



Guest Editorial

## Exploring Biomarkers and Contributing Factors in Obsessive-Compulsive Disorder: Hypothesis

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### INTRODUCTION

Obsessive-compulsive disorder (OCD) is a persistent psychiatric disorder marked by distracting ideas (preoccupations) and recurrent actions or psychological behaviors (Compulsions). Despite extensive research, the etiology of OCD remains elusive. This paper aims to delve into potential biomarkers and contributing factors associated with OCD, proposing hypotheses and theories to help us have a better grasp of this complex condition.

### BIOMARKERS IN OCD

Identifying reliable biomarkers is crucial for understanding the neurobiological basis of OCD. Several studies have explored potential candidates, with a focus on neuroimaging, genetic, and neurochemical markers.

#### Neuroimaging Biomarkers

Studies using brain imaging have repeatedly identified anomalies in the cortico-striato-thalamo-cortical (CSTC) circuitry in OCD. Functional magnetic resonance imaging (fMRI) examinations have showed stimulation in the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), and striatum, along with hypoactivity in the dorsolateral prefrontal cortex (DLPFC).<sup>[1,2]</sup> A potential biomarker hypothesis posits that aberrant connectivity within the CSTC circuit contributes to the manifestation of OCD symptoms.

#### Genetic Biomarkers

Genetic factors play a significant role in the susceptibility to OCD. Twin, family, and adoption studies suggest a heritability estimate of around 40–60%. The serotonin transporter gene (SLC6A4) and the glutamate transporter gene (SLC1A1) were related to OCD. However, no single gene can account for the entire genetic basis of OCD, suggesting a polygenic nature.<sup>[3]</sup> A hypothesis emerges that a combination of genetic variants contributes to the dysregulation of neurotransmitter systems, particularly serotonin and glutamate.

#### Neurochemical Biomarkers

OCD is believed to be associated with dysregulation of serotonin and glutamate neurotransmitters. The serotonin hypothesis posits that imbalances in serotonin levels contribute to the pathophysiology of OCD. In addition, abnormal glutamate signaling, especially in the

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cortico-striatal pathway, may play a role.<sup>[4]</sup> A hypothesis emerges that a cascade of neurochemical events disrupts normal neurotransmission, leading to the development and persistence of OCD symptoms.

### CONTRIBUTING FACTORS IN OCD

Understanding the environmental and psychosocial factors that contribute to OCD is essential for a comprehensive model of the disorder.

#### Early Life Stress and Trauma

Childhood adversity, including physical, emotional, or sexual abuse, has been linked to an increased risk of developing OCD. The hypothesis is that early life stress may lead to alterations in brain development, affecting the CSTC circuit and increasing vulnerability to OCD.

#### Neuroinflammation

Emerging evidence suggests a role for neuroinflammation in the pathogenesis of OCD. Inflammatory markers, such as cytokines, have been found to be elevated in individuals with OCD.<sup>[5]</sup> A concept has emerged that persistent neuroinflammation may lead to morphological and physiological alterations in the nervous system, potentially encouraging the development and maintenance of OCD symptoms.

#### Gut–Brain Axis Dysfunction

The gut–brain axis, connecting the gastrointestinal system with the central nervous system, has gained attention in psychiatric research. Alterations in gut microbiota and increased intestinal permeability have been observed in individuals with OCD.<sup>[6]</sup> The hypothesis is that dysfunction in the gut–brain axis may influence neuroinflammation and neurotransmitter systems, contributing to the pathophysiology of OCD.

### CONCLUSION

OCD is a multifaceted disorder with both hereditary and external variables contributing to its manifestation. This paper has explored potential biomarkers, including neuroimaging, genetic, and neurochemical markers, as well as contributing factors like early life stress, neuroinflammation, and gut–brain axis dysfunction. The proposed hypotheses and theories aim to integrate existing knowledge and guide future research toward a more comprehensive understanding of OCD. Continued interdisciplinary investigations are crucial to unravel the intricacies of this debilitating disorder, ultimately leading to improved diagnostic accuracy and targeted therapeutic interventions.

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