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Exploring the Gut Microbiome's Influence on Obsessive-Compulsive Disorder: Review of Current Literature

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Abstract

Introduction: Obsessive-compulsive disorder (OCD) is a common and disabling neuropsychiatric condition characterized by obsessions and compulsions, significantly impacting quality of life. Current treatments include pharmacological and psychological therapies. However, the exact etiology remains unclear. Growing research highlights the gut-brain axis and the gut microbiome's influence on mental health.

Aim of the study: This review aims to examine the existing literature on the interplay between the gut microbiome and OCD, exploring its pathophysiological role and therapeutic implications.

Materials and method: An extensive literature search was conducted in the PubMed database up to the year 2025.

Conclusions: This review highlights growing evidence of a significant link between the gut microbiome, the gut-brain axis, and obsessive-compulsive disorder. Changes in gut bacteria can impact brain function and contribute to OCD symptoms. While animal studies have confirmed the existence of the microbiota-gut-brain axis and suggested its role in psychiatric disorder pathogenesis, studies on human remain limited. This gap is especially apparent in out understanding of gut microbiota changes in patients with OCD, marking a critical area for future research. Although research is still emerging, microbiome-targeted interventions – such as probiotics, fecal microbiota transplantation (FMT), and dietary changes – show promise, especially for treatment-resistant OCD. To translate these findings into better patient care, we need increased awareness and more large-scale human studies. These efforts may help identify specific microbial profiles and mechanisms, leading to more effective and personalized treatments for OCD.

Keywords: gut microbiota; gut-brain axis; OCD; obsessive–compulsive disorder; probiotics, fecal microbiota transplantation.

1. Introduction

Obsessive-compulsive disorder (OCD) is a common and severely disabling neuropsychiatric condition that affects an estimated 1.3% of the population worldwide [1]. OCD is characterized by the presence of unwanted and distressing thoughts (obsessions) and repetitive rituals that the patient is compelled to perform (compulsions). The primary function of these compulsions is to reduce the tension or distress generated by obsessions [2]. These core symptoms cause a high

degree of distress or impairment in function, are extremely time-consuming, and might be a source of shame for patients [3]. The impact of OCD is not only limited to its primary symptomatology, but it also commonly results in disability and a lower quality of life [4].

Effective treatment is of critical importance in the management of symptoms and the quality of life in patients. The current standard approaches to treating OCD are pharmacological interventions and psychological therapies. First-line OCD treatment typically includes selective serotonin reuptake inhibitors (SSRIs) [5] and cognitive-behavioral therapy with exposure and response prevention [6]. In situations where traditional pharmacological and psychotherapeutic interventions are not enough, neuromodulation techniques such as deep brain stimulation (DBS) or repetitive transcranial magnetic stimulation (rTMS) may be employed [7, 8]. These interventions aim to modulate the neural circuits implicated in OCD, potentially offering alternatives for patients with severe, treatment-resistant cases.

The definite etiology of obsessive-compulsive disorder is a subject of ongoing debate, with various hypotheses being investigated. A comprehensive understanding of these underlying mechanisms is pivotal for the development of novel therapeutic strategies, especially those more effective for patients who do not respond to standard treatment. The etiology includes the genetic component that affects serotonergic, dopaminergic, and glutamatergic systems, as well as environmental influences such as trauma. Psychological theories focus on cognitive dysfunctions, while comparative research indicates the involvement of the neurobiological circuits in the expression of the symptoms and familial risk [9]. A growing area of research centers on the gut-brain axis (GBA) – the bidirectional communication between the gut and the brain, and its significant implications for the development and manifestation of mental disorders. The gut microbiome – the trillions of microbes within the human gastrointestinal tract – can influence central nervous system activity, holding particular significance for mental health [10]. This review will examine the existing literature concerning the interplay between the gut microbiome and obsessive-compulsive disorder.

2. Mechanisms of Gut-Brain Communication

The pathways of gut-brain communication involve neural, endocrine, immunological and metabolic mechanisms. Neural connections provide rapid, direct channels of communication within the GBA. The vagus nerve plays a fundamental role, transmitting afferent signals from the gut to the central nervous system (CNS) and efferent signals from the CNS to the gut. This

cranial nerve directly innervates visceral organs, including the intestines, and allows the brain to monitor gut physiological states and, conversely, enables gut-derived signals to influence brain function and behavior. For example, metabolites produced by microbes, and inflammatory signals from the gut lumen, can activate vagal afferents, subsequently impacting mood, anxiety, and stress responses [11].

Enteric nervous system (ENS) is another part of the neurological gut-brain communication system. The ENS is a semi-autonomous neuronal network embedded within the gut wall that controls gastrointestinal functions, including motility, secretion, and local blood flow. The ENS communicates extensively with the CNS, and its proper functioning can be profoundly influenced by the gut microbiome, which can modulate neurotransmitter production (e.g., serotonin) within the gut, having either a local effect on ENS neurons or being systemically transported [12].

The endocrine system's role in gut-brain communication is primarily represented by the hypothalamic-pituitary-adrenal (HPA) axis – the body's central stress response system. Dysbiosis can lead to increased gut permeability, allowing microbial products to enter systemic circulation, resulting in low-grade inflammation. Systemic inflammation can activate the HPA axis, leading to increased levels of cortisol, which, when chronic, are linked to various neuropsychiatric disorders, including depression and anxiety (14). A balanced microbiome may stabilize the activity of the HPA axis, contributing to better stress resilience.

The gut contains a significant portion of the body's immune system, and the gut microbiota significantly impacts immune function, with direct consequences for neuroinflammation and mental health. Gut bacteria, particularly through the fermentation of dietary fibers, produce short-chain fatty acids (SCFAs) such as butyrate, propionate, and acetate. These SCFAs are vital energy sources for colonocytes, but also have strong immunomodulatory effects. Butyrate, for example, strengthens intestinal barrier integrity, reducing systemic inflammation, and can cross the blood-brain barrier to exert direct neuroprotective and anti-inflammatory effects within the CNS [15]. Dysbiosis may result in a compromised gut barrier ("leaky gut"), with bacterial components like lipopolysaccharides (LPS) gaining entry into the bloodstream. This triggers a systemic inflammatory response, leading to the release of pro-inflammatory cytokines (e.g., IL-1β, TNF-α, IL-6) that can cross the blood-brain barrier and induce neuroinflammation. Chronic neuroinflammation is increasingly implicated in the pathophysiology of a wide range of mental disorders, including depression, anxiety, and potentially OCD [16, 17]. Research has

demonstrated that chronic gastrointestinal inflammation can induce anxiety-like behavior and alter CNS biochemistry in animal models [18].

Another way gut microbes influence the brain function is through their ability to directly produce or modulate the bioavailability of critical neurotransmitters such as serotonin, gamma-aminobutyric acid (GABA), and dopamine. For example, a substantial percentage of the body's serotonin is produced in the gut by enterochromaffin cells, a process that can be influenced by specific microbial species [13, 19]. Likewise, certain gut bacteria can synthesize GABA, the key inhibitory neurotransmitter in the CNS, or produce metabolites that influence GABAergic signaling [20]. Besides the direct synthesis of neurotransmitters, the microbiome also modulates the metabolism of host-derived compounds (e.g., tryptophan, the precursor of serotonin and kynurenine). Dysbiosis can alter the kynurenine pathway, shifting metabolism toward neurotoxic metabolites, which have been linked to depressive and anxiety-like behaviors [21]. These metabolic interactions highlight the profound indirect influence of the gut microbiome on neurochemistry and, consequently, on mental health.

3. Evidence for Gut Microbiome Dysbiosis in OCD Pathophysiology

The connection between the gut microbiome's makeup and the manifestation of mental health disorders like OCD is a subject of ongoing research.

The study by Turna et al. [22] involved analyzing the stool microbiome and inflammatory markers from 43 patients. They also took into account potential confounding factors, including a history of depression and the use of antibiotics, probiotics, and SSRIs. Twenty-one OCD patients, who were depression-free and not on any medication, were recruited for the study. They had age- and gender-matched control subjects. The findings revealed that the OCD group had lower species richness and evenness of the gut microbiome and the gut microbiome was less diverse. They had a significantly lower relative abundance of three genera: Oscillospira, Odoribacter, and Anaerostipes. The lower count of these three bacteria could mean less production of butyrate, a beneficial short-chain fatty acid. This suggests that the host may not be receiving the health advantages associated with adequate butyrate levels.

In a study by Domènech et al. [23] analyzing the stool microbiome of 32 OCD patients and 32 age- and gender-matched controls, researchers also observed notable variations. The OCD group's stool samples showed a tendency towards lower bacterial α-diversity – total number of bacterial species, indicating less variety in their gut bacteria. Specifically, there was an increase

in the relative abundance of the Rikenellaceae family, particularly the genus Alistipes, which has been linked to gut inflammation in mice [24]. Conversely, the OCD patients exhibited a lower relative abundance of Prevotellaceae and two genera within the Lachnospiraceae: Agathobacer and Coprococcus. Notably, Coprococcus is associated with DOPAC synthesis. Despite these findings, the study did not find a significant difference in the Bacteroidetes to Firmicutes ratio between the OCD and control groups.

Furthermore, Quagliariello et al.[25] conducted a study on the gut microbiome composition of 30 individuals with pediatric acute-onset neuropsychiatric syndrome (PANS) and pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections syndrome (PANDAS), neurological disorders characterized by the sudden onset of tics, obsessive-compulsive disorder, and other behavioral symptoms in children. To mitigate age-related bias, the researchers divided patients and controls into two age groups: 4-8 years old and over 9 years old, which revealed distinct microbial patterns. Children in the younger cohort exhibited a significant increase in the abundance of Bacteroidetes, specifically Bacteroides, Odoribacter, and Oscillospira as potential microbial biomarkers. Conversely, they showed a reduction in Firmicutes. The metabolic profile of their microbiota indicated an activation of pathways associated with immune response and inflammation, alongside a deficiency in pathways crucial for brain function, such as those involved in dopamine, tyrosine metabolism, and short-chain fatty acids (SCFAs). The gut microbiome of older patients was more challenging as it displayed a less uniform bacterial profile. However, this group was characterized by a greater abundance of Peptostreptococcaceae and Erysipelotrichaceae, with reduced levels of Rikenellaceae and Barnesiellacea. According to this study, streptococcal infections can alter gut bacteria, creating a pro-inflammatory environment by promoting the growth of specific bacteria linked to gut inflammation and a heightened immune response.

In a study by Jung et al. [26], researchers examined the role of gut microbiota changes in the development of compulsive checking and locomotor sensitization in rats, behaviors induced by chronic quinpirole injections. Rats were divided into two groups, receiving either weekly quinpirole or saline injections over several weeks, with their behavior recorded and fecal samples collected after each session for 16S rRNA gene profiling. The findings revealed that the onset of both locomotor sensitization and compulsive checking was accompanied by alterations in several bacterial communities, primarily those belonging to the order Clostridiales (within the phylum Firmicutes), with significant changes observed in the Lachnospiraceae and

Ruminococcaceae families. While the study provides compelling evidence of a link between behavioral changes and shifts in the gut microbiota, it does not establish a clear cause-effect relationship. The authors note that the observed microbial changes could be a result of drug injections or other influencing factors, such as stress.

In a study by Scheepers et al. [27], researchers investigated the gut microbiota of deer mice to determine whether a naturally occurring obsessive-compulsive-like behavior, known as large nest building (LNB), was associated with changes in their gut bacteria. The gut microbiomes of LNB mice were found to be distinctly different in overall composition compared to control mice with normal nest-building behavior. While statistical corrections for multiple comparisons showed no significant differences in individual genera, the analysis did reveal some notable patterns. The normal-phenotype mice tended to have higher levels of Prevotella and Anaeroplasma, considered anti-inflammatory species [28, 29], whereas the OCD-like phenotype in the LNB mice was associated with higher levels of Desulfovermiculus, Aestuariispira, Peptococcus, and Holdemanella – all pro-inflammatory species [27, 30, 31].

4. Therapeutic Implications: Modulating the Gut Microbiome for OCD

Given the potential role of a dysfunctional gut microbiome in psychiatric disorders, probiotics present themselves as a possible alternative treatment for these conditions. Research has already demonstrated their ability to reduce depression-like symptoms, improve gut function, and regulate immune response [32], as well as alleviate symptoms of autism spectrum disorder symptoms and

improve mood through an anti-inflammatory effect [33]. Nonetheless, we currently have limited data regarding the impact of probiotics on individuals with OCD.

In a series of experiments, Kantak et al. [34] investigated the impact of Lactobacillus rhamnosus GG (LGG) probiotic pretreatment on RU 24969-induced OCD-like behaviors in BALB/cJ mice. The initial experiment demonstrated that both two and four weeks of daily oral LGG administration attenuated the induction of OCD-like behaviors, including increased perseverative open-field locomotion, stereotypic turning, and marble burying. Building on these findings, a second experiment further confirmed the efficacy of a two-week LGG pretreatment, showing it to be comparable to a four-week fluoxetine regimen in blocking the induction of these OCD-like behaviors.

In an animal model of OCD, a study by Sanikhani et al. [35] investigated the therapeutic effects of the probiotic Lactobacillus casei Shirota. Researchers induced OCD-like symptoms in rats through chronic injection of the dopamine agonist quinpirole hydrochloride. These rats were then treated with either the probiotic, the antidepressant fluoxetine, or a combination of both, while control groups received only quinpirole or saline. The results showed that all three treatment regimens significantly improved the observed OCD-like behaviors. This behavioral improvement was mirrored by changes in gene expression in the orbitofrontal cortex. The quinpirole-induced model was characterized by a decrease in the expression of the Bdnf gene and an increase in the Htr2a gene. Crucially, treatment with either the probiotic or fluoxetine reversed this pattern, increasing Bdnf and decreasing Htr2a expression. These findings suggest that L. casei Shirota is effective in this rat model of OCD, possibly because of the modulation of serotonin-related gene expression.

In a case report by Kobliner et al. [36], a boy with comorbid autism spectrum disorder, obsessive-compulsive disorder (OCD), and self-injurious behavior (SIB) was successfully treated with the probiotic yeast Saccharomyces boulardii. The report notes that gastrointestinal dysfunction and OCD are common in autism, potentially sharing a common cause related to a disturbed gut microbiome. Alterations in the gut's microbial balance can influence neuroinflammation, which is linked to mood disorders, abdominal pain, and SIB. In this case, the probiotic yeast, which is known to support a healthy microbiome and enhance immune function, was administered to the patient. Following treatment, the boy's OCD and SIB symptoms were significantly reduced, suggesting that targeting the gut microbiome may be a viable therapeutic approach for managing these complex conditions.

Another therapeutic intervention that has been identified as promising in OCD treatment is fecal microbiota transplantation (FMT) - the transfer of fecal matter from a healthy donor to a patient [37]. In a study focused on analyzing severity of anxiety, depression and obsession in inflammatory bowel disease (IBS) patients, Kilinçarslan et al. (2020) reported that, following FMT, individuals experienced a reduction in the severity of various psychological symptoms, including obsessive tendencies [38].

5. Future directions and challenges

Research into the role of the gut microbiota in OCD is an emerging field, yet it is currently constrained by several key limitations. A major challenge in human studies is the use of small sample sizes, which often limits the statistical power and generalizability of findings.

Furthermore, these studies are complicated by numerous confounding factors that are difficult to control, including variations in diet, lifestyle, and the use of medications such as SSRIs and antibiotics, all of which can independently alter the gut microbiome. The high rate of psychiatric and physical comorbidities in OCD – particularly with anxiety disorders, depression, and irritable bowel syndrome – presents another significant hurdle, as alterations in the gut microbiome associated with these co-occurring conditions can obscure specific findings related to OCD. While animal studies provide valuable mechanistic insights, a notable translational gap exists: the animal models of OCD-like behaviors, while useful, may not fully replicate the complex cognitive and behavioral aspects of the human disorder. Moving forward, larger, well-controlled human studies are needed to better delineate the specific microbial profiles of OCD patients from those of common comorbidities, ultimately advancing our understanding and therapeutic potential in this area.

Looking ahead, the investigation into the gut microbiome's role in obsessive-compulsive disorder faces both exciting opportunities and significant challenges. While fecal microbiota transplantation has demonstrated encouraging results in various other diseases by restoring microbial balance [39-41], there is currently limited research on its application or efficacy in OCD patients. This represents a critical gap that future studies should address. Furthermore, dietary interventions hold considerable potential – specific dietary patterns are known to profoundly shape the gut microbiome, suggesting they could exert a direct influence on OCD symptomatology. Understanding how targeted nutritional approaches might modulate the gut environment to alleviate OCD symptoms is another vital area for future exploration. Overcoming the existing limitations, such as small sample sizes and confounding factors in human studies, will be crucial for fully harnessing these therapeutic possibilities and translate promising preclinical findings into effective clinical strategies for OCD.

6. Conclusions

This review examines the accumulating evidence that highlights a significant interplay between the gut microbiome, the intricate gut-brain axis, and the underlying pathophysiology of obsessive-compulsive disorder (OCD). The established bidirectional communication pathways – encompassing neural, endocrine, immune, and metabolic mechanisms – underscore how alterations in microbial composition can influence central nervous system function and contribute to the manifestation of OCD symptomatology. While this field of research is still in

its formative stages, the potential of microbiome-targeted interventions, particularly to cases of treatment-resistant OCD, is becoming increasingly evident. Emerging approaches, such as the use of probiotics and the largely unexplored avenues of fecal microbiota transplantation and specific dietary modifications, present promising new therapeutic frontiers. However, to effectively translate these preclinical insights into tangible clinical benefits, it is imperative to enhance public and professional awareness of OCD and its potential biological underpinnings. Furthermore, there is a pressing need for larger, meticulously controlled human studies to precisely delineate specific microbial profiles and mechanistic pathways that may trigger or exacerbate OCD development. Such focused research is crucial to advancing our understanding and ultimately developing more effective, personalized treatment strategies that can significantly improve patient outcomes and quality of life.

Disclosure

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