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REVIEW

Fecal Microbiota Transplantation for Attention-Deficit/Hyperactivity Disorder: Mechanisms, Evidence, and Future Directions

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Abstract: Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental condition characterized by inattention, hyperactivity, and impulsivity. While pharmacological and behavioral therapies remain first-line treatments, their limitations in efficacy, tolerability, and long-term adherence underscore the need for innovative interventions. Growing evidence highlights the role of the microbiota–gut–brain axis (MGBA) in ADHD pathophysiology, particularly involving immune dysregulation, neurotransmitter imbalance, metabolic disruption, and epigenetic alterations. Fecal microbiota transplantation (FMT), as a microbiota-based intervention, has shown promise in restoring MGBA homeostasis and modulating neural function through multiple mechanisms. This review summarizes current preclinical and clinical research on FMT in ADHD, covering its effects on neuroinflammation, neurotransmitter pathways, vagus nerve and HPA axis signaling, and epigenetic reprogramming. Although preclinical models and early human data indicate potential behavioral benefits and mechanistic plausibility, methodological heterogeneity, limited sample sizes, and incomplete mechanistic validation pose significant challenges. Future research should prioritize protocol standardization, randomized controlled trials, biomarker discovery, and ethical regulation to facilitate the safe and effective clinical translation of FMT in ADHD treatment.

Keywords: attention-deficit hyperactivity disorder, ADHD, fecal microbiota transplantation, FMT, microbiota–gut–brain axis, MGBA, gut microbiota dysbiosis, neuroinflammation

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common neurodevelopmental disorders, primarily characterized by symptoms of inattention, hyperactivity, and impulsivity.[1,2] ADHD typically emerges in early childhood and frequently persists beyond childhood. Persistence estimates vary by cohort and by definition: population-based studies using stricter adult thresholds often report approximately 35–65%, whereas a recent prospective clinical cohort reported 87.5% persistence into adulthood.[3,4] Some adults experience reduced functional impact through coping and compensatory strategies despite residual symptoms, rather than complete resolution. ADHD is associated with long-term effects on academic performance, interpersonal relationships, social functioning, and mental health.[5,6] Globally, ADHD affects an estimated 5–8% of children and adolescents, while the adult prevalence ranges from 2–5% in some countries.[7–9] With increasing public awareness, refined diagnostic criteria, and evolving environmental factors, the global prevalence of ADHD has shown a rising trend.[9] Furthermore, ADHD is frequently comorbid with learning disorders, autism spectrum disorder (ASD), anxiety, and conduct disorders, adding to its clinical complexity.[10]

The etiology of ADHD is multifactorial, involving genetic susceptibility, environmental exposures, and neurobiological abnormalities.[11] Twin and family studies indicate a heritability estimate of 70–80%.[12] Neuroimaging and molecular studies have revealed structural and functional abnormalities in brain regions associated with attention and executive function, such as the prefrontal cortex, basal ganglia, and cerebellum. Dysfunction of neurotransmitter systems,

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particularly dopaminergic and noradrenergic pathways, is believed to play a central role in ADHD pathophysiology.[1,13,14] However, genetic factors alone cannot fully account for the phenotypic heterogeneity or the increasing prevalence of the disorder, prompting growing interest in early-life environmental influences such as maternal stress, premature birth, nutritional status, and gastrointestinal health.[15,16] Accumulating evidence suggests that the gut microbiota may influence neurodevelopment and potentially modulate the risk of developing ADHD.

Current treatment strategies for ADHD primarily rely on pharmacological and behavioral interventions. Stimulants such as methylphenidate and amphetamine derivatives remain first-line medications. However, 20–30% of patients show suboptimal response or experience adverse effects, including appetite suppression, sleep disturbances, and mood fluctuations, which hinder long-term adherence.[17] Non-stimulant options such as atomoxetine and α2 adrenergic agonists provide alternatives, yet their efficacy remains limited.[18] Behavioral therapies, although effective, are often constrained by limited accessibility and patient compliance.[19] Consequently, there is a growing need for novel treatment approaches with stronger mechanistic specificity and fewer side effects, particularly those targeting the underlying neurodevelopmental and behavioral pathways.

In recent years, the microbiota–gut–brain axis (MGBA) has emerged as a critical bidirectional communication network between the central nervous system and the gastrointestinal tract, involving immune, neural, endocrine, and metabolic pathways. Studies have demonstrated that the gut microbiota can modulate cognition, emotion, and behavior by influencing immune responses, neurotransmitter metabolism (eg, dopamine, serotonin, γ-aminobutyric acid [GABA]), and hypothalamic–pituitary–adrenal (HPA) axis activity.[20,21] In animal models, gut dysbiosis has been shown to induce hyperactivity, anxiety-like behaviors, and cognitive impairments.[22] Human studies also suggest that children with ADHD have distinct gut microbial compositions compared to healthy controls, typically characterized by reduced abundance of beneficial short-chain fatty acid (SCFA)-producing bacteria such as *Faecalibacterium*, and increased levels of potential pro-inflammatory taxa such as *Enterococcus*.[15,23] Although some heterogeneity exists, the overall trend supports a link between ADHD and gut microbiota imbalance.

Fecal microbiota transplantation (FMT), a therapeutic approach that introduces gut microbiota from a healthy donor into the gastrointestinal tract of a patient, has shown significant efficacy in treating *Clostridioides difficile* infection, inflammatory bowel diseases, and ASD.[24–26] In the context of ASD, an open-label study reported long-term improvements in both gastrointestinal and core behavioral symptoms following FMT, with benefits sustained for up to two years.[27] Given the overlap in clinical features and pathophysiological mechanisms between ASD and ADHD, this provides a theoretical and empirical basis for exploring FMT as a potential intervention for ADHD. Preliminary findings from animal models suggest that FMT may alleviate core behavioral symptoms, including hyperactivity and inattention deficits by reshaping gut microbial communities, reducing inflammation, and modulating neurotransmitter expression.[28] Additionally, a case report described symptom improvement and increased microbial diversity following FMT in an adult with treatment-resistant ADHD,[29] providing preliminary clinical evidence supporting its feasibility and therapeutic promise.

Given the growing recognition of MGBA dysfunction in ADHD pathophysiology and the neuroregulatory potential of FMT as a microbiota-modulating strategy, a systematic evaluation of current evidence is warranted. This review endeavors to: (1) elucidate the underlying mechanisms linking MGBA and ADHD; (2) summarize the potential biological pathways through which FMT may exert therapeutic effects, such as immune modulation, neurotransmitter regulation, metabolic control, and neural circuit modulation; (3) assess existing preclinical and clinical research on FMT in the context of ADHD; and (4) discuss current challenges and future directions in developing FMT as an adjunctive treatment strategy for ADHD. By integrating multidisciplinary evidence and mechanistic insights, this review seeks to deepen scientific understanding of microbiota-targeted interventions in ADHD and inform future efforts toward clinical translation and precision therapy.

The Microbiota–Gut–Brain Axis and Its Relationship with ADHD

Overview of the Microbiota–Gut–Brain Axis (MGBA)

The microbiota–gut–brain axis (MGBA) refers to a complex bidirectional communication network between the gut-resident microbiota and the central nervous system (CNS).[30,31] This axis involves multiple interconnected signaling pathways: (1) neural routes (eg, the vagus nerve and enteric nervous system); (2) endocrine signaling (hormones and

neurotransmitters); (3) immune modulation (pro- and anti-inflammatory cytokines); (4) microbial metabolites such as short-chain fatty acids (SCFAs) and indole derivatives. Gut microbes play critical roles in maintaining intestinal barrier integrity, shaping immune system development, and synthesizing neuroactive substances. They also regulate brain function via interactions with the vagus nerve and the hypothalamic–pituitary–adrenal (HPA) axis.[30,32] Conversely, the brain influences gut motility, secretion, and microbial composition through stress responses and autonomic signaling,[33] thereby forming a dynamic feedback loop.

Homeostasis of the MGBA is essential for normal neurodevelopment. Recent evidence has linked MGBA dysfunction to several neuropsychiatric conditions, including autism spectrum disorder (ASD), anxiety, depression, and ADHD.[30,34] In patients with ADHD, microbial dysbiosis, altered metabolite profiles, and elevated systemic inflammation have been reported. These findings suggest that MGBA disruption may play a key role in ADHD pathophysiology and provide a theoretical basis for microbiota-targeted interventions.[35]

Gut Microbial Characteristics in Patients with ADHD

Clinical and metagenomic studies have revealed significant differences in gut microbial composition between individuals with ADHD and healthy controls.[36] Human data further demonstrate reduced gut microbial α-diversity in ADHD, with β-diversity differences or trends also observed, indicating impaired microbial homeostasis.[36,37] At the genus level, commensal SCFA-producing bacteria, such as *Faecalibacterium* prausnitzii, *Lactobacillus*, and *Bifidobacterium*, are significantly reduced in patients with ADHD. These bacteria contribute to anti-inflammatory regulation, maintenance of mucosal barrier integrity, and neurotransmitter metabolism.[38]

In contrast, some potentially pro-inflammatory bacteria, including *Enterococcus* and *Odoribacter*, are more abundant in ADHD populations. These taxa may promote systemic inflammation and interfere with neuroregulatory processes.[15,38] An imbalanced *Bacteroides*/*Prevotella* ratio has also been observed, which may affect carbohydrate metabolism and mucosal immune stability.[15] Yang et al measured SCFAs in *plasma* and reported lower levels in ADHD.[39] Consistently, a human *fecal* study found significant reductions in acetic, propionic, isobutyric, isovaleric, and valeric acids in ADHD, whereas *butyrate was not consistently decreased*. Lower SCFA concentrations may compromise the blood–brain barrier, promote chronic inflammation, and disrupt neurotransmitter synthesis.[30,37]

Although heterogeneity exists across studies in terms of sample sources and analytical methods, gut microbiota dysbiosis in ADHD is increasingly recognized as a biologically significant feature.

Functional Implications of MGBA Dysregulation: Neurobehavioral Disruption Through Multi-Mechanistic Interactions

The observed microbial imbalances in individuals with ADHD may disrupt MGBA homeostasis through multiple biological mechanisms. These disruptions ultimately affect central nervous system function and contribute to behavioral and cognitive abnormalities. This section outlines the potential pathological pathways across three dimensions: inflammatory responses, neuroregulation, and metabolic signaling.

Immune Activation and Neuroinflammation

Damage to the intestinal barrier can permit the translocation of bacterial endotoxins, such as lipopolysaccharides (LPS), into systemic circulation. This process triggers immune activation and the release of pro-inflammatory cytokines (eg, IL-6, TNF-α).[40] These cytokines can cross the blood–brain barrier and activate microglia, initiating neuroinflammatory cascades that impair attention regulation and emotional stability.[41] At the same time, reductions in beneficial probiotic populations weaken anti-inflammatory capacity and increase vulnerability to inflammation, a mechanism demonstrated in ADHD animal models.[42]

Neurotransmitter and Endocrine Dysregulation

The gut microbiota modulates the synthesis and regulation of multiple neurotransmitters, including dopamine, serotonin (5-HT), and gamma-aminobutyric acid (GABA). These neurotransmitters are essential for attention and impulse control.

Depletion of probiotic strains may reduce the availability of precursors such as tryptophan and tyrosine, thereby contributing to neurochemical imbalances.[43]

In addition, microbial alterations can influence hypothalamic–pituitary–adrenal (HPA) axis function and modify cortisol secretion patterns. Dysregulated HPA axis activity, frequently observed in individuals with ADHD, has been linked to impulsivity and attentional deficits.[44] Gut microbes may also regulate the expression of brain-derived neurotrophic factor (BDNF), thereby affecting synaptic plasticity and cognitive capacity.[45]

Microbial Metabolites and Epigenetic Regulation

Microbial metabolites, particularly SCFAs (eg, butyrate, propionate) and tryptophan derivatives (eg, indoles), can cross the blood–brain barrier and participate in central signaling processes. These compounds have also been shown to exert epigenetic effects.[46] For example, SCFAs may regulate neural gene expression by modulating histone acetylation and DNA methylation, thereby influencing behavioral phenotypes.[47]

In addition, microbial-derived GABA and serotonin can signal to the brain via the vagus nerve or the enteric nervous system, contributing to the regulation of emotional arousal and impulse control.[48] Animal studies have confirmed that these gut-derived neuroactive signals may play critical roles in modulating ADHD-like behaviors.[15]

Collectively, these findings suggest that MGBA dysfunction may underlie ADHD symptoms through a multifaceted interplay involving immune activation, neurotransmitter dysregulation, endocrine disturbances, altered microbial metabolism, and epigenetic modifications. While direct causal evidence remains limited, these mechanisms provide a strong theoretical foundation for gut microbiota-based interventions, such as fecal microbiota transplantation (FMT). Future research should aim to identify key microbial taxa and functional metabolites that can serve as therapeutic targets for precision interventions in ADHD.

Mechanistic Basis of FMT in ADHD

Fecal microbiota transplantation (FMT) is a microbiota-based intervention that involves the transfer of a complete and healthy donor-derived gut microbial ecosystem into the recipient's intestine to restore microbial balance and ecological homeostasis.[49] Unlike conventional probiotic supplementation, FMT introduces a complex consortium of live symbiotic bacteria, microbial metabolites, bacteriophages, and signaling molecules, enabling multifaceted regulation of the microbiota–gut–brain axis (MGBA).[30,50] With ADHD increasingly recognized as a neurodevelopmental disorder closely associated with MGBA dysfunction, FMT has emerged as a promising neuromodulatory strategy due to its multitarget and multi-pathway regulatory potential.

This section explores four core mechanisms by which FMT may exert therapeutic effects in ADHD: (1) immune modulation and neuroinflammation attenuation, (2) rebalancing neurotransmitter metabolism, (3) regulation of neural circuit signaling pathways, and (4) epigenetic modulation of neuroplasticity. Preclinical and emerging clinical findings are summarized to support the biological plausibility of these mechanisms.

Immune Modulation and Attenuation of Neuroinflammation

Patients with ADHD often exhibit elevated levels of peripheral pro-inflammatory cytokines (eg, IL-6, TNF-α), increased microglial activation in the central nervous system, and enhanced blood–brain barrier permeability. These alterations suggest that neuroinflammation plays a crucial role in disease progression.[51,52] Disruption of intestinal barrier function (ie, leaky gut) and systemic translocation of microbial-associated molecular patterns (MAMPs), such as lipopolysaccharides (LPS), are considered key triggers of inflammatory activation.[53,54]

FMT has been shown to reduce systemic inflammation by restoring beneficial microbial taxa (eg, *Faecalibacterium prausnitzii*), strengthening epithelial barrier integrity, inducing regulatory T cell activity, and promoting anti-inflammatory cytokine production (eg, IL-10).[55,56] In animal models, FMT significantly downregulates IL-6 and NF-κB expression in brain tissue, suppresses microglial activation, and improves behavioral phenotypes related to attention and impulse control.[57] A clinical case study also demonstrated increased abundance of anti-inflammatory bacteria after FMT, coinciding with reductions in core ADHD symptoms.[29] Collectively, these findings support the role of FMT in modulating neuroimmune imbalances in ADHD via the gut–immune–brain axis.

Neurotransmitter Modulation and Systemic Rebalancing

A hallmark of ADHD neurobiology is the functional disruption of key neurotransmitter systems, including dopamine (DA), norepinephrine (NE), serotonin (5-HT), and γ-aminobutyric acid (GABA). These alterations are particularly evident in the prefrontal–striatal circuitry that governs executive function and attentional control.[58,59]

The gut microbiota contributes to neurotransmitter homeostasis by synthesizing key molecules, regulating precursor availability, modulating metabolic pathways, and influencing receptor expression. For example, *Lactobacillus* and *Bifidobacterium* strains produce GABA and 5-HT, while *Streptococcus* species contribute to dopamine precursor synthesis. In addition, amino acid metabolism (eg, tryptophan, phenylalanine) is strongly influenced by gut microbial activity.[60,61] Patients with ADHD frequently exhibit reduced levels of short-chain fatty acids (SCFAs), such as butyrate, which can negatively affect neurotransmitter synthesis, brain-derived neurotrophic factor (BDNF) expression, and synaptic plasticity.[39]

FMT restores functional microbial populations and enhances SCFA production. These changes may promote neuroprotective factors and improve cognitive function.[62] FMT has also been reported to activate GABAergic and dopaminergic pathways, normalize neurotransmitter concentrations in the brain, and restore neural transmission.[63] Probiotic-based studies further suggest that strains such as *Lactobacillus rhamnosus* GG and *Lactobacillus acidophilus* LB can improve attention and impulsivity in children with ADHD by modulating neurotransmitter synthesis.[64,65] Collectively, FMT may help reconstruct the gut–neurotransmitter–brain network in ADHD.

Neural Circuitry Modulation via the Vagus Nerve and HPA Axis

Beyond immune and metabolic interactions, the MGBA also involves direct neural communication pathways, particularly through the vagus nerve and the hypothalamic–pituitary–adrenal (HPA) axis. ADHD is frequently characterized by reduced vagal tone and hyperactivation of the HPA axis, both of which contribute to impairments in attention, emotional regulation, and stress responsiveness.[66,67]

FMT-induced microbial restoration increases the production of neuroactive molecules such as SCFAs and 5-HT. These metabolites enhance afferent vagal signaling to brain regions including the limbic system and cortex. Moreover, FMT attenuates systemic inflammation, reduces chronic LPS-driven activation of the HPA axis, lowers cortisol levels, and alleviates stress hypersensitivity.[68,69] Animal experiments have demonstrated that FMT can normalize HPA stress responses and parasympathetic activity in germ-free mice, accompanied by behavioral improvements.[70] Collectively, these findings suggest that FMT may regulate ADHD-related symptoms by reestablishing functional gut–neural–brain signaling networks.

Epigenetic Modulation and Restoration of Neuroplasticity

The susceptibility to ADHD arises from interactions between genetic and environmental factors, with epigenetic mechanisms (eg, DNA methylation, histone acetylation) serving as critical mediators.[71] Adverse environmental exposures, such as prenatal stress or toxin exposure, can lead to aberrant methylation of neurodevelopmental genes and contribute to disrupted brain function.

Gut microbes participate in one-carbon metabolism and epigenetic regulation through their metabolites. Butyrate, for example, is a natural histone deacetylase (HDAC) inhibitor that promotes transcription of genes such as BDNF and synaptic proteins.[72] Folate and vitamin B_{12}, which are microbially modulated cofactors, also regulate DNA methylation by influencing methyl donor availability and methyltransferase activity.[73] By increasing levels of these epigenetically active metabolites, FMT may reshape the epigenomic landscape of neurodevelopmental genes and promote synaptogenesis and neuronal differentiation.

Although direct evidence in ADHD remains limited, FMT has been shown to reverse aberrant DNA methylation and histone modifications in animal models of autism and depression, accompanied by improvements in behavioral phenotypes.[74] These findings provide a rationale for further exploration of FMT's epigenetic effects in ADHD.

Multimodal Integration and Therapeutic Potential

FMT offers a multifaceted, systems-level intervention targeting the core pathophysiological processes of ADHD, including: (1) amelioration of neuroinflammation and immune dysregulation; (2) restoration of neurotransmitter synthesis and signaling; (3) normalization of gut–brain circuit communication (via the vagus nerve and HPA axis); and (4) epigenetic remodeling of neurodevelopmental gene expression.

Rather than acting in isolation, these mechanisms form a complex, interdependent network, in which modulation of one pathway may potentiate the others. For instance, reduction of inflammation stabilizes neurotransmitter dynamics, and epigenetic reprogramming may facilitate synaptic reconstruction and cognitive enhancement.

Therefore, FMT may represent a promising strategy for systemically remodeling the MGBA to improve neurobehavioral outcomes in ADHD. For patients unresponsive to conventional pharmacological and behavioral treatments, FMT may provide a viable adjunctive or alternative therapy. While current evidence is largely based on preclinical models and case reports, its mechanistic plausibility is well supported. Future high-quality randomized controlled trials (RCTs) are needed to validate these mechanisms and translate them into precision therapeutic protocols.

Application of FMT in ADHD: Preclinical and Clinical Research Progress

Although fecal microbiota transplantation (FMT) has demonstrated therapeutic potential in various microbiota-related disorders, its application in attention-deficit/hyperactivity disorder (ADHD) remains in the exploratory stage. This section reviews current progress in preclinical and clinical studies, focusing on behavioral outcomes, mechanistic insights, and translational challenges, with the aim of providing theoretical support and practical guidance for future research.

Preclinical Studies: Animal Models and Mechanistic Findings

Preclinical investigations have provided foundational biological evidence supporting the application of FMT in ADHD. Commonly used animal models include maternal immune activation (MIA), antibiotic-induced dysbiosis, spontaneously hyperactive strains (eg, spontaneously hypertensive rats [SHR], Lister Hooded rats), and germ-free (GF) mice. These models recapitulate core features of ADHD, such as reduced microbial diversity (eg, lower Chao1 and Shannon indices), systemic inflammation, and neurotransmitter imbalances.[75,76]

FMT interventions in these models have been shown to reverse attentional deficits, hyperactivity, and impulsive behaviors while restoring key neurotransmitter systems, including dopamine, serotonin (5-HT), and GABA/glutamate pathways.[77] The underlying mechanisms may involve enhancement of intestinal barrier integrity (eg, upregulation of tight junction proteins, reduced circulating LPS levels) and modulation of synaptic plasticity and inflammation-related gene expression in the prefrontal cortex. Taken together, these processes suggest that FMT contributes to brain remodeling through neuroimmune and transcriptional pathways.[78,79]

Notably, Tengeler et al demonstrated a causal link between gut microbiota and brain function: transplantation of fecal microbiota from children with ADHD into GF mice resulted in white matter abnormalities, heightened anxiety-like behaviors, and altered functional connectivity compared with healthy controls.[76] Similarly, Watanangura et al observed behavioral improvements in a canine model following FMT, even in the absence of significant changes in microbial composition. This finding underscores the importance of microbial metabolic function, particularly short-chain fatty acid (SCFA) production, in neuromodulation.[77] A systematic review by Caputi et al further highlighted the potential of FMT in neurodevelopmental disorders but also emphasized the limitations of current animal studies, including model heterogeneity, short intervention durations, and insufficient mechanistic validation.[80] Collectively, these findings underscore the need for more standardized animal models and mechanistically rigorous experimental designs to validate FMT's therapeutic role in ADHD. Importantly, beyond animal studies, human investigations have also demonstrated reduced fecal SCFA concentrations in ADHD.[37] They further reveal lower α-diversity and β-diversity differences.[37] Together, these findings reinforce that dysbiosis and metabolite alterations are consistently observed across species.

Clinical Studies: Case Reports and Early Trials

Clinical research on FMT in ADHD remains at an early stage, with available data primarily derived from case reports, small-scale open-label studies, and pilot prospective trials.

Hooi et al reported a case of an adult female with both ADHD and *Clostridioides difficile* infection who experienced not only resolution of diarrhea but also improvements in attention and mood following FMT.[29] Fecal analysis showed an increase in SCFA-producing bacteria such as *Faecalibacterium prausnitzii* and an overall rise in microbial diversity, suggesting that functional remodeling may underlie behavioral benefits. Although large-scale studies are lacking, preliminary evidence indicates that FMT may influence host neurophysiology by modulating inflammatory cytokines (eg, IL-6, TNF-α) and microbial metabolites (eg, SCFAs), potentially supporting recovery of attentional networks and behavioral regulation.[81,82]

Early pediatric evidence, particularly from ASD cohorts, indicates that washed microbiota transplantation (WMT) is generally well tolerated.[28,83] ADHD-specific trials remain needed. Ongoing prospective studies incorporate multimodal assessments, including gut metabolomics, behavioral scoring, and neuroimaging, to comprehensively evaluate FMT mechanisms and individual response characteristics. This represents the first structured attempt to perform multidimensional evaluation of FMT in ADHD and may hold translational value for advancing microbiota-targeted therapies in neurodevelopmental disorders.

Summary and Outlook

Together, current animal and preliminary clinical studies suggest that FMT may improve ADHD-related behavioral symptoms and neurofunction by modulating the microbiota–gut–brain axis at multiple levels, including immune regulation, metabolic reprogramming, and neurotransmitter modulation. While the existing evidence is exploratory, the biological plausibility, mechanistic specificity, and behavioral relevance of FMT support its consideration as a potential adjunctive therapeutic approach.

Future research should prioritize mechanistic validation, standardization of FMT protocols, and individualized treatment strategies to facilitate the transition of FMT from an experimental intervention toward clinical application in ADHD management.

Limitations and Future Perspectives

As a gut microbiota–centered therapeutic approach, fecal microbiota transplantation (FMT) is emerging as a novel direction in the treatment of attention-deficit/hyperactivity disorder (ADHD). However, several challenges currently limit its clinical translation, standardization, and widespread adoption.

Key Limitations of Current Research

Methodological heterogeneity: Existing studies differ substantially in donor selection criteria, microbiota preparation protocols (eg, whole microbiota vs washed microbiota), delivery routes (oral capsules, enemas, colonoscopy), and dosing frequency. Such methodological inconsistencies hinder cross-study comparability and limit the ability to derive generalizable conclusions.

Weak study design: Most available evidence is derived from case reports or open-label studies lacking placebo controls, randomization, or long-term follow-up. As a result, treatment effects may be overestimated, and the overall evidence quality remains low.

Unclear donor effects and inter-individual variability: There is no consensus on the definition of a so-called *super-donor*. Patient-specific factors, including baseline microbiota composition, immune status, and dietary habits, can significantly influence therapeutic outcomes and contribute to inconsistent efficacy.

Incomplete mechanistic validation: Many proposed mechanisms of FMT are inferred from animal or in vitro studies. Direct causal links in humans (for example, those connecting microbial shifts to metabolic remodeling and behavioral improvement) remain largely unestablished.

Ethical and regulatory gaps: Particularly in pediatric populations, standardized protocols concerning donor screening, long-term safety monitoring, and informed consent are still underdeveloped, hindering clinical implementation and public confidence.

Future Research Priorities

To accelerate the clinical translation of FMT in ADHD, future studies should focus on the following five strategic areas:

Standardization and optimization of intervention protocols: Researchers should develop unified donor screening criteria and identify key traits of so-called *super-donors*. Standardized, quality-controlled washed microbiota transplantation (WMT) technology should be promoted. Delivery routes and dosing frequencies should be compared to assess their effects on treatment adherence and efficacy.

Implementation of high-quality randomized controlled trials (RCTs): Multicenter, double-blind, placebo-controlled RCTs are needed across diverse age groups and ADHD subtypes. Evaluation frameworks should be multidimensional. These frameworks should integrate behavioral assessments, microbiome profiling, SCFA metabolomics, and neuroimaging.

Identification of predictive biomarkers: Future research should focus on microbiota taxa (eg, *Faecalibacterium*), metabolic pathways (eg, butyrate synthesis), and host immune markers associated with clinical response. Machine learning models can be leveraged to develop personalized microbiota-based treatment strategies.

Bridging basic and clinical science: Multi-omics platforms should be applied to reconstruct the complete gut–microbiota–behavior causal network. These platforms include metagenomics, metabolomics, and brain imaging. Innovative validation models should also be established. These may include organoids, gut–brain co-culture systems, and humanized animal models, all of which can enhance translational relevance.

Establishment of ethical and regulatory frameworks and product innovation: Regulatory mechanisms should be developed to cover donor health evaluation, microbial quality control, and long-term post-treatment surveillance. Research should also explore the development of next-generation microbial therapies to improve safety and acceptance. These therapies may include defined microbial consortia (so-called *ecobiotic drugs*) and functional postbiotics. Patient education and ethical oversight should be strengthened to prevent off-label misuse and address public misconceptions. Together, these measures can support the safe, effective, and ethically sound implementation of microbiota-based therapies in ADHD care.

Conclusions

Emerging evidence indicates that fecal microbiota transplantation (FMT) may alleviate ADHD-related symptoms by restoring microbiota–gut–brain axis homeostasis through immune, metabolic, and neuroregulatory pathways. Preclinical studies consistently demonstrate improvements in attention, hyperactivity, and impulsivity, while early clinical observations suggest feasibility and potential benefit. Nevertheless, the evidence remains preliminary, limited by small sample sizes, protocol heterogeneity, and incomplete mechanistic validation. To advance translation, future research should prioritize standardized FMT/WMT procedures, high-quality randomized controlled trials with multimodal endpoints, and the identification of predictive biomarkers to enable precision patient selection. Robust ethical and regulatory frameworks, particularly in pediatric populations, will also be essential to ensure safety and public confidence. Overall, FMT remains an exploratory but promising adjunctive strategy for ADHD that warrants rigorous clinical evaluation.

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References

1. Thapar A, Cooper M. Attention deficit hyperactivity disorder. *Lancet*. 2016;387(10024):1240–1250. doi:10.1016/S0140-6736(15)00238-X
2. Hours C, Recasens C, Baleyte JM. ASD and ADHD comorbidity: what are we talking about? *Front Psychiatry*. 2022;13:837424. doi:10.3389/fpsyt.2022.837424
3. Sibley MH, Mitchell JT, Becker SP. Method of adult diagnosis influences estimated persistence of childhood ADHD: a systematic review of longitudinal studies. *Lancet Psychiatry*. 2016;3(12):1157–1165. doi:10.1016/S2215-0366(16)30190-0
4. Jiménez-Muñoz L, Lopez-Fernandez O, Peñuelas-Calvo I, et al. Persistence of ADHD into adulthood and associated factors: a prospective study. *Psiquiatr Biol*. 2025;32(2):100529. doi:10.1016/j.psiq.2024.100529
5. Canela C, Buadze A, Dube A, Eich D, Liebrenz M. Skills and compensation strategies in adult ADHD - A qualitative study. *PLoS One*. 2017;12(9):e0184964. doi:10.1371/journal.pone.0184964
6. Hechtman L, Swanson JM, Sibley MH, et al. Functional adult outcomes 16 years after childhood diagnosis of attention-deficit/hyperactivity disorder: MTA results. *J Am Acad Child Adolesc Psychiatry*. 2016;55(11):945–952.e942. doi:10.1016/j.jaac.2016.07.774
7. Salari N, Ghasemi H, Abdoli N, et al. The global prevalence of ADHD in children and adolescents: a systematic review and meta-analysis. *Ital J Pediatr*. 2023;49(1):48. doi:10.1186/s13052-023-01456-1
8. Song P, Zha M, Yang Q, Zhang Y, Li X, Rudan I. The prevalence of adult attention-deficit hyperactivity disorder: a global systematic review and meta-analysis. *J Glob Health*. 2021;11:04009. doi:10.7189/jogh.11.04009
9. Ayano G, Demelash S, Gizachew Y, Tsegay L, Alati R. The global prevalence of attention deficit hyperactivity disorder in children and adolescents: an umbrella review of meta-analyses. *J Affect Disord*. 2023;339:860–866. doi:10.1016/j.jad.2023.07.071
10. Arnold LE, Roy A, Taylor E, et al. Predictive utility of childhood diagnosis of ICD-10 hyperkinetic disorder: adult outcomes in the MTA and effect of comorbidity. *Eur Child Adolesc Psychiatry*. 2019;28(4):557–570. doi:10.1007/s00787-018-1222-0
11. Shen F, Zhou H. Advances in the etiology and neuroimaging of children with attention deficit hyperactivity disorder. *Front Pediatr*. 2024;12:1400468. doi:10.3389/fped.2024.1400468
12. Faraone SV, Larsson H. Genetics of attention deficit hyperactivity disorder. *Mol Psychiatry*. 2019;24(4):562–575. doi:10.1038/s41380-018-0070-0
13. Valladão SC, França AP, Pandolfo P, Dos Santos-Rodrigues A. Adenosinergic system and nucleoside transporters in attention deficit hyperactivity disorder: current findings. *Neurosci Biobehav Rev*. 2024;164:105771. doi:10.1016/j.neubiorev.2024.105771
14. Agoalikum E, Klugah-Brown B, Wu H, Hu P, Jing J, Biswal B. Structural differences among children, adolescents, and adults with attention-deficit/hyperactivity disorder and abnormal Granger causality of the right pallidum and whole-brain. *Front Hum Neurosci*. 2023;17:1076873. doi:10.3389/fnhum.2023.1076873
15. Liu Y, Zhang P, Sun H. A narrative review of research advances in gut microbiota and microecological agents in children with attention deficit hyperactivity disorder (ADHD). *Front Psychiatry*. 2025;16:1588135. doi:10.3389/fpsyt.2025.1588135
16. Jallow J, Hurtig T, Kerkelä M, Miettunen J, Halt AH. Prenatal maternal stress, breastfeeding and offspring ADHD symptoms. *Eur Child Adolesc Psychiatry*. 2024;33(11):4003–4011. doi:10.1007/s00787-024-02451-5
17. Yu CL, Kao YC, Thompson T, et al. Response trajectories and temporal trends of Viloxazine treatment for young people with ADHD: a meta-analysis. *JAMA Network Open*. 2024;7(11):e2445885. doi:10.1001/jamanetworkopen.2024.45885
18. Radonjić NV, Bellato A, Khoury NM, Cortese S, Faraone SV. Nonstimulant medications for Attention-Deficit/Hyperactivity Disorder (ADHD) in adults: systematic review and meta-analysis. *CNS Drugs*. 2023;37(5):381–397. doi:10.1007/s40263-023-01005-8
19. Strålin EE, Thorell LB, Lundgren T, Bölte S, Bohman B. Cognitive behavioral therapy for ADHD predominantly inattentive presentation: randomized controlled trial of two psychological treatments. *Front Psychiatry*. 2025;16:1564506. doi:10.3389/fpsyt.2025.1564506
20. Charitos IA, Inchingolo AM, Ferrante L, et al. The gut microbiota's role in neurological, psychiatric, and neurodevelopmental disorders. *Nutrients*. 2024;16(24):4404. doi:10.3390/nu16244404
21. Lewis N, Villani A, Lagopoulos J. Gut dysbiosis as a driver of neuroinflammation in attention-deficit/hyperactivity disorder: a review of current evidence. *Neuroscience*. 2025;569:298–321. doi:10.1016/j.neuroscience.2025.01.031
22. Kumar A, Pramanik J, Goyal N, et al. Gut microbiota in anxiety and depression: unveiling the relationships and management options. *Pharmaceuticals*. 2023;16(4):565. doi:10.3390/ph16040565
23. Shirvani-Rad S, Ejtahed HS, Ettehad Marvasti F, et al. The role of gut microbiota-brain axis in pathophysiology of ADHD: a systematic review. *J Attention Disord*. 2022;26(13):1698–1710. doi:10.1177/10870547211073474
24. Nicholson MR, Mitchell PD, Alexander E, et al. Efficacy of fecal microbiota transplantation for Clostridium difficile infection in children. *Clin Gastroenterol Hepatol*. 2020;18(3):612–619.e611. doi:10.1016/j.cgh.2019.04.037
25. Bieganska EA, Kosinski P, Wolski M. Possible applications of fecal microbiota transplantation in the pediatric population: a systematic review. *Biomedicines*. 2025;13(6):1393. doi:10.3390/biomedicines13061393
26. Zhang J, Zhu G, Wan L, et al. Effect of fecal microbiota transplantation in children with autism spectrum disorder: a systematic review. *Front Psychiatry*. 2023;14:1123658. doi:10.3389/fpsyt.2023.1123658
27. Kang DW, Adams JB, Coleman DM, et al. Long-term benefit of microbiota transfer therapy on autism symptoms and gut microbiota. *Sci Rep*. 2019;9(1):5821. doi:10.1038/s41598-019-42183-0
28. Harigai W, Mikami K, Choudhury ME, et al. Effects of fecal microbiota transplantation on behavioral abnormality in attention deficit hyperactivity disorder-like model rats. *J Pharmacol Sci*. 2025;157(3):189–198. doi:10.1016/j.jphs.2025.01.007
29. Hooi SL, Dwiyanto J, Rasiti H, Toh KY, Wong RKM, Lee JWJ. A case report of improvement on ADHD symptoms after fecal microbiota transplantation with gut microbiome profiling pre- and post-procedure. *Curr Med Res Opin*. 2022;38(11):1977–1982.
30. Loh JS, Mak WQ, Tan LKS, et al. Microbiota-gut-brain axis and its therapeutic applications in neurodegenerative diseases. *Signal Transduct Target Ther*. 2024;9(1):37. doi:10.1038/s41392-024-01743-1
31. Ramadan YN, Alqifari SF, Alshehri K, et al. Microbiome gut-brain-axis: impact on brain development and mental health. *Mol Neurobiol*. 2025;62:10813–10833. doi:10.1007/s12035-025-04846-0
32. Bertollo AG, Santos CF, Bagatini MD, Ignácio ZM. Hypothalamus-pituitary-adrenal and gut-brain axes in biological interaction pathway of the depression. *Front Neurosci*. 2025;19:1541075. doi:10.3389/fnins.2025.1541075
33. Tan HE. The microbiota-gut-brain axis in stress and depression. *Front Neurosci*. 2023;17:1151478. doi:10.3389/fnins.2023.1151478

34. Abildinova GZ, Benberin VV, Vochshenkova TA, et al. The gut-brain-metabolic axis: exploring the role of microbiota in insulin resistance and cognitive function. *Front Microbiol*. 2024;15:1463958. doi:10.3389/fmicb.2024.1463958
35. Dam SA, Mostert JC, Szopinska-Tokov JW, Bloemendaal M, Amato M, Arias-Vasquez A. The role of the gut-brain axis in attention-deficit/hyperactivity disorder. *Gastroenterol Clin North Am*. 2019;48(3):407–431. doi:10.1016/j.gtc.2019.05.001
36. Fassarella M, Blaak EE, Penders J, Nauta A, Smidt H, Zoetendal EG. Gut microbiome stability and resilience: elucidating the response to perturbations in order to modulate gut health. *Gut*. 2021;70(3):595–605. doi:10.1136/gutjnl-2020-321747
37. Steckler R, Magzal F, Kokot M, Walkowiak J, Tamir S. Disrupted gut harmony in attention-deficit/hyperactivity disorder: dysbiosis and decreased short-chain fatty acids. *Brain Behav Immun Health*. 2024;40:100829. doi:10.1016/j.bbih.2024.100829
38. Wan L, Ge WR, Zhang S, Sun YL, Wang B, Yang G. Case-control study of the effects of gut microbiota composition on neurotransmitter metabolic pathways in children with attention deficit hyperactivity disorder. *Front Neurosci*. 2020;14:127. doi:10.3389/fnins.2020.00127
39. Yang LL, Stiernborg M, Skott E, et al. Lower plasma concentrations of short-chain fatty acids (SCFAs) in patients with ADHD. *J Psychiatr Res*. 2022;156:36–43. doi:10.1016/j.jpsychires.2022.09.042
40. Kearns R. Gut-brain axis and neuroinflammation: the role of gut permeability and the Kynurenine pathway in neurological disorders. *Cell Mol Neurobiol*. 2024;44(1):64. doi:10.1007/s10571-024-01496-z
41. Deng W, Yi P, Xiong Y, et al. Gut metabolites acting on the gut-brain axis: regulating the functional state of microglia. *Aging Dis*. 2024;15(2):480–502. doi:10.14336/AD.2023.0727
42. Lu J, Lu L, Yu Y, Baranowski J, Claud EC. Maternal administration of probiotics promotes brain development and protects offspring's brain from postnatal inflammatory insults in C57/BL6J mice. *Sci Rep*. 2020;10(1):8178. doi:10.1038/s41598-020-65180-0
43. Virk MS, Virk MA, He Y, et al. The anti-inflammatory and curative exponent of probiotics: a comprehensive and authentic ingredient for the sustained functioning of major human organs. *Nutrients*. 2024;16(4). doi:10.3390/nu16040546
44. Liu T, Ji H, Li Z, et al. Gut microbiota causally impacts adrenal function: a two-sample mendelian randomization study. *Sci Rep*. 2024;14(1):23338. doi:10.1038/s41598-024-73420-w
45. Molska M, Mruczyk K, Cisek-Woźniak A, et al. The Influence of Intestinal Microbiota on BDNF Levels. *Nutrients*. 2024;16(17):2891. doi:10.3390/nu16172891
46. Fock E, Parnova R. Mechanisms of blood-brain barrier protection by microbiota-derived short-chain fatty acids. *Cells*. 2023;12(4):657. doi:10.3390/cells12040657
47. watt R, Parkin K, Martino D. The Potential Effects of Short-Chain Fatty Acids on the Epigenetic Regulation of Innate Immune Memory. *Challenges*. 2020;11(2):25. doi:10.3390/challe11020025
48. Braga JD, Thongngam M, Kumrungsee T. Gamma-aminobutyric acid as a potential postbiotic mediator in the gut-brain axis. *NPJ Sci Food*. 2024;8(1):16. doi:10.1038/s41538-024-00253-2
49. Yadegar A, Bar-Yoseph H, Monaghan TM, et al. Fecal microbiota transplantation: current challenges and future landscapes. *Clin Microbiol Rev*. 2024;37(2):e0006022.
50. Eslami M, Adampour Z, Fadaee Dowlat B, et al. A novel frontier in gut-brain axis research: the transplantation of fecal microbiota in neurodegenerative disorders. *Biomedicines*. 2025;13(4):915. doi:10.3390/biomedicines13040915
51. Sayed SZ, Hassan ZO, Abdelraheem WM, Refaat RS, Abuelela IS. Is there a link between peripheral inflammation and blood brain barrier integrity in children with attention-deficit/hyperactivity disorder? A case-control study. *BMC Pediatric*. 2024;24(1):769. doi:10.1186/s12887-024-05254-4
52. Vázquez-González D, Carreón-Trujillo S, Alvarez-Arellano L, et al. A potential role for neuroinflammation in ADHD. *Adv Exp Med Biol*. 2023;1411:327–356.
53. Di Vincenzo F, Del Gaudio A, Petito V, Lopetuso LR, Scaldaferri F. Gut microbiota, intestinal permeability, and systemic inflammation: a narrative review. *Int Emerg Med*. 2024;19(2):275–293. doi:10.1007/s11739-023-03374-w
54. Neurath MF, Artis D, Becker C. The intestinal barrier: a pivotal role in health, inflammation, and cancer. *Lancet Gastroenterol Hepatol*. 2025;10(6):573–592. doi:10.1016/S2468-1253(24)00390-X
55. Soveral LF, Korczaguin GG, Schmidt PS, Nunes IS, Fernandes C, Zárate-Bladés CR. Immunological mechanisms of fecal microbiota transplantation in recurrent Clostridioides difficile infection. *World J Gastroenterol*. 2022;28(33):4762–4772. doi:10.3748/wjg.v28.i33.4762
56. Luu LDW, Pandey A, Paramsothy S, et al. Profiling the colonic mucosal response to fecal microbiota transplantation identifies a role for GBP5 in colitis in humans and mice. *Nat Commun*. 2024;15(1):2645.
57. Jang HM, Kim JK, Joo MK, et al. Transplantation of fecal microbiota from patients with inflammatory bowel disease and depression alters immune response and behavior in recipient mice. *Sci Rep*. 2021;11(1):20406. doi:10.1038/s41598-021-00088-x
58. MacDonald HJ, Kleppe R, Szigetvari PD, Haavik J. The dopamine hypothesis for ADHD: an evaluation of evidence accumulated from human studies and animal models. *Front Psychiatry*. 2024;15:1492126.
59. Ferranti AS, Luessen DJ, Niswender CM. Novel pharmacological targets for GABAergic dysfunction in ADHD. *Neuropharmacology*. 2024;249:109897. doi:10.1016/j.neuropharm.2024.109897
60. Dziedzic A, Maciak K, Bliźniewska-Kowalska K, Gałecka M, Kobierecka W, Saluk J. The power of psychobiotics in depression: a modern approach through the microbiota-gut-brain axis: a literature review. *Nutrients*. 2024;16(7):1054. doi:10.3390/nu16071054
61. Tette FM, Kwofie SK, Wilson MD. Therapeutic anti-depressant potential of microbial GABA produced by Lactobacillus rhamnosus strains for GABAergic signaling restoration and inhibition of addiction-induced HPA axis hyperactivity. *Curr Issues Mol Biol*. 2022;44(4):1434–1451. doi:10.3390/cimb44040096
62. Hediyal TA, Vichitra C, Anand N, et al. Protective effects of fecal microbiota transplantation against ischemic stroke and other neurological disorders: an update. *Front Immunol*. 2024;15:1324018. doi:10.3389/fimmu.2024.1324018
63. Li N, Chen H, Cheng Y, et al. Fecal microbiota transplantation relieves gastrointestinal and autism symptoms by improving the gut microbiota in an open-label study. *Front Cell Infect Microbiol*. 2021;11:759435. doi:10.3389/fcimb.2021.759435
64. Allahyari P, Abbas Torki S, Aminnezhad Kavkani B, et al. A systematic review of the beneficial effects of prebiotics, probiotics, and synbiotics on ADHD. *Neuropsychopharmacol Rep*. 2024;44(2):300–307. doi:10.1002/npr2.12437
65. Elhossiny RM, Elshahawy HH, Mohamed HM, Abdelmageed RI. Assessment of probiotic strain Lactobacillus acidophilus LB supplementation as adjunctive management of attention-deficit hyperactivity disorder in children and adolescents: a randomized controlled clinical trial. *BMC Psychiatry*. 2023;23(1):823. doi:10.1186/s12888-023-05324-4

66. Zhi J, Zhang S, Huang M, et al. Transcutaneous auricular vagus nerve stimulation as a potential therapy for attention deficit hyperactivity disorder: modulation of the noradrenergic pathway in the prefrontal lobe. *Front Neurosci*. 2024;18:1494272. doi:10.3389/fnins.2024.1494272
67. Bellato A, Arora I, Hollis C, Groom MJ. Is autonomic nervous system function atypical in attention deficit hyperactivity disorder (ADHD)? A systematic review of the evidence. *Neurosci Biobehav Rev*. 2020;108:182–206. doi:10.1016/j.neubiorev.2019.11.001
68. Rusch JA, Layden BT, Dugas LR. Signalling cognition: the gut microbiota and hypothalamic-pituitary-adrenal axis. *Front Endocrinol*. 2023;14:1130689. doi:10.3389/fendo.2023.1130689
69. Zhang Q, Bi Y, Zhang B, et al. Current landscape of fecal microbiota transplantation in treating depression. *Front Immunol*. 2024;15:1416961. doi:10.3389/fimmu.2024.1416961
70. Marcondes Ávila PR, Fiorot M, Michels M, et al. Effects of microbiota transplantation and the role of the vagus nerve in gut-brain axis in animals subjected to chronic mild stress. *J Affect Disord*. 2020;277:410–416. doi:10.1016/j.jad.2020.08.013
71. Cecil CAM, Nigg JT. Epigenetics and ADHD: reflections on current knowledge, research priorities and translational potential. *Mol Diagn Ther*. 2022;26(6):581–606. doi:10.1007/s40291-022-00609-y
72. Alpino G, Pereira-Sol GA, Dias MME, Aguiar AS, Peluzio M. Beneficial effects of butyrate on brain functions: a view of epigenetic. *Crit Rev Food Sci Nutr*. 2024;64(12):3961–3970. doi:10.1080/10408398.2022.2137776
73. Hou H, Zhao H. Epigenetic factors in atherosclerosis: DNA methylation, folic acid metabolism, and intestinal microbiota. *Clin Chim Acta*. 2021;512:7–11. doi:10.1016/j.cca.2020.11.013
74. Avolio E, Olivito I, Rosina E, et al. Modifications of behavior and inflammation in mice following transplant with fecal microbiota from children with autism. *Neuroscience*. 2022;498:174–189. doi:10.1016/j.neuroscience.2022.06.038
75. Hiergeist A, Gessner J, Gessner A. Current limitations for the assessment of the role of the gut microbiome for Attention Deficit Hyperactivity Disorder (ADHD). *Front Psychiatry*. 2020;11:623. doi:10.3389/fpsyt.2020.00623
76. Tengeler AC, Dam SA, Wiesmann M, et al. Gut microbiota from persons with attention-deficit/hyperactivity disorder affects the brain in mice. *Microbiome*. 2020;8(1):44. doi:10.1186/s40168-020-00816-x
77. Watanangura A, Meller S, Farhat N, et al. Behavioral comorbidities treatment by fecal microbiota transplantation in canine epilepsy: a pilot study of a novel therapeutic approach. *Front Vet Sci*. 2024;11:1385469. doi:10.3389/fvets.2024.1385469
78. Morys J, Małecki A, Nowacka-Chmielewska M. Stress and the gut-brain axis: an inflammatory perspective. *Front Mol Neurosci*. 2024;17:1415567. doi:10.3389/fnmol.2024.1415567
79. Cerna C, Vidal-Herrera N, Silva-Olivares F, et al. Fecal microbiota transplantation from young-trained donors improves cognitive function in old mice through modulation of the gut-brain axis. *Aging Dis*. 2025.
80. Caputi V, Hill L, Figueiredo M, et al. Functional contribution of the intestinal microbiome in autism spectrum disorder, attention deficit hyperactivity disorder, and Rett syndrome: a systematic review of pediatric and adult studies. *Front Neurosci*. 2024;18:1341656. doi:10.3389/fnins.2024.1341656
81. Checa-Ros A, Jeréz-Calero A, Molina-Carballo A, Campoy C, Muñoz-Hoyos A. Current evidence on the role of the gut microbiome in ADHD pathophysiology and therapeutic implications. *Nutrients*. 2021;13(1):249. doi:10.3390/nu13010249
82. Kwak MJ, Kim SH, Kim HH, et al. Psychobiotics and fecal microbial transplantation for autism and attention-deficit/hyperactivity disorder: microbiome modulation and therapeutic mechanisms. *Front Cell Infect Microbiol*. 2023;13:1238005. doi:10.3389/fcimb.2023.1238005
83. Hu DX, Lu CM, Si XY, et al. Effects of gastrointestinal symptoms on the efficacy of washed microbiota transplantation in patients with autism. *Front Pediatr*. 2025;13:1528167. doi:10.3389/fped.2025.1528167

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