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What Causes Borderline Personality Disorder and What Can We Do?

-A Brief Literature Review on the Etiology of BPD in the Last 20 Years

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Abstract: Borderline personality disorder (BPD) is an increasingly prevalent psychological disorder in modern society, with approximately 1-2% of the population suffering from it, but its etiology is still unknown. The search for the causes of BPD is urgent, as research into the etiological mechanisms is crucial to clinical diagnosis and treatment. This paper has shown that significant advances have been made in the biological and psychological fields regarding the causes of BPD. Besides, macro-environmental factors also significantly impact BPD, such as discrimination and stigmatization of psychological problems and a lack of attention to mental health care. This paper aims to update the latest research on the etiology of BPD, draw out new findings from it, and suggest what can be changed and what can be done within our control.

Keywords: borderline personality disorder, etiology, biology, genetics, psychology, macro environment, revelation

1. Introduction

As modern society becomes increasingly concerned with psychological issues, the author notices that more people around us have psychological disorders. However, the etiology of these psychological problems is very complex, and an understanding of the etiological mechanisms directly affects clinical diagnosis and treatment. In addition to common psychological problems such as depression, autism, and obsessive-compulsive disorder, a borderline personality disorder is becoming more common in clinical practice. According to research, approximately 1-2% of the population in countries such as the USA, the UK, and Norway have borderline personality disorder problems, which is even more common in psychiatric clinics [1]. So, what exactly is causing borderline personality disorder, and what can the author do about it? It is a question worth exploring.

This review of research on the etiology of BPD reveals that:

1. Researchers agree on describing the symptoms of BPD but not on its etiology.

2. The diagnostic criteria for BPD have changed positively with the efforts of researchers, but there is still debate about its classification.

3. The neurological findings of the brain in patients with BPD have been remarkable, and the central brain regions that affect BPD have been identified. However, genetically, although

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individual genes that may affect BPD have been identified as having interactions between genes and the environment or between genes, it is not clear how heritable BPD is and which genetic changes affect the development and progression of BPD.

4. Research into the etiology of BPD is better developed in psychology, offering more possibilities for treating BPD through psychology.

5. Research on the etiology of BPD is mainly based on psychology and biology, neglecting systemic factors.

6. Emphasis has been placed on the impact of co-morbidities on BPD.

These findings suggest a need for more research in genetics and systemic factors, while co-morbidity's impact should not be ignored. The academic community will soon reach a consensus on the diagnostic criteria and classification of BPD so that more patients can benefit and make the clinical diagnosis and treatments more delightful.

2. Literature Review

Stern first introduced borderline personality disorder (BPD) in 1938. However, at the time, it was impossible to analyze its causes, there was no effective treatment, and there was considerable disagreement in different countries about how BPD should be diagnosed and treated [2]. Furthermore, the name BPD alone has been long debated, with many believing it to be a mood disorder rather than a personality disorder [3]. The author is still in the exploratory stage of this disorder. Still, it is good to know that the author has made some discoveries about its etiological mechanisms. Research on the etiology of BPD has mainly focused on psychological and biological aspects. However, some researchers believe that external macro factors should be considered and that BPD's etiology should be a complete system of elements that interact directly [4]. Research on the etiology of BPD has mainly focused on psychological and biological aspects. However, some researchers believe that external macro factors should be considered and BPD's etiology should be a complete system of elements that interact directly. Therefore, in this article, the author will review the results of the last 20 years of research on the etiology of BPD, mainly through a systematic review of the current study. It is hoped that the etiology analysis will provide insight into the pathogenesis of BPD and allow us to discover how to avoid and respond to the disease.

3. Biological Etiology

Research on the biological aspects of etiology has mainly focused on the neurological and genetic characteristics of the brain. However, some studies have also found that gender and ethnicity impact BPD, which the author describes here in three parts [5,6].

3.1. Ethnicity & Gender

It has been suggested that the race of people with BPD symptoms should be considered when assessing and treating them [6]. Depending on the ethnicity, specific symptoms may have unique correlates. For example, when treating white patients with BPD characteristics, assessing their emotional symptoms may be essential, as developing a treatment plan that addresses emotional issues. Moreover, it may be necessary for Hispanic or African American patients to explore the sociocultural factors of race and how they are perceived.

Regarding gender, one study showed that BPD is diagnosed more often in women than men. Women show more emotional instability, identity issues, and borderline characteristics regarding negative relationships, but not self-harm, compared to men. This study also suggested that BPD symptoms and general personality traits are stable between 30 and 40 and diminish with age [7]. Considerations of race and gender factors have implications for the clinical diagnosis of BPD.

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3.2. Neuroscience

Brain regions with abnormalities in BPD are concentrated in the front-limbic system, including areas of the orbitofrontal cortex, dorsolateral prefrontal lobe, anterior cingulate, amygdala, and hippocampus. Although compared to healthy groups, the hippocampus, and amygdala are smaller in size in BPD patients, especially on the left side, brain regions used for emotion regulation and control are hypometabolic in BPD patients [8]. In contrast, limbic regions are over-activated, and resting-state functional connectivity in the amygdala and dorsolateral prefrontal lobe is also abnormal in BPD patients [9,10]. Orbitofrontal cortical dysfunction may contribute to some of the core features of BPD, such as impulsivity. However, other features, such as heightened moodiness and personality irregularities, people do not appear significantly related to the dysfunction produced by orbitofrontal cortical damage [11]. It has also been shown superficially that patients with BPD have abnormalities in brain gray matter [8]. A study in which BPD symptoms no longer met the diagnostic criteria of the DSM-5TR following an increase in brain gray matter in critical areas of the brain following the practice of positive thinking in patients with BPD seems to provide side evidence that changes in brain gray matter in key brain areas have an impact on the symptoms of BPD [12]. However, it has also been suggested that BPD is unrelated to brain structure [13].

3.3. Genetics

The genetic aspects of the etiology of BPD have been less studied, and genetic studies of BPD are still at a very early stage compared to other major psychiatric disorders, including mainly individual gene polymorphisms, gene and environment interactions, gene and gene interactions, and epigenetic studies. The most numerous individual gene studies are of the 5-hydroxytryptamine transporter (5-HTT) gene and the dopamine transporter gene SLC6A3 (DAT1), both of which are thought to play an essential role in causing some of the symptoms of BPD. However, a growing body of research recognizes that the complexity of BPD cannot be described by single gene effects alone and that genetic and environmental impacts should be combined. Some studies have shown the contribution of HPA axis gene variants to BPD's pathogenesis and highlighted the moderating role of childhood trauma in developing the disease [14,15]. Genes and gene interactions can also influence the development of BPD. Several studies of genes related to the dopaminergic system have identified associations between BPD and the dopamine transporter gene (DAT1), 223, the DRD4 gene 224, and the catechol-O-methyltransferase gene 225, which may underlie some of the symptoms and features of BPD [16]. Gene methylation plays a vital role in maintaining genes in a long-term inactivated state, and epigenetic-related studies have shown that promoter methylation of rDNA and PRIMA1 is significantly abnormal in the blood of BPD patients compared to healthy populations [17].

It has been suggested that BPD is partially heritable and that some traits that people with BPD have, such as emotional instability and impulsivity, are more common in their relatives than in relatives of people with other personality disorders. Behavioral genetic studies have also shown that emotional stability and impulsivity are primarily heritable, with heritability coefficients of 50% and 80% [5].

BPD may be heritable, but the samples in studies on the genetics of BPD are relatively small, and analyses are difficult to achieve statistical significance. Furthermore, with the complexity of the etiology of BPD and the difficulty of entirely reducing factors in animal studies, there is still a long way to go in studying the genetic etiology of BPD [18].

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4. Psychological Etiology

This review divides the psychological etiology of BPD into three parts: pregnancy, childhood, and adolescence.

4.1. Prenatal Factors

While most research suggests that adverse childhood experiences are the leading cause of BPD symptoms, some studies indicate that prenatal adversity, such as tobacco exposure, medical complications, and prenatal maternal stress, may play a role in the pathogenesis of BPD and may interact with childhood adversity to play an essential role in the development of BPD [19]. However, there are very few studies on prenatal adversity, and more research is needed to demonstrate its effectiveness.

4.2. Childhood

Borderline personality-related traits in children share standard etiological features with borderline personality disorder in adults, and adverse childhood experiences play a vital role in the development of BPD [20]. The author divides negative childhood experiences into trauma, damaging attachment types, and educational styles.

The most common types of childhood trauma are various types of abuse, including physical, psychological, and sexual abuse. Moreover, abuse from different parental parties affects patients differently by gender; in females, maternal and paternal abuse is directly associated with BPD-related symptoms. In males, only paternal abuse is directly related to BPD-related symptoms [21]. Extremely stressful environments during childhood can lead to physiological changes that alter the functional connections between emotion regulation structures and social cognitive responses (part of the frontal mind circuitry). Such alterations can disrupt notions of self and others and produce unrealistic, unstable, and disproportionate representations of perceived and expressed emotions, indicative of the clinical features of individuals with BPD, including aggression, impulsivity, and dissociative symptoms [22]. Children exposed to more unique maternal negative expressions of emotion and physical abuse show more BPD-related signs, and this was confirmed by a study of childhood abuse in people with BPD from China, which suggested that maternal physical abuse was as strong a predictor of BPD as sexual abuse in China [20, 23].

In addition to physical and psychological abuse, poor attachment type and upbringing during childhood are also associated with BPD symptoms. In females, paternal abuse was indirectly related to BPD-related symptoms through attachment anxiety rather than attachment avoidance. In males, maternal abuse was indirectly associated with BPD-related symptoms through attachment anxiety rather than attachment avoidance [21]. There are also significant differences in how parents treat their children in families with a positive psychiatric history compared to families without a history of psychosis, with families with a positive psychiatric history treating children relatively harshly, making them more likely to develop BPD-related symptoms [20].

These studies of the direct association between adverse childhood experiences and BPD symptoms provide insight into the prevention and treatment of BPD.

4.3. Adolescent

The etiology of adolescent BPD is similar to that of children, with the difference that at this time, adolescents enter adolescence and begin to develop interpersonal relationships. Negative interpersonal relationships can also develop BPD-related symptoms [24]. Rejection and bullying among peers are common negative relationships, and the harm caused by bullying can lead to

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negative relationship patterns, altered social perceptions, and a tendency to over-psychologize. Negative prejudice appears to be particularly acute for BPD following exclusion and bullying. In such cases, a person exhibiting dysregulated behavior and repeatedly encountering negative interactions with others may develop maladaptive social strategies or 'emotionally unstable patterns of interaction,' manifesting as core relational symptoms of BPD.

The neurological abnormalities observed in adults with BPD may not only result from chronic depression or long-term substance use [25]. Consistent with adult reports, some adolescents with BPD exhibit structural (gray-white) changes in the marginal frontal area and neuropsychological abnormalities (i.e., decreased executive functioning and social cognitive impairment).

There is a lack of research on the development of BPD in adolescence, as it is in the transition from childhood to adulthood and a period of rapid psychological growth, with a massive number of influencing factors that make it difficult to predict.

4.4. Macro Environmental Factors

As the author mentioned, the prevalence of BPD reaches 1% to 2% of the total population in many countries, but there is very little awareness among the general public. One researcher surveyed people's knowledge of BPD through the Vignette-identification Task and showed that most people believe that the etiology and treatment of BPD come from psychology, sociology, biology, or theology [26]. Unfortunately, current research into the etiology of BPD is also primarily based on psychology and biology, ignoring systemic factors such as discriminatory health and mental health care. Many derogatory terms are even used in psychological diagnosis and treatment to describe the symptoms of BPD, for example, not very sick and controlling. The stigmatization of BPD and its sufferers can hinder the exploration of its etiology from the outset and prevent effective treatment [27].

Although BPD is recognized in clinical settings worldwide, it is relatively uncommon in traditional societies and has yet to be broadly accepted. Furthermore, it has been suggested that developing countries' rapidly changing social environment may increase the risk of developing BPD in the modern population [1].

5. Summary and Insights

In this section, the author summarises the findings and tries to draw insights into what else the author can do.

Finding 1: Researchers agree on describing the symptoms of BPD but not its etiology. The descriptions of BPD symptoms are mainly of a general pattern of instability in interpersonal relationships, self-image and affect, and marked impulsivity, beginning in early adulthood and present in various settings (DSM-5-TR). Symptoms have also been categorized into four main areas: emotional instability, disturbed patterns of thinking or perception, impulsive behavior, and intense but unstable relationships with others (NHS). While researchers agree on the diagnostic criteria for symptoms, some consider childhood trauma the central aspect of etiology. In contrast, others think the brain's nervous system disruptions are the leading cause. As this paper seeks to explore, the etiology of BPD is complex, and the author is still refining it.

Finding 2: The diagnostic criteria for BPD have changed positively with the efforts of researchers, but there is still debate as to whether to classify it as a personality disorder. Many researchers believe most people with personality disorders refuse or resist treatment, but people with BPD do not. They even seek treatment [3], arguing that BPD is a state of mind or mood disorder rather than a personality disorder. The correct categorization and positioning of this mental

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health problem will help standardize terminology and diagnostic methods worldwide and enable doctors and patients to understand the condition better and treat it proactively.

Finding 3: The neurological findings in the brain of BPD patients have been remarkable. The central brain regions that affect BPD have been identified, which include the orbitofrontal cortex, dorsolateral prefrontal lobe, anterior cingulate gyrus, amygdala, hippocampus, and other areas. In terms of genetics, although individual genes that may affect BPD have been identified, as have interactions between genes and the environment or between genes, the author still does not know much about the heritability of BPD and which genetic changes affect the production and development of BPD. There is still a long way to go. Further research into the genetics of BPD is likely to be the next central area of research in this field. Further revelation of the genetic causes of the disease will provide new tools for the prevention and treatment of BPD.

Finding 4: Research into the etiology of BPD is better developed in psychology, offering more possibilities for treating BPD through psychology. By analyzing the patient's childhood experiences, psychologists try to identify the causes of fear of abandonment, unstable relationships, and impulsive behavior, which may and do not only include abuse, insecure attachments, incorrect upbringing, etc. Psychologists have developed several viable treatments to address patients' symptoms and experiences. Some more effective treatments for BPD are Dialectical Behavioral Therapy (DBT), Cognitive Behavioral Therapy (CBT), Positive Mindfulness Therapy, and art therapy. Many options are available to patients and offer more hope for treatment.

Finding 5: As mentioned above, research into the etiology of BPD has been primarily based on psychology and biology, neglecting systemic factors. Our families, communities, and even social environments influence our psychological development. Suppose the author takes mental health seriously, does not stigmatize mental illness, and understands psychological problems properly. In that case, all mental diseases may be detected and prevented early or even eliminated at the root.

Finding 6: The impact of co-morbidity on BPD needs to be considered, as people with BPD have a wide range of symptoms. Although most of them meet the diagnostic criteria, they may also have other physical or psychological symptoms due to individual differences. Sometimes co-morbid symptoms may completely mask symptoms of BPD, such as a depressed state after experiencing a dramatic emotional change, which can easily be mistaken for depression. As mentioned above, BPD is not fully understood in many developing countries, and the high rate of misdiagnosis may also be due to co-morbidities. In addition, a focus on co-morbidities may also help individual patients with severe BPD to be treated medically. For example, some doctors use stabilizers to moderate the symptoms of patients with highly erratic mood swings. Although the author does not yet have specific medication for BPD, many countries and doctors do not recognize the effectiveness of drugs for BPD. However, it has many similar symptoms to other psychological disorders, and treating BPD with the proper medication is an effective treatment modality. It has proven to be the one that many clinicians use more often today.

6. Conclusion

Unstable interpersonal relationships, emotions, unstable self-images, impulsive behavior, persistent feelings of emptiness, isolation, and some transient psychotic symptoms characterize borderline personality disorder. This "stable" pattern of all the "unstable manifestations" is the essential feature of borderline personality disorder.

Behind these complex symptoms are more complex causes, and over the past two decades, researchers worldwide have investigated the full range of causes of BPD with promising results. This paper summarizes the latest developments in research on the grounds of BPD over the last two decades in biology, genetics, psychology, and the macro-environment and presents several findings:

1. researchers agree on describing the symptoms of BPD but not its etiology.

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2. The diagnostic criteria for BPD have changed positively with the efforts of researchers, but there is still debate about its classification.

3. The neurological findings of the brain in patients with BPD have been remarkable, and the central brain regions that affect BPD have been identified. Genetically, although individual genes that may affect BPD have been identified, as have interactions between genes and the environment or between genes, it is not clear how heritable BPD is and which genetic changes affect the development and progression of BPD.

4. Research into the etiology of BPD is better developed in psychology, offering more possibilities for treating BPD through psychology.

5. Research on the etiology of BPD is mainly based on psychology and biology, neglecting systemic factors.

6. emphasis is placed on the impact of co-morbidities on BPD.

However, this paper is a literature review and asks what the author can do within our control from different perspectives—for example, correctly categorizing and positioning BPD as a prevalent psychological disorder and standardizing its diagnostic criteria to facilitate medical treatment worldwide. Society should also actively educate itself on mental health and not stigmatize mental illness so that people have a proper understanding of psychological problems and no longer have a taboo against treatment. Finally, this paper also provides an outlook on the future direction of research into the causes of BPD, with genetic analysis intensifying and the combination of medication and psychotherapy becoming increasingly popular.

I hope the findings in this paper will facilitate researchers' understanding of recent research advances, and these recommendations will be recognized as contributing to the future prevention and treatment of BPD.

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