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Gut-Brain Axis Dynamics: Elucidating Bidirectional Interactions Between Gut Microbiota Composition and Autism Spectrum Disorder Pathogenesis

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Abstract. Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by impaired social communication, restricted interests, and repetitive stereotyped behaviors. Accumulating clinical evidence has demonstrated associations between gut microbiota dysbiosis and various neurological disorders, particularly neurodevelopmental conditions. This study investigates the gut microbiota-ASD correlation through the lens of the gut-brain axis. By synthesizing findings from prior metagenomic sequencing analyses of fecal samples from human cohorts and murine models, we identified distinct divergence in gut microbial composition between ASD individuals and neurotypical controls. Controlled intervention trials involving supplementation with ASD-associated microbial strains (e.g., Lactobacillus reuteri) in both ASD patients and rodent models revealed significant improvement in social behavioral deficits following microbial modulation. Through a comprehensive review of recent literature, we established that gut microbiota alterations constitute a pathological mechanism in ASD etiology, with targeted microbial supplementation demonstrating therapeutic potential for ameliorating core social dysfunction. These findings support a bidirectional relationship between gut microbial ecosystems and neurodevelopment: while microbial dysbiosis serves as a hallmark of ASD pathophysiology, intentional microbiota remodeling conversely exerts beneficial neuromodulatory effects on clinical manifestations.

Keywords: Autism spectrum disorders, Gut-brain axis, Gut microbiota, Neurodevelopment, Neuropsychiatric diseases.

1. Introduction

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized primarily by impairments in social communication, restricted interests or activity patterns, and repetitive and stereotyped behaviors. This condition exerts significant global impact, and no definitive cure currently exists for its core symptoms. The influence of the gut microbiota on the central nervous system (CNS) through the gut-brain axis and its role in the development of autism spectrum disorder (ASD) have emerged as a focal point in current research. Clinical studies have demonstrated

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associations between gut microbial composition and various neurological conditions, with particularly notable correlations observed in neurodevelopmental disorders.

The current research landscape faces multiple limitations: insufficient sample sizes of individuals with autism spectrum disorder (ASD), restricted age ranges, and inadequate clinical trials collectively constrain the generalizability of findings; the predominant absence of longitudinal follow-up studies impedes assessment of therapeutic long-term effects; existing treatment evaluations have predominantly focused on restricted outcome measures; and the complexity of ASD renders singular mechanistic explanations insufficient to account for its multifactorial etiology.

The aim of this review is to explore the association between the gut microbiome and autism spectrum disorders (ASD) and its underlying mechanisms from the perspective of the gut microbiome, considering recent research findings in related fields. The article systematically summarizes the current state-of-the-art on the gut microbiome in the pathological mechanisms and therapeutic applications of ASD, aiming to provide a theoretical basis for future research directions and a reference framework for clinical practice.

2. Neural implications for gut-brain axis

2.1. Mechanism of the gut-brain axis

The gut-brain axis constitutes a bidirectional communication network between the gastrointestinal tract and the central nervous system, encompassing neural, endocrine, immune, and metabolic pathways [1]. The brain regulates gut function through the hypothalamic-pituitary-adrenal axis and the autonomic nervous system; in turn, the gut influences brain function through microbial-derived metabolites and products, neuroactive substances, and gut hormones via the enteric nervous system, vagus nerve, circulatory system, or immune system [2].

2.1.1. Neural pathways

The brain and gut communicate directly via the autonomic nervous system and vagus nerve, thereby influencing the expression of various receptors in the central nervous system, such as GABA receptors [3] .

Gut microbes influence gut motility and sensation by modulating the activity of the autonomic nervous system (ENS), which in turn affects brain function. For example, gut microbes produce metabolites that activate neurons in the ENS and regulate gut motility [4].

As the longest parasympathetic nerve connecting the enteric nervous system to the brainstem, the vagus nerve detects diverse signals in the gut, transmits them, and subsequently modulates the central nervous system [5]. For example, Lactobacillus reuteri increases oxytocin levels by stimulating the vagus nerve and can modulate GABA receptor expression in the brain, which reduces anxiety and depression-like behaviors and improves social impairment in a mouse model of autism spectrum disorder [5, 6].

2.1.2. Endocrine pathways

The gut microbiota can modulate the activity of the hypothalamic-pituitary-adrenal (HPA) axis by triggering hypothalamic release of corticotropin-releasing hormone (CRH), which subsequently activates the HPA axis to secrete glucocorticoids [3,7].

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Simultaneously, enteroendocrine cells sense microbial metabolites to regulate hormone secretion, including insulin and cortisol, which participate in gut-brain communication and exert effects on the central nervous system [7].

2.1.3. Chemical signaling pathways

Gut microbiotas produce diverse chemical substances—such as neurotransmitters, short-chain fatty acids (SCFAs), tryptophan metabolites, and microbial derivatives—that influence cerebral function.

Neurotransmitters like γ-aminobutyric acid (GABA) and serotonin (5-HT) serve as critical mediators of neuronal communication, modulating host mood, cognition, and immune responses, with direct relevance to depression, anxiety, and affective disorders [4].

SCFAs, as primary metabolic byproducts of gut microbiota, impact brain function through multiple mechanisms: regulating neurotransmitter synthesis, modulating neuroinflammation, and enhancing intestinal barrier integrity. These compounds play pivotal roles in blood-brain barrier (BBB) regulation and neuroimmune-endocrine integration [5,7] , contributing to glucose homeostasis, mucosal serotonin release, lymphocyte function, and learning and memory. It has also been shown that the integrity of the intestinal barrier is impaired in germ-free (GF) mice and that it can be restored by recolonizing SCFAs-producing bacteria [2].

Tryptophan metabolites (e.g., indole derivatives) inhibit NF-κB signaling pathway activation and NLRP3 inflammasome formation while significantly influencing neurotransmitter synthesis and functionality [4]. In addition, certain gut bacteria (e.g., some species of the genus Lactobacillus) can convert tryptophan to indole compounds, which affects cell cycle regulation and T cell differentiation [2].

Microbial derivatives such as lipopolysaccharide (LPS) released by Gram-negative bacteria can be recognized by Toll-like receptor 4 on microglia in the CNS, leading to the production and proliferation of pro-inflammatory cytokines, which are associated with neuroinflammation, microglial activation, and neuronal loss, and may lead to cognitive impairment and mood disorders. [2]

2.1.4. Immunization pathways

The gut microbiota interacts with the host's immune system and influences the development and function of immune cells. Immune system abnormalities, including elevated levels of inflammatory markers and dysfunctional immune cells, have been observed in patients with ASD [8].

Imbalances in gut microbes (e.g., leaky gut due to impaired gut barrier function) can trigger an inflammatory response that activates the immune system (e.g., Toll-like receptors, TLRs), which in turn affects brain function. Inflammatory factors (e.g., IL-6, TNF-α, etc.) can cross the blood-brain barrier and affect the synthesis and release of neurotransmitters [7]. In addition, H. pylori infection has been shown to affect the gut-brain axis through its secretions, activating the STAT3 signaling pathway, inducing neuroinflammation, and promoting related pathological changes. H. pylori-infected AGS cells showed significant inflammatory responses, including increased expression of pro-inflammatory cytokines (e.g., TNFα, IL-6, IL-1β, etc.) and chemokines (e.g., CXCL1, CXCL3, etc.). H. pylori infection can also induce oxidative stress in neuronal cells through its secretions. After treatment with the STAT3 inhibitor BP-1-102, a significant decrease in the expression of inflammatory response and AD-related markers induced by H. pylori infection was found, suggesting that STAT3 plays a key role in neuroinflammation, and AD pathology induced by H. pylori infection [9].

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Gut microbes can modulate the activity and function of immune cells, e.g., by regulating T-cell differentiation and cytokine secretion, which in turn affects the development of neuroinflammatory and neurodegenerative diseases [7]. In addition, microglia are immune cells of the central nervous system and are involved in neuroinflammation and synaptic pruning. Gut microbes regulate microglia maturation and function through metabolites such as SCFAs. For example, germ-free mice (GF mice) exhibit deficient microglia maturation, and supplementation with SCFAs restores their function [5].

2.2. Altered gut microbiome and neurodevelopmental disorders

A cohort study tracking 16,440 Swedish children, including 1,197 later diagnosed with neurodevelopmental disorders (autism spectrum disorder [ASD], attention-deficit/hyperactivity disorder [ADHD], or language disorders), revealed significant gut microbial community differences between infants subsequently diagnosed and undiagnosed peers. Specific microbial signatures were associated with elevated neurodevelopmental risks: lower abundances of beneficial bacteria (e.g., Bifidobacterium and Lactobacillus) and higher abundances of potentially pathogenic taxa such as Proteobacteria [10,11].

Research on maternal-fetal interactions demonstrated that the maternal microbiome and its metabolites may influence fetal neurodevelopment via placental pathways. Maternal immune activation (MIA) models showed that maternal microbial dysbiosis induces offspring behavioral phenotypes resembling ASD, highlighting transgenerational microbial-immune-neural crosstalk.

Furthermore, studies have demonstrated that germ-free (GF) mouse models exhibit neurodevelopmental abnormalities along with cognitive and behavioral alterations due to the absence of gut microbiota, indicating the critical role of intestinal microorganisms in normal brain development and behavioral regulation [12].

3. Gut microbiome and autism

3.1. Changes in autistic gut microbiome

Studies of fecal samples from children with autism have revealed changes in the composition of the gut microbiota in children with autism compared to healthy children, such as increased levels of Lactobacillus, Clostridium, and Desulfovibrio, and decreased levels of Bifidobacterium and decreased levels of Prevotella [8].

A study conducted metagenomic sequencing on fecal samples from 1,627 children (including children with autism spectrum disorder [ASD] and neurotypical children), analyzing the composition and functional profiles of archaea, bacteria, fungi, and viruses in the gut. In terms of composition, the study revealed that ASD children exhibited significantly reduced gut microbial diversity. Specifically, 14 archaeal species, 51 bacterial species, 7 fungal species, and 18 viral species showed significant differences in abundance between ASD and neurotypical children. Notably, certain beneficial bacteria, such as Streptococcus thermophilus, Weissella confusa, and Weissella cibaria, were markedly diminished in ASD children [13].

Another study investigating gut microbial composition in mice reported distinct differences between ASD-model and typically developing (TD) control groups. For instance, Bacteroides ovatus and Parabacteroides merdae were more prevalent in TD controls, whereas Eisenbergiela tayi was enriched in the ASD group [14].

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A study enrolled 456 participants, comprising 249 individuals with autism spectrum disorder (ASD), 106 typically developing siblings, and 101 control individuals. By analyzing the 16S rRNA gene in fecal samples, the researchers investigated gut microbial composition differences among the ASD group, sibling group, and control group. The study demonstrated that Bifidobacterium longum exhibited a negative correlation with the Childhood Autism Rating Scale (CARS), and Streptococcus salivarius showed a negative association with the Social Responsiveness Scale (SRS) in the ASD group. Additionally, cluster analysis based on microbial composition identified two distinct enterotypes (E1 and E2) within the ASD group. The E2 subgroup displayed significantly higher SRS scores and markedly lower Vineland Adaptive Behavior Scale (VABS) scores compared to other subgroups [15].

3.2. Altered gut microbiota as a pathologic mechanism

Research by Su Q et al. has demonstrated that children with autism spectrum disorder (ASD) exhibit significant reductions in the synthesis pathways of coenzyme Q7 (ubiquinol-7) and thiamine diphosphate compared to neurotypical children. These diminished metabolic pathways may be linked to ASD pathological mechanisms, such as impaired antioxidant function and disrupted neural signaling. [13]

In this study, ASD-associated bacteria (e.g., Lactobacillus reuteri) were introduced into an ASD model based on C57BL/6J mice, and the behavioral performance of each group was evaluated with the aid of a three-box socialization test, a reciprocal social interaction test, an open-field test, and a burying-feeding test.

Shank3B-/- mice were bred from Shank3B+/- mice and compared to wild-type (WT) mice from the same litter, and this model was used to investigate the mechanisms associated with genetically induced ASD. It was found that this mouse model has social behavioral deficits, and its intestinal flora composition was altered; especially the level of Lactobacillus reuteri showed a decreasing trend. The experimental results showed that Shank3B-/- mice showed significant changes in the composition of the gut microbial community, especially in the levels of Lactobacillus reuteri, and that supplementation with Lactobacillus reuteri was effective in restoring social behavior in these mice. Behavioral performance.

BTBR mice, an idiopathic model of ASD, showed abnormal social behavior, with significant differences in gut flora structure and reduced Lactobacillus reuteri levels compared to C57BL/6J mice. The BTBR mice were also found to have defective gut microbiota composition and social behavior, and supplementation with Lactobacillus reuteri also improved their social behavior.

Gastrofluid-free (GF) mice are C57BL/6J mice bred in an aseptic environment and were used primarily to study the independent effects of Lactobacillus reuteri on social behavior. It was shown that Lactobacillus reuteri supplementation alone was also able to restore social behavior in germ-free (GF) mice. Using controlled experiments, it was shown that the gut microbial composition of ASD mouse models is altered compared to normal mice and that supplementation with L. reuteri ameliorates social behavioral deficits in a variety of ASD mouse models and that its effects are not dependent on other gut microbes. [16]

However, experimental studies have revealed that while L. reuteri treatment ameliorates social behavioral deficits in children with autism spectrum disorder (ASD), it exhibits no significant impact on other core ASD symptoms, such as repetitive behaviors. Furthermore, these therapeutic benefits may be restricted to specific strains (e.g., L. reuteri ATCC PTA 6475), rather than constituting a universal effect across all L. reuteri strains [17].

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4. Limitations and future prospects

4.1. Limitations

The most enlightening finding lies in the multifaceted mechanisms of the gut-brain axis. Specifically, alterations in gut microbiota composition have been identified as one of the pathological mechanisms underlying autism spectrum disorder (ASD), while supplementation with autism-associated microbes has demonstrated potential in ameliorating social behavioral deficits in ASD.

Unlike numerous previous studies that primarily focused on unidirectional relationships between gut microbiota alterations and ASD pathogenesis, this research highlights a bidirectional interaction between gut microbial composition and neurodevelopmental processes. The study demonstrates that while gut microbiota dysbiosis represents a characteristic feature of ASD patients, intentional modulation of microbial composition can conversely induce beneficial responses in clinical manifestations of ASD.

However, inconsistencies were observed among the study findings. Although microbial supplementation showed efficacy in improving overall clinical presentations of ASD, the therapeutic effects appeared behavior-specific, with significant improvements restricted to social deficits while showing limited impact on other symptom domains. Consequently, given the current inability to establish correlations between distinct clinical manifestations in ASD patients, the feasibility of this approach remains inconclusive. These findings suggest the need for more nuanced investigations into phenotype-specific microbial interventions and their underlying mechanisms.

4.2. Future directions

Future research directions should focus on further exploring the relationship between gut microbes and autism spectrum disorders (ASD) and provide a more nuanced and reliable scientific basis for potential treatment strategies. Despite the remarkable results in mouse models, these findings need to be validated in humans. Future studies need to conduct clinical trials in ASD patients to evaluate the safety and efficacy of specific microorganisms such as Lactobacillus reuteri (L. reuteri) or its metabolites in improving ASD symptoms. There is also a demand to study the long-term effects of microbial interventions, including their continued impact on patient behavior, cognitive functioning, and social adjustment.

While research has revealed some of the mechanisms by which gut microbes influence ASD-related behaviors, further studies are needed to examine how brain function is affected through the vagus nerve or other pathways and to explore undiscovered new pathways. Future research needs to further parse out how specific microbes, such as 5AV- and taurine-producing bacteria, modulate neurotransmitter levels and neuronal excitability, and how these changes translate into behavioral improvements.

Developing microbiome-based diagnostic tools can identify which patients with ASD may benefit from microbial interventions. This involves a detailed analysis of the patient's gut microbiome to determine the presence of specific microbial signatures associated with ASD. Individualized microbial treatment plans are developed based on a patient's specific microbiome profile and behavioral symptoms. For example, some patients may require supplementation with specific probiotics or metabolites, while others may require more complex microbial interventions.

Future research needs to further integrate microbiological, neuroscientific, and behavioral approaches to more fully understand how gut microbes affect brain function and behavior. Utilizing

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gene editing technologies (e.g., CRISPR), single-cell sequencing, and neuroimaging, the mechanisms of microbe-host interactions can be more precisely studied in the future.

5. Conclusion

Alterations in gut microbiota composition constitute one of the pathological mechanisms underlying autism spectrum disorder (ASD), and supplementation with ASD-associated microbes has been shown to ameliorate social behavioral deficits in ASD.

A bidirectional relationship exists between gut microbiota and neurodevelopmental processes, where gut microbial dysbiosis represents a hallmark feature of ASD patients, while intentional modulation of microbial composition can conversely elicit beneficial responses in clinical manifestations of ASD.

However, limited sample sizes have restricted the identification of ASD-associated gut microbial taxa. Furthermore, the therapeutic effects of specific microbial supplementation appear behavior-specific, demonstrating significant improvements exclusively in social deficits without observable changes in other symptom domains. Therefore, the feasibility of ASD-associated microbial supplementation remains inconclusive.

Future studies should prioritize the identification of potential ASD-related gut microbes, investigate the effects of supplementing other ASD-associated gut microbes on additional clinical manifestations, and explore potential synergistic effects through combinatorial microbial interventions.

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