The Biological Basis to Personality Disorders

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ABSTRACT

Objective: To provide understanding into the biological basis of thinking and behavior in people with personality disorders, explain anatomic findings, and appraise therapeutic options.

Data Sources: PubMed was searched with no date restrictions using the terms *personality disorders DSM-5*, *cluster B personality disorders*, *biological psychiatry of personality disorders*, *neurobiology of personality disorders*, and *neurobiology of cluster B personality disorders*.

Study Selection/Data Extraction: We identified 2,790 English-language articles and utilized 18 in this report.

Results: There are anatomic features typical to the brains of individuals with cluster B personality disorders, for example, abnormalities in the superior frontal cortex and amygdala and enlarged striatal volumes. Emotional dysregulation and impulsiveness are 2 prominent symptoms. Hereditary factors may contribute to the development of such conditions.

Conclusion: Understanding the neurobiology of cluster B personality disorders expands knowledge that hopefully results in better clinical management and development of improved treatments. Psychotherapy is currently the most effective intervention for borderline personality disorders. Symptomatic pharmacotherapies may be prescribed adjunctively on an individualized basis if clinically indicated (eg, with a coexistant depression).

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Biological psychiatry is the study of the biological function of the nervous system in persons with mental disorders.[1] The discipline focuses on research to clarify the basis of psychiatric diagnoses. Investigations include the biochemical, genetic, physiologic, neurologic, and anatomic basis of behavior. On a clinical level, biological psychiatry reviews therapies such as drugs, diet, exercise, avoidance of environmental threat, and development of coping skills to deal with life stress; all of these interventions can induce biochemical changes.[2] The field explores functional neuroanatomy, imaging, neuropsychology, and pharmacotherapeutic options.[1]

Psychotherapy was prominent from the 1900s until the 1950s, when the first antipsychotic and antidepressant medicines were introduced. Research on pharmaceutical mechanisms of action provided an early biological explanation of mental disorders known as the catecholamine theory.[3] Later, the catecholamine theory was replaced by the monoamine theory, which included serotonin. The monoamine theory formulated the "chemical imbalance" hypothesis of psychiatric diagnoses, which conceptualized modern biological psychiatry.[3] With a focus on biological brain function, development of drug-based treatments for mental disorders became more prominent.

Personality disorders are long-term patterns of experience and behavior that are pervasive and inflexible and deviate markedly from cultural expectations. Personality disorder onset in adolescence leads to distress and social impairment. Personality disorders are divided into 3 clusters. People with cluster A disorders (prevalence of 5.7%) often appear odd or eccentric.[4] Individuals with cluster B (prevalence of 1.5%) are dramatic, emotional, or erratic.[3] Those with cluster C patterns (prevalence of 6%) feel anxious or fearful. Approximately 15% of adults have at least 1 personality disorder.[4] The 3 clusters share symptoms; however, their neurobiology has not been investigated. Personality disorders are often associated with conditions such as substance use and mood and anxiety disorders and are prevalent in psychiatric patients. Understanding symptoms of personality disorders might help in their management, including comorbid psychiatric concerns.

The aim of this report is to provide understanding into the biological basis of thinking and behavior in people with personality disorders, explain anatomic findings, and appraise therapeutic options. Cluster B is more commonly seen in clinical practice, thus we focus on this personality pattern with regard to neurobiology.

LITERATURE SEARCH

PubMed was searched with no date restrictions using the terms *personality disorders DSM-5*, *cluster B personality disorders*, *biological psychiatry of personality disorders*, *neurobiology of personality disorders*, and *neurobiology of cluster B personality disorders*. We identified 2,790 English-language articles and used 18[1–3,5–19] in this report.

CLUSTER B NEUROBIOLOGY

The psychobiology of personality disorders is best understood in the cluster B group. Expressions of borderline personality disorder include emotional dysregulation, poor response inhibition, impulsive or externalizing behaviors, and strong substance use associations.[5] The amygdala evidences an important role in modulating vigilance and generating a negative emotional state; it processes emotional stimuli and reactions. Substantial amygdala activation is related to emotional vulnerability, especially with

Clinical Points

- People may be genetically predisposed to personality disorders.
- Neurophysiologic changes in the amygdala and limbic system of persons with borderline personality disorders are documented.
- Psychotherapy is the primary intervention in treating patients with personality disorders.

disturbed interpersonal relations. A hyperreactive amygdala could predispose individuals with borderline personality disorder to be hypervigilant and overreactive to other people's emotional expressions; these individuals might also exhibit ambiguity about the attitudes of others.[6] In addition to excessive amygdala reactivity, a low threshold for impulsive aggression may also be related to reduced prefrontal inhibition and reduced serotonergic brain activity. Affective instability may be mediated by excessive limbic activity in GABAergic or cholinergic circuits, resulting in an increased reactivity to emotional stimuli.[7] Neuroimaging studies[8–10] of subjects with symptoms of borderline personality disorder or a diagnosis of a cluster B personality disorder reveal abnormalities in frontolimbic circuitry, including at the ventral striatum, amygdala, hippocampus, insula, and orbitofrontal, prefrontal, or cingulate cortexes. There is a significant imaging-recorded inverse correlation between trait impulsiveness and the left superior frontal cortex; it is postulated that cortical thickness of the superior frontal cortex predicts impulsivity.[11] Striatal volumes tend to be enlarged in subjects with symptoms of cluster B personality disorder.[8]

There may be a genetic component for the development of borderline personality disorder in that there are genetic influences on traits such as neuroticism, impulsivity, anxiousness, lability, and insecurity. Borderline personality disorder is about 5 times more common among first-degree relatives than in the general population. The clinical course of borderline personality disorder suggests that it is a heterogeneous condition. Childhood sexual abuse, early age at first psychiatric contact, chronicity of symptoms, affective instability, aggression, substance abuse, and other comorbidities are predictors of a poor prognosis. These factors and physical abuse or witnessing domestic violence are associated with borderline personality disorder.[12]

Psychopathic traits are associated with abnormalities in the amygdala, orbitofrontal cortex, anterior cingulate, posterior cingulate, hippocampus, and superior temporal gyrus.[13] Several neurobiological models of psychopathy have been postulated but have limitations. One model proposes dysfunction of the amygdala and orbital frontal cortex.[14] Another theory relates to paralimbic structures.[14] Psychopathy is sometimes considered to have a genetic component.[15] A low-expression variant polymorphism influencing monoamine oxidase is linked to the structural volume of the amygdala, cingulate cortex, insula, hypothalamus, and orbitofrontal cortex; these abnormalities are associated with impulsive, reactive aggression.[15,16] Antisocial personality disorder is more common among first-degree biological relatives than in the general population.[4] Adoption studies[3] indicate that genetic and environmental factors contribute to people developing an antisocial personality disorder. Adopted and biological children of parents with antisocial personality disorder have an increased rate of that same disorder.[17] The adoptive family environment influences the risk of developing a personality disorder and psychopathology. Being subjected to abuse or neglect during childhood and experiencing an unstable or violent, chaotic family life during childhood are other contributing factors.

TREATMENT

The traditional intervention for borderline personality disorder is psychotherapy. Dialectical behavior therapy, schema-focused therapy, mentalization-based therapy, systems training for emotional predictability, and problem-solving or transference-focused psychotherapy are the psychotherapies that reportedly evidence efficacy.[18] Medications may be prescribed to curb symptoms of depression, impulsiveness, aggression, or anxiety.[19] Psychotherapy is sometimes helpful to mitigate overt expression of antisocial personality disorder symptoms. Therapy may include anger/violence management and treatment for substance abuse or other psychiatric conditions. Psychotherapy is most effective when symptoms are of moderate intensity in patients who are motivated for improvement.

CONCLUSION

Understanding the neurobiology of cluster B personality disorders expands knowledge that hopefully results in better clinical management and development of improved treatments. Psychotherapy is currently the most effective intervention for borderline personality disorders. Symptomatic pharmacotherapies may be prescribed adjunctively on an individualized basis if clinically indicated (eg, with a coexistent depression).

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