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Borderline Personality Disorder and Psychosis: A Review

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Abstract Early views of borderline personality disorder (BPD) were based on the idea that patients with this pathology were "on the border" of psychosis. However, more recent studies have not supported this view, although they have found evidence of a malevolent interpersonal evaluation and a significant proportion of BPD patients showing psychotic symptoms. For example, in one study, 24% of BPD patients reported severe psychotic symptoms and about 75% had dissociative experiences and paranoid ideation. Thus, we start with an overview regarding the prevalence of psychotic symptoms in BPD patients. Furthermore, we report findings of studies investigating the role of comorbidity (eg, post-traumatic stress disorder) in the severity and frequency of psychotic symptoms in BPD patients. We then present results of genetic and neurobiological studies comparing BPD patients with patients with schizophrenia or nonschizophrenic psychotic disorders. In conclusion, this review reveals that psychotic symptoms in BPD patients may not predict the development of a psychotic disorder but are often permanent and severe and need careful consideration by clinicians. Therefore, adequate diagnosis and treatment of psychotic symptoms in BPD patients is emphasized.

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Cognitive Characteristics of Borderline Personality Disorder and Psychotic Disorders: Are Borderline Personality Disorder Patients on the Border of Psychosis?

Early views of borderline personality disorder (BPD) were based on the idea that patients with this pathology were "on the border" of psychosis. Thus, the term *borderline* was introduced to emphasize this idea [1]. Psychoanalytic thinkers assumed that BPD patients experienced shifts toward "primary process" thinking in the absence of structure or in the presence of strong affects [2]. In BPD and schizophrenic disorder, the "secondary process," consisting of semantic and logical operations with an organizing and synthesizing function, was thought to be impaired, giving way to "primary process" thinking, resulting from a developmentally early mode of mental organization based on affects, imagery, and fantasy.

Several early empiric studies seemed to support this theory, showing normal scores on the "structured" Wechsler Adult Intelligence Scale (WAIS) but psychotic thought processes on the "unstructured" Rorschach inkblot projective test in BPD patients [3]. However, several authors have pointed out serious methodologic problems with these studies, including diagnosis and sample heterogeneity, inadequate sample descriptions, power problems, base rate issues, systematic differences in selection criteria among studies, lack of multimethod test validation, different outcome measures, and omission of quantitative test results [4]. Furthermore, it is unclear whether the results of these early studies, which were primarily carried out before the *DSM* formulations of BPD, apply to borderline pathology as we know it today.

More recent studies do not unequivocally support the "normal WAIS/disturbed Rorschach" hypothesis for BPD. Following O'Leary [4], these studies can be divided by research on 1) neuropsychological characteristics (eg, memory and visual perception, WAIS-Revised) and 2) emotional processing characteristics (as tapped with the Rorschach test, Thematic Apperception and Picture Arrangement Test, and several other tests of affective perceptions).

Regarding neuropsychological characteristics, several studies of BPD populations do not support the "normal WAIS" hypothesis [5, 6•]. However, it remains unclear whether the neuropsychological deficits among BPD patients are as deviant as those of psychotic patients. Thus far, only one study has investigated cognitive inhibition deficits in BPD patients and patients with schizophrenia [6•]. This study found that inhibition deficits, as measured by the antisaccadic eye movement task, characterized patients with schizophrenia and BPD, but only BPD patients with psychotic-like symptoms.

With respect to emotional processing characteristics, many studies have investigated spontaneous interpretations of evocative pictures (eg, Thematic Apperception Test) or inkblots (eg, Rorschach). In line with the "disturbed Rorschach" hypothesis, most of these studies showed less integrated, less differentiated, and less accurate interpretations in BPD patients compared with "neurotic" (ie, nonborderline and nonpsychotic) patients [7]. Compared with psychotic patients, BPD patients generated interpretations that were equally or more cognitively advanced [8]. However, some BPD patients even seemed capable of making interpretations that were cognitive developmentally more advanced than those of neurotic patients [9]. Moreover, recent experimental studies show nonprimitive interpersonal attributions [10] and enhanced emotional recognition abilities in BPD patients [11]. These outcomes do not support the psychoanalytical idea of a cognitive-emotional developmental continuum from psychotic to neurotic personality organization, with borderline personality organization placed in between.

Instead of primitive cognitions, several studies demonstrate negativistic or malevolent evaluations and lack of positive evaluations in BPD patients [12•]. In conclusion, we therefore suggest that it is not a cognitive developmental deficit but rather a tendency to construe interpersonal relations as malevolent that characterizes BPD, and this may be shared with certain psychotic disorders.

Prevalence of Psychotic Symptoms in Borderline Personality Disorder

Recent studies have shown that psychotic symptoms occur often in patients with BPD [13••, 14]. For example, in one study, 24% of BPD patients showed severe psychotic symptoms [15] and about 75% reported dissociative experiences and paranoid ideation [14]. Coid et al. [16] recently found a rate of 8.8% of patients with positive psychotic symptoms in a sample of 113 BPD patients. Several studies also reported hallucinatory experiences in BPD patients [13••, 17], and one study found that perceptual aberration and physical anhedonia predicted BPD features in young adults [18]. These findings support the incorporation of transient stress-related paranoid ideation or severe dissociation as the ninth criterion in the *DSM-IV* BPD classification [19]. However, the *DSM-IV* notes these symptoms as occurring only for brief periods in situations of stress [19], following the work of Zanarini and colleagues [14], who emphasized that the psychotic symptoms seen in BPD patients are often transient (quasi–or pseudohallucinations). Recent studies have shown, however, that psychotic symptoms such as auditory hallucinosis and delusions often persist over time and occur in a significant proportion of patients with BPD [13••, 17]. For example, Yee et al. [17] reported that 29% of their sample of 171 BPD patients had auditory hallucinations and that those hallucinations were for the most part distressing and present since early childhood. In conclusion, these authors emphasize that the sharp distinction between the concepts of quasi–(or pseudo-) psychotic experiences and "true" psychotic experiences may be clinically problematic. In the following section, we present a case illustration supporting this assumption of a female BPD patient who developed severe psychotic symptoms during inpatient therapy.

Case Illustration

J. was a 21-year-old single woman with 12 years of education and part-time employment in a shopping center. She showed severe dissociative experiences (eg, feelings of being disconnected, derealization, depersonalization, and dissociative seizures). At the beginning of therapy, she reported suicidal ideation, tenseness, self-injuring behavior, and deep feelings of emptiness and hostility. After watching the film *Harry Potter and the Chamber of Secrets*, she developed severe psychotic symptoms. She reported hearing voices 50% of the time. She further reported that the voice came from a dementor (the evil "soul-sucking fiends" serving as the guardians of the prison Azkaba). She also experienced perceptual disturbances in the visual and olfactory areas. She said that the dementor would come and take her with him. She did not leave the ward because she was convinced that dementors were real phenomena and that they would suck her soul out of her body. She felt very frightened. The use of antidissociative skills and other supportive measures did not help her much; therefore, we treated her with an atypical neuroleptic. After some weeks, the psychotic symptoms and her paranoid tendencies disappeared, and her hostility decreased.

Does Comorbidity Increase the Risk of Psychosis in Borderline Personality Disorder?

The appearance of psychotic symptoms in BPD patients has been attributed to comorbidity with mood disorders [20], post-traumatic stress disorder (PTSD) [21••, 22, 23], and severe substance use disorders (SUDs) [24]. For example, in a study of our group, we found comorbidity rates of 27% for SUDs, 88% for affective disorders, and 59% for PTSD in a clinical sample of 202 BPD patients (67% female) [25]. Other studies reported comorbidity with bipolar disorders in individuals with BPD ranging from 7% to 15% [26].

Mood Disorders

Some authors have argued that mood disorders may indicate a predisposition to the development of BPD [21••] or that BPD should be better classified as a condition of recurrent unstable mood [27]. Accordingly, an increasing number of scientists argue that psychotic disorders represent a continuum of variation of a mood disorder, undermining the Kraepelinian dichotomous classification of the psychoses [28]. Consistent with this assumption, Benvenuti and colleagues [20] found that lifetime manic-hypomanic mood dysregulation was associated with psychotic experiences in BPD patients, even in the absence of a mood disorder.

Substance Abuse and Psychotic Experiences

As mentioned previously, SUDs often appear as a comorbid disorder with BPD. Most studies have revealed comorbidity rates for SUDs in BPD patients of more than 50% [29]. Diagnoses of BPD and an SUD lead to worse outcomes [30], longer remission times [26], and a greater severity of suicidality than a BPD diagnosis only [31]. SUDs seem to influence the experience of psychotic symptoms. For instance, in a sample of drug users (cocaine, cannabis, opiates, and amphetamine), 27% of opiate abusers and up to 100% of severely amphetamine-dependent individuals reported the experience of psychotic symptoms during the use of or withdrawal from the specific substances. Independent of the specific substance, there was a linear relationship between psychotic symptoms and dependence severity [32•]. Thus, it was assumed that BPD patients with severe SUDs may also have a higher risk of developing psychotic symptoms. However, although some studies found an increase in psychotic symptoms in BPD patients with SUDs compared with BPD patients without SUDs (eg, visual hallucinations) [15, 33], other studies could not replicate these findings [34]. In sum, there is no consistent empiric evidence for an increased risk in psychotic symptoms in BPD patients with SUDs.

Post-traumatic Stress Disorder

Considering that BPD patients often experience severe childhood abuse (eg, 70% reported sexual or physical abuse or severe emotional neglect [35] and ~ 60% also have PTSD [25]), psychotic symptoms in BPD may be attributable to these negative experiences and comorbid PTSD [35, 36]. Therefore, some authors suggested that it is not the BPD symptoms per se that predispose individuals to psychotic symptoms, but instead traumatic experiences and a comorbid diagnosis of PTSD [21••, 22, 23]. In the following section, we present studies examining the link between traumatic events and psychosis in general and in BPD patients in particular.

Association Between Maltreatment in Childhood and Psychotic Symptoms

There is accumulating evidence for a link between childhood trauma and psychotic or schizotypical symptoms in the general population [21••, 22, 23, 37-39]. The link is still significant when investigators control for family history of psychotic symptoms and neurodevelopmental disturbances [21••], although some authors reported that the associations between childhood trauma and psychotic symptoms are much stronger for those with psychosis proneness (eg, high neuroticism) [38].

Although most studies found that more severe childhood abuse leads to more severe psychotic symptoms [22, 23, 37-39], different findings emerged with regard to specific associations between the type of trauma and type of symptom. For example, emotional abuse and neglect are most strongly associated with schizotypical symptoms, and physical assault is related to positive symptoms in general, whereas sexual assault is specifically associated with hallucinations [21••, 23]. Furthermore, one study revealed that all types of childhood trauma (rape, molestation, physical abuse, neglect) were associated with visual hallucinations, whereas rape and molestation were specifically related to auditory hallucinations, and physical and sexual abuse were linked to tactile hallucinations [37]. In another study, childhood sexual abuse was more strongly related to hallucinations than childhood physical abuse, while the combination was worst [39]. However, adult abuse or experience of a traumatic event seems to be less strongly associated with psychotic symptoms than childhood trauma [21••, 39], suggesting that childhood traumatic experiences may cause neurodevelopmental changes that in turn increase the risk of subsequent psychotic reactions.

BPD patients in particular experience many traumatic life events, such as physical and sexual abuse, as well as invalidation. Moreover, individuals with BPD seem to react very strongly to daily life stress with increased general psychotic symptoms and hallucinations, even compared

with patients with psychotic disorder and cluster C personality disorder [13••, 40]. This altered stress reactivity may be explained by a dysregulation of the hypothalamic-pituitary-adrenal axis [35, 41] that in turn may lead to a process of sensitization [35, 42]. For example, BPD patients with high dissociation levels show a delayed cortisol response to psychosocial stress [40] and greater cortisol and noradrenergic reactivity to stress, supporting a neurobiological basis for these symptoms [43]. Figure 1 presents a heuristic model that summarizes the above-mentioned findings showing possible pathways from trauma to BPD and psychotic symptoms.

Is There a Genetic Link Between Borderline Personality Disorder and Schizophrenia or Other Psychotic Disorders?

Because stress-related psychotic experiences are not specific to psychotic disorders and are also evident in BPD, it was assumed that the two disorders share a broader vulnerability that underlies both [44].

In terms of a diathesis-stress approach, the current main etiologic theories of both BPD and psychotic disorders assume, along with environmental factors, a substantial genetic component as the underlying cause of the respective disorders. Indeed, results of twin, family, and molecular genetic studies strongly suggest that BPD [45, 46•] and psychotic disorders [47] are moderately to highly heritable.

This raises the question of whether psychotic symptoms in BPD can be explained by a common genetic factor underlying BPD and psychotic disorders. Family studies that assessed psychotic Axis I and II disorders in relatives of BPD probands found prevalence rates ranging from 0.7% to 4.6% but mostly no significant coaggregation [48]. One study that found a heightened prevalence of schizophrenia in relatives did not use the *DSM-III* borderline criteria in the probands or family members but relied on Kernberg's criteria, making it difficult to compare this study with the others [49]. Additionally, most of the studies reporting an increased prevalence of psychotic disorders in relatives of BPD individuals did not control for schizotypal participants in the borderline sample [49, 50]. This suggests that it is the comorbidity of BPD and schizotypal personality

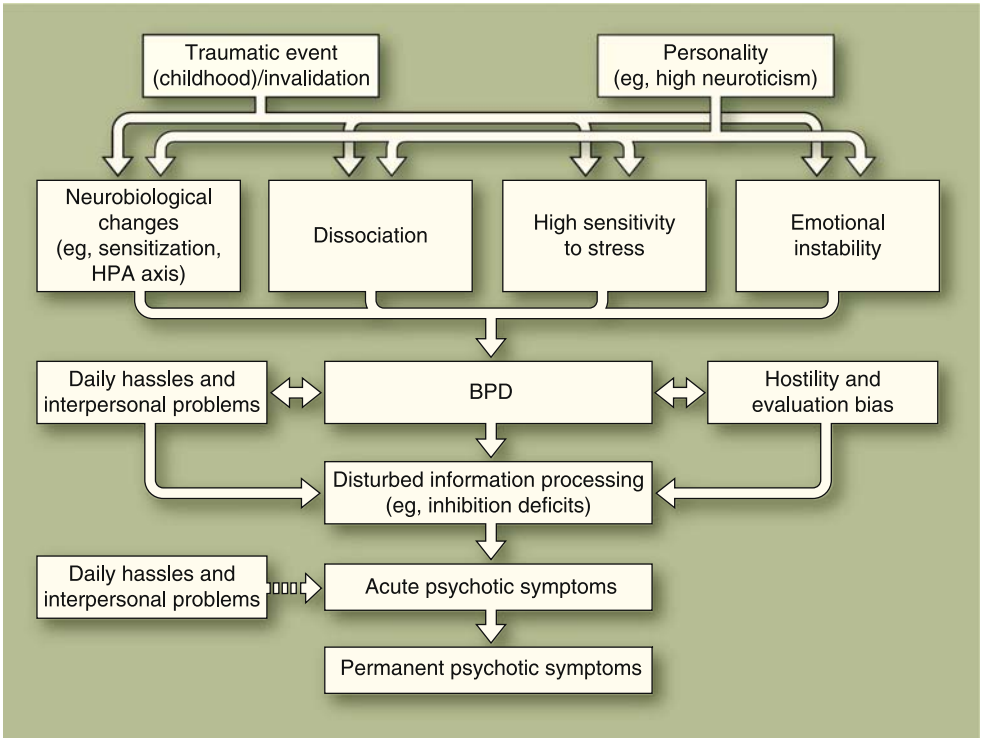

Fig. 1 Heuristic model suggesting a link between childhood trauma/invalidation and several trauma-related neurobiological and psychological changes. These changes include sensitization of the hypothalamic-pituitary-adrenal (HPA) axis, dissociation, high sensitivity to stress, and emotional instability that in turn may increase the risk for borderline personality disorder (BPD). Furthermore, current main approaches assume that a sensitive temperament (eg, neuroticism) plays a crucial role in the etiology of BPD, and BPD symptoms in particular have been shown to be related to neuroticism. In the next step, we hypothesize that BPD patients who experience severe interpersonal stress may develop information processing problems (eg, inhibition deficits) that in turn lead to acute psychotic reactions and related symptoms. Also, comorbid disorders such as post-traumatic stress disorder or manic depressive disorder and a malevolent evaluation bias may increase the vulnerability for psychosis in BPD patients and may have an impact on the course of psychotic symptoms in those patients

disorder (SPD) rather than BPD alone that correlates with an increased prevalence of psychotic disorders in relatives. However, as noted by Stone et al. [48], some studies that did not find significant coaggregation had only low prevalence rates of schizophrenia among the relatives and thus may have underestimated the coaggregation with this disorder due to the lack of sufficient statistical power to detect small or medium effects. Furthermore, it can be argued that family studies alone cannot give final answers to the questions regarding the genetic link between BPD and psychotic disorders, as they cannot distinguish between the causal effects of genetic and environmental factors. Thus, previous results of family studies should be confirmed in future research, assuring sufficient statistical power and adopting more sophisticated methods (eg, molecular genetic approaches). Nevertheless, summarizing the research findings received thus far, no empiric evidence indicates that BPD is genetically linked to psychotic Axis I and II disorders.

However, despite lacking support for a shared genetic vulnerability (ie, direct genetic link between BPD and psychosis), there is some evidence for an association via other (third) variables (ie, a more indirect link). For example, recent studies have demonstrated a genetic link between affective spectrum disorders and schizophrenia [32•] and BPD [45]. Together with the fact that BPD and depressive disorders often co-occur [25, 48], this has led to an actual debate over whether BPD should be reclassified as a mood disorder [43].

A further possible link between BPD and psychosis may be mediated by common personality dimensions that may underlie both conditions. Current main approaches [51] assume that a sensitive temperament (eg, neuroticism), which has been shown to be largely heritable [46•], plays a crucial role in the etiology of BPD, whereas some authors even conceptualize BPD as an extreme form of neuroticism [52]. In fact, BPD, and psychotic symptoms in BPD patients in particular, have been shown to be related to neuroticism [46•, 53]. Regarding psychotic disorders, evidence suggests that early neuroticism predicts later psychotic symptoms [54] and increases the risk for schizophrenia. Thus, BPD and psychosis may be linked by a common genetic factor underlying neuroticism. Further studies are needed to discover the role of neuroticism leading to psychotic symptoms in BPD and psychotic disorders.

Neurobiological Findings in Psychosis and Borderline Personality Disorder

Structural Magnetic Resonance Tomography Studies

BPD and schizophrenic patients exhibit common abnormalities in brain regions that are associated with emotion regulation (amygdala, prefrontal cortex), memory (hippocampus), and the integration of sensory information or attention (parietal cortex) [55], although some studies have found preserved volumes (Table 1). The unspecific findings may imply a general biological vulnerability to the development of psychiatric disturbances. Many studies have found evidence for a reversed temperoparietal asymmetry in schizophrenia and reduced brain volume in the superior temporal cortex in schizophrenia and SPD [51]. In a recent study comparing SPD and BPD patients, brain reduction in the superior temporal cortex—but not in the prefrontal cortex and amygdala—was found in SPD patients, indicating a core region of psychosis [56••]. Studies investigating the structural neural correlates of psychotic features (acoustic hallucinations or delusions) in BPD are sparse. Only one imaging study has compared BPD patients with and without SPD [57]. Psychotic symptoms and schizoid personality traits (emotional coldness, no interest in social relationships) in BPD patients were associated with reduced parietal leftward asymmetry resulting from reduced right parietal cortex volume.

Functional Imaging Studies

Dysfunctions in the fronto-limbic system have been found in BPD and schizophrenia patients. BPD patients typically show exaggerated activation in the amygdala and dysfunctional prefrontal activity during emotional processing. In contrast, schizophrenic patients have decreased activation in the amygdala and prefrontal cortex with concomitant abnormally increased phasic arousal, indicating a disconnection in brain and body systems for processing and appraising signals of potential danger [58].

Pathophysiology

Previous studies have reported aberrant dopamine and serotonin signaling in the prefrontal cortex, striatum, and temporal cortex in schizophrenia patients [59]. Biological, neuroendocrine, and imaging studies also provide evidence for the involvement of serotonergic activity in impulsive aggression in BPD, whereas evidence of dopaminergic dysfunction in BPD originates only from psychopharmacologic studies [60]. Also, hypofunction of the N-methyl-D-aspartate receptor, a class of glutamate receptors, in the prefrontal cortex has been found to be critically associated with the symptom manifestation of schizophrenia and BPD and is assumed to be a fundamental basis of psychosis [60]. The findings indicate that similar neurotransmitter systems are dysfunctional in BPD and psychosis. These overlapping findings may originate from traumatic experiences in schizophrenia and BPD patients [61]. Table 1 displays the neurobiological findings in BPD and schizophrenia.

Table 1 Structural and functional imaging findings in BPD and schizophrenia in fronto-limbic, temporal, and parietal brain regions

Brain region	Findings in BPD	Findings in schizophrenia
Hippocampus		
MRT	• Reduced volume [69]	• Reduced volume [55]
	• No volume reductions [57, 70]	
fMRI	–	• Hypofunction in response to facial expressions [71, 72]
Amygdala		
MRT	• Reduced volume [69]	• Reduced volume [55]
	• Increased volume [73]	• Increased volume in women [74]
	• No volume reductions [70, 75]	
fMRI	• Enhanced activity in response to affective pictures and trauma scripts [69]	• Decreased activity in response to negative stimuli in paranoid schizophrenia [58]
Anterior cingulate cortex		
MRT	• Reduced volume [69]	• Reduced volume [76]
	• No volume reductions [57, 70]	• No volume reductions [77]
fMRI	• Dysfunctional activation in response to negative emotions and trauma scripts [69]	• Hypofunction in response to facial expressions [71]
PFC (inferior, middle, orbital)		
MRT	• Reduced volume in orbital cortex [78]	• Reduced volume in right inferior and middle PFC [79]
fMRI	• Decreased activation to trauma scripts [80]	• Decreased activity of orbital PFC in response to facial expressions [71]
Dorsolateral PFC		
fMRT	• No activation in response to trauma scripts [81]	–
Parietal cortex		
MRT	• Reduced volume [57]	• Reduced volume
	• Reduced leftward asymmetry in psychotic BPD patients [57]	• Reduced leftward asymmetry [51]
fMRI	–	• Decreased activity in response to facial expressions [71]
Superior temporal cortex		
MRT	• No differences	• Reduced volume in schizophrenia [55] and SPD [56••]

BPD borderline personality disorder, *fMRI* functional MRI, *fMRT* functional magnetic resonance tomography, *MRT* magnetic resonance tomography, *PFC* prefrontal cortex, *SPD* schizotypal personality disorder

Treatment of Borderline Personality Disorder Patients with Psychotic Symptoms

Patients with BPD who show severe and permanent psychotic symptoms also often suffer from psychosocial disintegration and isolation and therefore often need a structured and supportive inpatient treatment setting. In the case of acute and severe psychotic symptoms, stress management and affect stabilization in combination with psychopharmacologic treatment are suggested [62, 63]. For instance, the American Psychiatric Association guidelines state that psychopharmacotherapy is indicated to treat symptoms during periods of acute decompensation [62]. More specifically, the American Psychiatric Association guidelines recommend choosing antipsychotics for cognitive perceptual symptoms such as paranoid ideation, hallucinations, and delusions. These suggestions are based on early studies regarding the treatment of BPD with atypical antipsychotics that revealed the efficacy of clozapine [63, 64]. More recently, one study that reviewed several psychopharmacologic treatment studies with BPD patients concluded that clozapine, quetiapine, and olanzapine are effective in treating cognitive-perceptual symptoms in BPD patients [63]. In another study that examined individuals with BPD who experienced psychotic symptoms but had no comorbid psychotic disorder, the authors revealed that a combination of clozapine and psychotherapy yielded significant improvements in cognitive-perceptual distortions [65]. Furthermore, the combination of dialectical behavioral therapy and atypical neuroleptics may be more effective than psychopharmacologic treatment alone. For example, preliminary data show that the combination of dialectical behavioral therapy and trauma therapy is effective in treating PTSD symptoms in BPD patients with comorbid PTSD and high dissociation [66].

Conclusions

To conclude, psychotic symptoms in BPD patients are often trauma–and stress-related. Therefore, clinicians need to adequately contextualize the patient's symptoms by investigating trauma-related histories as well as daily stressors that may trigger symptom presentation. Dissociative symptoms such as derealization, depersonalization, and paranoid ideation often disappear when the trigger can be identified or when patients learn to cope with dissociation. However, a substantial group of BPD patients develop permanent and more severe psychotic symptoms. Furthermore, empiric evidence indicates that comorbidity with bipolar or cyclothymic disorder and associated mood dysregulation and/or additional PTSD may predispose BPD patients to develop more severe psychotic symptoms under stressful conditions. Thus, functional assessment of psychotic symptoms in BPD patients is strongly emphasized.

Although some overlap exists in structural, functional, and physiologic findings in BPD and psychosis, results of several studies also indicate some notable differences: 1) schizophrenia is associated with a reversed temporoparietal asymmetry and reduced volume in the superior temporal cortex and 2) although both disorders show fronto-limbic dysfunctions, exaggerated amygdala response seems to be specific to BPD, whereas decreased amygdala activity is related to positive symptoms in schizophrenia. Studies on neurotransmitters yield similar results for BPD and schizophrenia, indicating nonspecific dysfunctions. Future research should control for psychotic symptoms in BPD and trauma experience to reveal brain abnormalities and dysfunctions specifically related to these conditions.

Regarding treatment of BPD patients with cognitive-perceptual symptoms, one study summarized that antipsychotics may have some benefits for BPD patients, but no evidence indicates an advantage of one antipsychotic over another [67]. Moreover, most studies have major limitations, including small sample sizes, short duration of treatment, high dropout rates, heterogeneity of selection criteria, and missing long-term follow-up evaluations [68••]. Thus, further research should examine the effects of antipsychotic treatment in BPD patients with narrowly defined psychotic symptoms, but without comorbid psychotic disorder, using longitudinal data. To determine the differential effect of psychotherapy and psychopharmacotherapy, BPD patients also should be randomly assigned according to antipsychotic treatment alone or in combination with psychotherapy. The evidence for the importance of traumatic events during childhood and PTSD in the development of subsequent psychotic symptoms in BPD patients would support the usefulness of trauma-focused therapy in these patients, although only preliminary data support this assumption. In summary, we emphasize four therapy steps that may be helpful for clinicians confronted with BPD patients who show psychotic symptoms:

1. Functional assessment of symptoms to understand which factors have triggered psychotic symptoms and how severe and permanent they are.
2. Stabilization of patients: providing a highly structured and supportive environment (inpatient treatment) and/or treating psychotic symptoms with atypical neuroleptics. Additionally, providing skills to reduce tension and dissociation.
3. After stabilization of the patient, evidence-based therapy (eg, mentalization-based therapy, dialectical behavioral therapy, schema-focused therapy, or transference-focused psychotherapy) in outpatient or partial hospitalization settings is necessary. Additionally, cognitive trauma therapy for BPD patients with PTSD and high levels of dissociation seems to be helpful.
4. Booster sessions (eg, 3 months after therapy) should be considered: does the patient use skills to reduce interpersonal vulnerability? How does he or she cope with daily hassles? Is antipsychotic medication still necessary?

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