depressive illness (De la Fuente et al., 2002; 2004). Recent receptor studies have found the hippocampal 5HT(2A) receptor binding, which is decreased in MD (Mintun et al., 2004), is increased in BPD with binding values being related to comorbid MD but not to depressed mood (Soloff et al., 2007).

Recurrent brief depression (RBD) has been proposed to overlap with BPD but RBD has been found to share perturbed biological substrates (endocrine and sleep-EEG) with MD (Staner et al., 1992) whereas others (De la Fuente et al., 2002; 2004) have not found this pathological shared substrate in BPD. From a biological point of view, this seems to distance BPD from RBD.

We suggest that a new subcategory be included in the DSM-V criteria sets for further study, which could be named ‘BPD-related affective disorder’ characterized by depressive symptoms of a distinct quality than in MD, a brief duration of the episodes, less than two weeks, and by a biological substrate different from those found in MD. BPD patients without concomitant MD have not shown abnormality in endocrine tests (De la Fuente et al., 2002). In the subcategory proposed, the hypothalamic pituitary axis would probably not be altered, or at least not in the same way than in MD. The same would be true for the sleep-EEG.

The existence of this new subcategory in a structured framework as the DSM-V would aid to replicate, or not, previous results with larger samples and it would make it possible to perform homogeneous neurotransmission, molecular, genetic, nosology and epidemiological research. This would also allow testing new molecules in uniform BPD-affective and non-affective samples and in fine to reduce the enormous burden that BPD produces, not only in terms of suicide and impulsivity-related mortality but also in terms of quality of life.

REFERENCES


---

José Manuel De la Fuente M.D., Ph.D., Hôpital Psychiatrique de Lannemezan. Route de Toulouse, F-65300 Lannemezan. France and Faculty of Medicine, Oviedo University, Julian Clavería 6, 33006 Oviedo, Spain.

Julio Bobes M.D., Ph.D., Faculty of Medicine, Oviedo University, Julian Clavería 6, 33006 Oviedo, Spain.

Correspondence to: jdlf@ch-lannemezan.fr