EPIDEMIOLOGY, BIOLOGY, AND ETIOLOGY

To date, few population-based studies of personality disorders have been carried out. Torgersen (28) tabulated the results of eight epidemiological studies, including the large Norwegian study done by his own group; the mean prevalence of BPD across all studies was 1.2%. In Torgersen et al.’s study of 2,053 individuals in Norway, BPD was found to be equally prevalent among males and females (29). Although it is widely perceived that treatment populations of patients with BPD consist predominantly of females, published studies are in fact contradictory on this point (30). Prevalence of BPD in treatment populations remains much higher than in the general population, and a recent study of a primary care population reported BPD to be fourfold higher than in the general population (31).

New studies are examining the multiple etiologies of BPD, based on the clear relevance of genetic, neurobiological, and psychosocial factors (32, 33). Although the magnitude of the heritability of BPD is thought to be significant (34), genetic risk factors specific to BPD have not been identified. One promising approach may be the study of underlying traits, such as novelty seeking (35) or impulsivity (36). Among the environmental contributions to the etiology of BPD, the role of childhood abuse remains prominent (37), particularly severe abuse (38) and sustained abuse (39).

Deficits in cognitive functioning, such as decision making (40), conflict resolution (41), and "effortful control" (41, 42), are being reported. These deficits are thought to be mediated in the frontal lobe, although not all studies have found frontal lobe impairment in cognitive functioning (43). A number of studies have suggested reduced central nervous system serotonin levels in borderline patients and other patients who demonstrate impulsive-aggressive behavior, but such behavioral disinhibition may reflect dysfunction in multiple monoaminergic systems and may not be specific to BPD (44).

Brain imaging studies of patients with BPD suggest irregularities in both structure and function (44-46). Limbic studies have found reduced volume in the amygdala and hippocampus (47, 48), altered amygdala activation (49, 50), and hippocampal hypometabolism (51). Frontal lobe studies suggest dysfunction of the dorsolateral and medial prefrontal cortex in connection with memories of abandonment (52), of the medial orbital frontal cortex in connection with diminished regulation of impulsive behavior (53), and of the prefrontal cortex under resting conditions (51). In addition, decreased binding of a serotonin precursor has been reported in the medial prefrontal cortex and anterior cingulate cortex in patients with BPD (54). Taken together, these studies suggest abnormalities in prefrontal, corticostriatal, and limbic networks perhaps related to lowered serotonin neurotransmission and behavioral disinhibition (55). Although findings from brain imaging studies are "not ready for prime time" to guide treatment planning for patients with BPD, these techniques may well have relevance in the future for individualized prediction of treatment outcome (46).

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