International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	FOOD, MICROBES, AND THE MIND: INTERVENTIONS AND TECH-FOR-HEALTH IMPLICATIONS
DOI	https://doi.org/10.31435/ijitss.3(47).2025.3841
RECEIVED	28 July 2025
ACCEPTED	16 September 2025
PUBLISHED	30 September 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

© The author(s) 2025.
This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

FOOD, MICROBES, AND THE MIND: INTERVENTIONS AND TECH-FOR-HEALTH IMPLICATIONS

Wojciech Pabis *(Corresponding Author, Email: wojciech.pabis@wp.pl)*
5th Military Research Hospital and Polyclinic, Kraków, Poland
ORCID ID: 0009-0006-3455-4724

Patryk Biesaga
W. Orłowski Hospital, Warsaw, Poland
ORCID ID: 0009-0003-0242-9126

Konrad Kotte
St. Alexander Hospital, Kielce, Poland; W. Biegański Specialist Hospital, Jędrzejów, Poland
ORCID ID: 0009-0008-9745-985X

Olaf Jadanowski
University of Health Sciences in Bydgoszcz, Bydgoszcz, Poland
ORCID ID: 0009-0000-6279-3067

Kamil Łebek
St. Luke's Specialist Hospital, Końskie, Poland
ORCID ID: 0009-0005-4612-5022

Weronika Sobota
National Medical Institute of the MSWiA, Warsaw, Poland
ORCID ID: 0009-0000-7778-7030

Przemysław Piskorz
National Medical Institute of the MSWiA, Warsaw, Poland
ORCID ID: 0009-0000-9812-2230

Daria Litworska-Sójka
Provincial Combined Hospital, Kielce, Poland
ORCID ID: 0009-0005-4590-3777

Bartosz Komsta
Military Institute of Medicine – National Research Institute, Warsaw, Poland
ORCID ID: 0009-0003-1159-5855

Julia Lipiec
Provincial Combined Hospital, Kielce, Poland
ORCID ID: 0009-0004-3351-4056

ABSTRACT

Research objectives: To synthesise recent evidence on microbiome-brain relationships across Alzheimer's disease, Parkinson's disease, multiple sclerosis, autism spectrum disorder, depression and schizophrenia; to summarise therapeutic strategies (diet, probiotics/prebiotics, psychobiotics, faecal microbiota transplantation) and biological mechanisms; and to outline translational considerations relevant to technology and society.
Methods: Structured narrative review with a PRISMA-style workflow.
Sources: PubMed/MEDLINE and Web of Science (Core Collection), plus handsearch/citation chasing (English/Polish; 1 Jan 2013–31 Mar 2025). Ten authors performed duplicate screening and data charting. Heterogeneity precluded meta-analysis. Included n=21 studies after databases identified n=1,500 records, duplicates n=540, screened n=960, and full texts assessed n=252 (databases) and n=48 from other sources; the reference list also cites background/methodological works not counted in PRISMA.
Key findings: Across conditions, a consistent signal is loss of short-chain-fatty-acid (SCFA)–producing taxa, increased intestinal permeability and immune activation. High-fibre/polyphenol diets and multi-strain probiotics/prebiotics show the clearest-though modest-improvements in inflammatory markers and selected mood/quality-of-life outcomes; cognitive effects are mixed. Psychobiotics show preliminary benefits; faecal microbiota transplantation remains experimental in neurology. Mechanistic strands include SCFAs, tryptophan/kynurenine metabolism, vagal signalling and HPA-axis modulation.
Conclusions: The gut microbiome is a modifiable contributor to brain health. Low-risk dietary optimisation is warranted, and probiotic use should be strain-specific. Priorities include adequately powered, preregistered trials with harmonised microbiome pipelines and mechanistic endpoints, plus evaluation of precision-nutrition and data-driven decision support under robust privacy governance.

KEYWORDS

Gut–Brain Axis, Microbiome, Short-Chain Fatty Acids, Neuroinflammation, Probiotics, Precision Nutrition

CITATION

Wojciech Pabis, Patryk Biesaga, Konrad Kotte, Olaf Jadanowski, Kamil Łebek, Weronika Sobota, Przemysław Piskorz, Daria Litworska-Sójka, Bartosz Komsta, Julia Lipiec. (2025) Food, Microbes, and the Mind: Interventions and Tech-for-Health Implications. *International Journal of Innovative Technologies in Social Science*, 3(47). doi: 10.31435/ijitss.3(47).2025.3841

COPYRIGHT

© The author(s) 2025. This article is published as open access under the **Creative Commons Attribution 4.0 International License (CC BY 4.0)**, allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

Introduction

The gut–brain axis is a bidirectional communication system in which the gut microbiota helps regulate neural and immune processes underpinning mood, cognition and behaviour (Cryan et al., 2019). Across neurological disorders-including Alzheimer's disease, Parkinson's disease and multiple sclerosis-intestinal dysbiosis has been linked with disease onset or progression (Dinan & Cryan, 2017). Similar patterns are reported in developmental and psychiatric conditions such as autism, depression and schizophrenia (Góralczyk-Bińkowska et al., 2022). Mechanistically, the microbiome influences cognition (memory, attention, executive functions) and affect via microbially derived neurotransmitters (serotonin, GABA, dopamine) and metabolites-especially short-chain fatty acids (SCFAs)-and by shaping immune signalling (Lorenc et al., 2020). Human diet affects both mood (Sánchez-Villegas et al., 2013) and the gut microbiome (David et al., 2014); correspondingly, high-fibre dietary patterns, probiotics and prebiotics, and faecal microbiota transplantation (FMT) have emerged as candidate interventions to improve neurological status in selected conditions (Sarkar et al., 2016). Signals of microbiome involvement are reported even in severe disorders such as MS (Jangi et al., 2016). For a translational and policy perspective, see the section'Implications for technology & society'.

From a public-health perspective, the burden of neurological and psychiatric diseases has risen sharply over recent decades (Cadilhac & Mahal, 2024), with depressive disorders remaining among the leading causes of global disability (Modesto-Lowe et al., 2023). In parallel, advances in high-throughput'omics' have enabled precise characterisation of the microbiome's taxonomic and functional profiles (Tankou et al., 2018). This technological landscape increasingly intersects with society: consumer microbiome testing, mobile health apps

and data-driven nutrition tools promise personalised guidance but also raise questions about equity of access, data governance and clinical validity. At the same time, computational methods-including machine-learning on multi-omics-offer opportunities to identify reproducible microbial and metabolic signatures relevant to mood and cognition, while digital therapeutics and decision-support systems may help tailor diet, prebiotics and probiotic strains at scale. The gut microbiota itself is a diverse community of bacteria, viruses and fungi shaped by diet, age, sex and environment; it communicates with the central nervous system through immune, neural (vagus nerve, enteric nervous system) and endocrine (hypothalamic–pituitary–adrenal axis) pathways (Hsiao et al., 2013; Li et al., 2020).

Objective

Against this background, we provide a structured narrative review of clinical and translational evidence up to 2025 on the role of the gut microbiome in neurological diseases and cognitive functions. We (I) summarise biological mechanisms with emphasis on SCFAs, neurotransmitters and immune signalling; (II) appraise microbiota-targeted interventions (dietary fibre/polyphenols, multi-strain probiotics and prebiotics, FMT); and (III) discuss technological and societal implications, including opportunities for precision nutrition, data-driven decision support and ethical considerations around testing and FMT. This framing aligns the biomedical evidence with contemporary debates at the interface of technology and social science.

Disharmony of the microbiota ("dysbiosis") is associated with the release of unfavorable metabolites and pro-inflammatory signals, which may predispose to neurological disorders (Frej-Mądrzak et al., 2021). For example, reduced production of short-chain fatty acids (SCFAs such as butyrate) leads to weakening of intestinal-barrier integrity and increased systemic inflammation, which may intensify neurodegenerative processes (Kelly et al., 2016). Specific microbiome components influence the production of neurotransmitters (including serotonin, dopamine, and GABA) and modulate microglial activity, directly affecting cognitive functions and mood (Liu et al., 2022). Moreover, genetic and environmental factors such as diet and lifestyle alter the microbiota and may modify the risk of brain diseases (Zeng et al., 2018). The aim of this paper is to present the current state of knowledge on the role of the gut microbiome in the etiology and course of specific neurological and psychiatric diseases (Alzheimer's, Parkinson's, MS, autism, depression, schizophrenia) and its impact on cognitive functions and mood (Alli et al., 2022). We discuss the latest clinical studies and systematic reviews (up to 2025), the biological gaps of gut–brain communication, and the results of interventions aimed at modifying the microbiota (Nguyen et al., 2019).

Methodology

We conducted a structured narrative review reported with a PRISMA-style flow diagram; ten authors contributed to search, screening and data extraction. A protocol was not registered.

Information sources and timeframe

We searched PubMed/MEDLINE and Web of Science-Core Collection; in addition, we performed handsearching in Google Scholar and citation chasing from reference lists. The time window covered 1 January 2013–31 March 2025; eligible languages were English and Polish.

Search strategy

Database queries used explicit Boolean logic (PubMed Title/Abstract field; Web of Science Topic), combining terms for the gut microbiome/gut–brain axis with target conditions (Alzheimer's disease, Parkinson's disease, multiple sclerosis, autism spectrum disorder, depression, schizophrenia). Exact, replicable strings and filters are provided in Supplement S1.

Eligibility criteria

We included human clinical and translational animal studies examining the gut microbiome or gut–brain axis in relation to the above conditions and reporting relevant clinical, cognitive or mood outcomes. We excluded records (I) not focused on the gut microbiome; (II) without primary data (reviews/editorials/comments); (III) non-human without a translational link; (IV) with off-topic outcomes; (V) conference abstracts without full text; and (VI) duplicates.

Study selection and data charting

Records from each source were exported and deduplicated (exact DOI/PMID match, then fuzzy match on title+first author+year, with manual verification). Pairs of reviewers independently screened titles/abstracts and then full texts; disagreements were resolved by consensus, with a third reviewer available as arbiter. Data were charted in duplicate using a predefined template (population/setting; microbiome methods; taxa/metabolites including SCFAs; neurological/psychiatric outcomes; effect measures; main findings). Per-study characteristics are compiled in Supplement S3, with a qualitative mini risk-of-bias assessment in Supplement S4.

Synthesis and risk of bias

Given heterogeneity of designs and outcomes, we performed narrative synthesis by condition and mechanistic domains; no meta-analysis or formal risk-of-bias tool was applied.

Numbers (PRISMA 2020)

From databases we identified n=1, 500 records (PubMed 860; Web of Science 640). After removing duplicates n=540, n=960 records were screened at title/abstract; n=700 were excluded. We sought n=260 reports for full-text retrieval; n=8 were not retrieved; n=252 full texts were assessed, with n=233 exclusions for the following reasons: not microbiome-focused (92); review/editorial/no primary data (56); animal-only without translational link (38); insufficient data/inadequate methods (21); wrong population/age (14); language/no full text (12). In parallel, other sources contributed n=48 reports (handsearching/citation chasing): not retrieved n=0; assessed n=48; excluded n=46. In total, n=21 studies were included.

Note

In addition to the studies included in the PRISMA selection (n=21), we cite background reviews, methodological standards and regulatory statements that informed interpretation and reporting; these sources were not eligible for inclusion as primary studies and are therefore not counted in PRISMA.

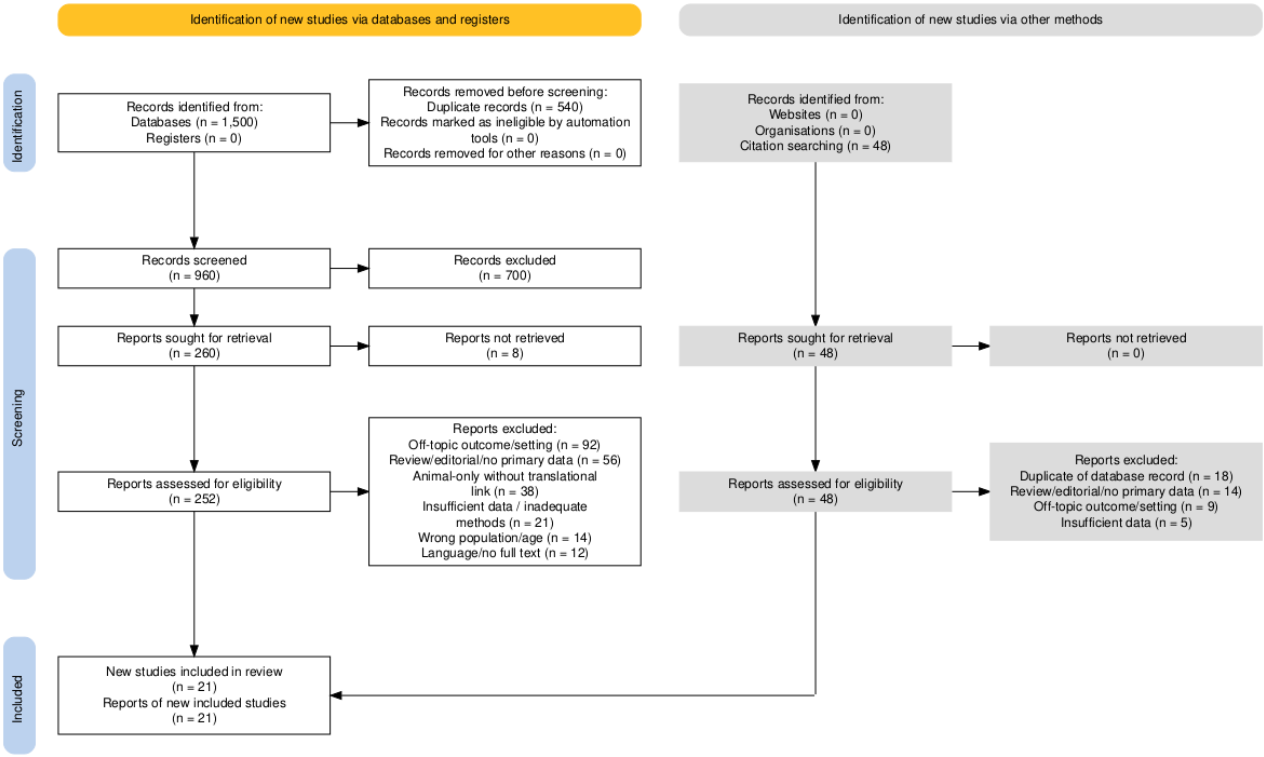

Fig. 1. *PRISMA 2020 flow diagram.*

Note: The reference list also contains background/methodological sources that were not eligible as included studies.

Results

We included n=21 studies following screening and full-text assessment (Figure 1). Study-level details are summarised in Supplement S3; mini risk-of-bias judgements are provided in Supplement S4.

Gut microbiome in neurodegenerative diseases

Alzheimer's disease (AD). Characteristic alterations in the gut microbiota are observed in AD (Vogt et al., 2017). Patients with AD show increased relative abundance of Bacteroidetes and decreased Firmicutes and Actinobacteria (Chandra et al., 2023). Such changes entail a decline in the production of beneficial metabolites, mainly SCFAs (e.g., butyrate), which favors inflammation (Maier et al., 2018). Transgenic mouse models of AD exhibit lower faecal SCFA content (Zajac et al., 2022). Preclinical evidence suggests that manipulating the microbiota-including germ-free or antibiotic-perturbed conditions and donor faecal transfers-can modulate amyloid pathology and cognition in AD models; however, findings are heterogeneous and largely hypothesis-generating (Strandwitz, 2018; Jiang et al., 2025; Nassar et al., 2022). In animal models, FMT from healthy donors reduced β-amyloid deposits while improving cognitive functions and synaptic plasticity (Jiang et al., 2025). Similar effects (improved MMSE scores, increased SCFA production) have been described in isolated human case reports after FMT (Park et al., 2021). Nevertheless, large clinical trials on FMT in AD are still lacking, and most data come from animal experiments and single observations (Nassar et al., 2022).

Parkinson's disease (PD). In PD, gastrointestinal disturbances are an early symptom. In a cohort study, 32.7% of patients reported constipation at the initial stages of the disease (Camacho et al., 2021). Microbiological studies demonstrate significant differences in microbiota composition in PD compared with healthy controls (Fung et al., 2017). PD patients commonly show reduced richness of butyrate-producing taxa-such as Faecalibacterium (Ruminococcaceae) and Coprococcus (Lachnospiraceae)-and increased mucin-degrading bacteria (e.g., Akkermansia muciniphila) (Vascellari et al., 2020). Individuals with PD exhibit decreased faecal SCFAs (Salim et al., 2023) and altered polyamines (e.g., spermidine) (Vrijsen et al., 2023). Such dysbiosis may impair the intestinal mucosal barrier, facilitating the translocation of toxins (LPS, pesticides) to the vagal ganglion, leading to pathological accumulation of α-synuclein and the formation of Lewy bodies (Salim et al., 2023). Additionally, animal studies show that intestinal colonization with Proteus mirabilis can induce PD-like symptoms (motor impairment, α synucleinopathy) (Choi et al., 2018).

Multiple sclerosis (MS). In MS patients, increased abundance of Methanobrevibacter and Akkermansia and decreased abundance of Butyricimonas have been observed. These changes correlated with differences in the expression of genes involved in dendritic-cell maturation, interferon signalling, and the NF-κB pathway in circulating T cells and monocytes. Moreover, patients receiving disease-modifying therapy exhibited higher Prevotella and Sutterella and lower Sarcina compared with untreated patients. These findings suggest that changes in the gut microbiota may influence immune responses in MS patients, potentially contributing to disease pathogenesis (Góralczyk-Bińkowska et al., 2022). Emerging evidence points to immunomodulatory effects of Lactobacillus/Bifidobacterium formulations and disease-associated microbiome shifts in MS; however, robust clinical trials remain limited (Jangi et al., 2016; Góralczyk-Bińkowska et al., 2022).

Gut microbiota in developmental and psychiatric disorders

Autism (ASD). Children with ASD frequently exhibit co-occurring gastrointestinal disturbances, and their microbiota differs markedly from that of neurotypical peers. Typical alterations include increases in Clostridium, Desulfovibrio, Sutterella and decreases in Bifidobacterium and Prevotella (Sarkar et al., 2016). Disruptions of the gut–brain axis may influence neurodevelopment through bacterial toxins, altered tryptophan metabolism, and immune signalling (Cryan et al., 2019)(Dinan & Cryan, 2017). Preclinical models demonstrate that microbiota-modulating interventions can alter social and repetitive behaviours, supporting a causal role of gut microbes in ASD-relevant phenotypes (Hsiao et al., 2013).

Depression. Individuals with depression usually have lower microbial diversity and reduced abundance of SCFA-producing bacteria, especially Faecalibacterium and Coprococcus, which are linked to dopamine and serotonin synthesis (Modesto-Lowe et al., 2023). In clinical studies, multi-strain probiotics have been associated with improved mood and reduced depressive symptoms (Alli et al., 2022; Liu et al., 2022). Some meta-analyses suggest moderate efficacy of so-called psychobiotics-probiotics that affect mental functions (Hsiao et al., 2013).

Schizophrenia. Gut dysbiosis observed in schizophrenia includes decreased Ruminococcaceae and Lachnospiraceae and increased Lactobacillus and Enterococcus (Li et al., 2020). Preclinical reports indicate that patient-derived microbiota can induce neurochemical and behavioural changes in rodents; clinically,

altered taxa associate with symptom severity (Li et al., 2020; Frej-Mądrzak et al., 2021). Supplementation with probiotics (including Lactobacillus rhamnosus) in pilot studies improved negative symptoms and cognitive functions (Kelly et al., 2016).

Therapeutic interventions targeting the gut microbiota

In recent years, a growing number of studies have focused on the possibility of modulating the gut microbiota to improve neurological and psychiatric functions. The main therapeutic strategies include:

1. Probiotics and prebiotics.

Probiotics-live microorganisms that confer health benefits on the host-have shown potential for alleviating symptoms of depression, anxiety, ASD, and even neurodegenerative diseases (Cryan et al., 2019). Prebiotics-non-digestible food components that promote the growth of beneficial bacteria (e.g., inulin, fructo-oligosaccharides)-favorably affect microbiota composition and SCFA production, translating into reduced inflammation and improved gut-barrier function (Dinan & Cryan, 2017).

2. Faecal microbiota transplantation (FMT).

FMT involves transferring intestinal contents (most often stool) from a healthy donor to a patient. Initially used mainly to treat Clostridioides difficile infection, it is now considered a potential therapy for neurological diseases. Preliminary studies suggest improvements in cognitive function in PD patients and mitigation of ASD symptoms (Jiang et al., 2025)(Park et al., 2021). However, clinical trials remain limited due to ethical and safety considerations, and FMT is still an experimental procedure in neurology.

3. Diet.

The Mediterranean diet, rich in fiber, vegetables, fruits, legumes, fish, and olive oil, favorably affects microbiota diversity and reduces the risk of depression and neurodegenerative diseases (Sánchez-Villegas et al., 2013). Conversely, low-fiber diets high in simple sugars and trans fats promote dysbiosis, inflammation, and cognitive decline (David et al., 2014).

4. Psychobiotics.

This group of probiotics and prebiotics exerts documented effects on mental functions. Their mechanisms include modulation of neurotransmitters (e.g., GABA, serotonin), reduction of oxidative stress, attenuation of inflammatory cytokines, and improved gut-barrier integrity (Sarkar et al., 2016). Although research on psychobiotics is still at an early stage, initial findings are promising and suggest their potential role in treating mood and anxiety disorders.

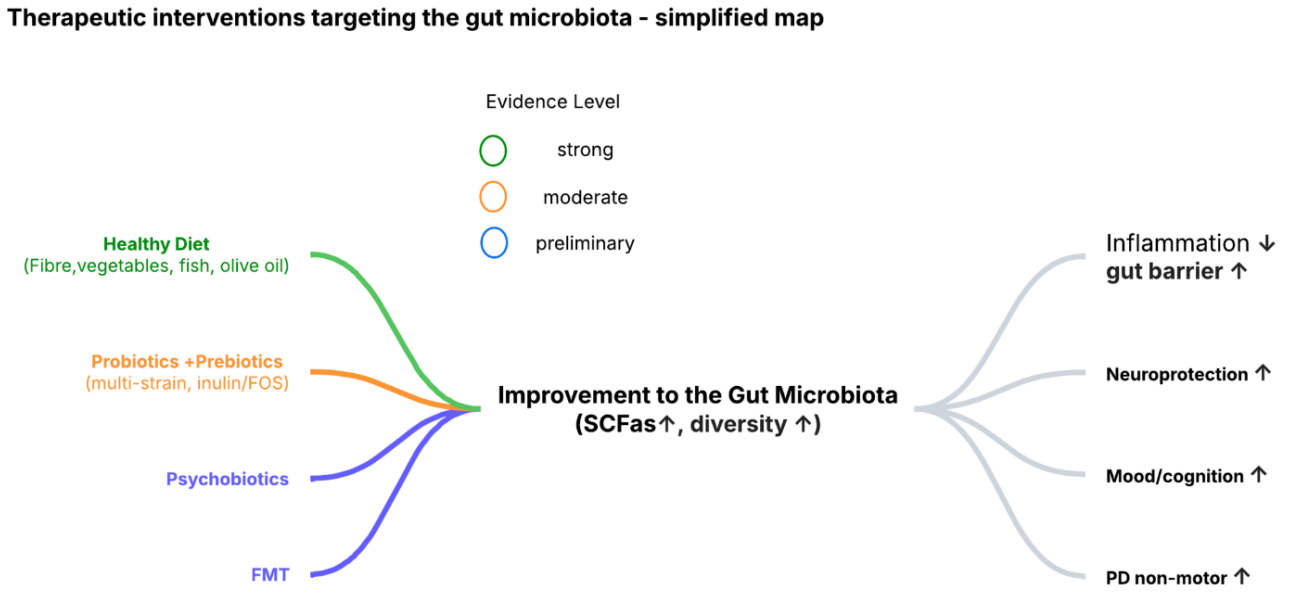

Fig. 2. *Therapeutic interventions targeting the gut microbiota-simplified map.*

Biological mechanisms of microbiome effects on brain centers

Communication between the microbiota and the brain is mediated by multiple pathways. Intestinal microorganisms produce a range of neuroactive substances: neurotransmitters (serotonin, dopamine, GABA, acetylcholine) and neuromodulators. For example, bacteria of the genera Escherichia, Bacteroides, and Bifidobacterium can synthesize GABA (Otaru et al., 2021), which, via GABA-A receptors in the brain, may reduce symptoms of depression and anxiety. Moreover, the gut flora strongly influences tryptophan metabolism-the precursors of serotonin and kynurenines-modulating systems responsible for mood (Agus et al., 2018). It is also believed that an adequate microbiota is necessary for proper maturation and activation of microglia, affecting synaptic health and brain plasticity.

A second key mechanism is modulation of the immune system. SCFAs (especially butyrate) produced by certain gut bacteria act as cellular regulators: they inhibit production of pro-inflammatory cytokines (TNF-α, IL-6) through epigenetic mechanisms (histone deacetylase inhibition) and enhance secretion of anti-inflammatory factors (IL-10). In this way, SCFAs can limit neuroinflammation and protect nerve cells. Conversely, dysbiosis increases intestinal permeability ("leaky gut"), and together with reduced SCFAs leads to the translocation into the circulation of endogenous bacterial toxins (lipopolysaccharides, LPS). These molecules penetrate the brain and stimulate microglia to produce inflammatory cytokines (IL-1β, IL-18) and chemokines, disrupting neuronal homeostasis. For example, SCFA deficiency observed in PD is attributed to weakening of the intestinal mucus barrier, which may initiate aggregation of pathological α-synuclein (Duan et al., 2024).

Neural and endocrine signalling is also an important element of the gut–brain axis. Through the vagus nerve and ENS connections, signals driven by gut metabolites are transmitted: for instance, SCFAs stimulate intestinal L-cells to secrete GLP-1 (glucagon-like peptide-1) and PYY (peptide YY), which modulate appetite as well as cognitive and neuroprotective processes. Both SCFAs and other bacterial metabolites (secondary bile acids, tryptophan and its derivatives) enter the bloodstream and act directly on brain centers (influencing the HPA axis) or indirectly via peripheral lymphocytes. Cytokines and metabolites can also cross the blood–brain barrier, communicating with neuroglia and neurons. As a result, the gut microbiota can regulate cognitive and emotional functions; for example, stimulation with the probiotic Lactobacillus rhamnosus in mice increases brain GABA levels and alleviates depressive symptoms (Kochalska et al., 2020).

Figure 3. Microbiome–brain mechanisms—main pathways in simplified view.

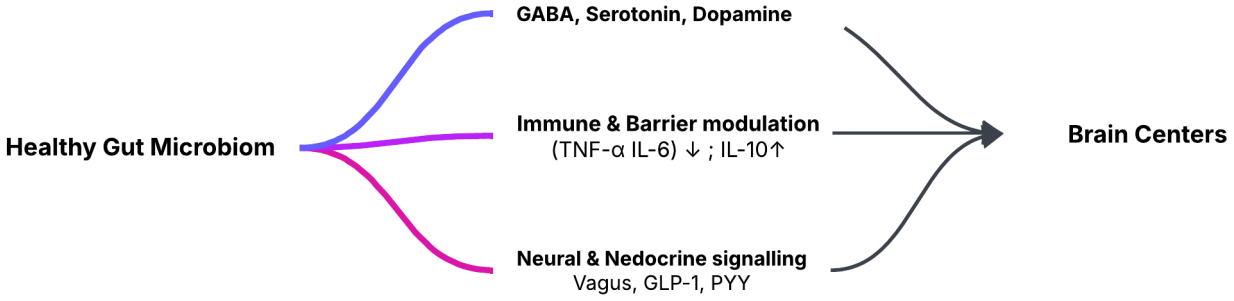

Fig. 3. *Microbiome-brain mechanisms-main pathways in simplified view.*

GLP-1=glucagon-like peptide-1; PYY=peptide YY; GABA=γ-aminobutyric acid; TNF-α=tumour necrosis factor-alpha; IL = interleukin (IL-6, IL-10).

Microbiome, cognitive functions, and mood

The gut microbiota affects not only disease risk but also brain function in both healthy and diseased individuals. Studies have shown that specific microbiome features are associated with memory performance, attention, and mood (Dinan & Cryan, 2017). In older adults, lower bacterial diversity in the gut correlated with poorer scores on cognitive tests. Low intestinal Bifidobacterium levels were associated with worse memory and attention (Chandra et al., 2023). In addition, patients with depressive disorders showed changes in GABA metabolism (increased GABA utilization by bacteria), which in turn exacerbated depressive symptoms. Importantly, specific microbiome features predicted the progression of cognitive and depressive disorders over time: lower abundance of certain bacteria (e.g., Akkermansia) was linked to faster cognitive decline. These findings suggest that the gut microbiota may constitute a biological marker (and potentially a therapeutic target) for both cognitive impairment and mood disorders (Li et al., 2022).

Table 1. Gut–brain conditions: microbiome alterations, mechanisms, and therapeutic implications (authors' compilation)

Condition	**Key gut–microbiota alterations**	**Dominant gut–brain mechanisms**	**Clinical takeaways/interventions (concise)**
Alzheimer's disease (AD)	Reduced SCFA-producing taxa (e.g., Faecalibacterium); altered diversity; bile-acid and tryptophan pathway shifts	SCFAs ↓ → immune activation; tryptophan →serotonin/kynurenines; preclinical modulation of amyloid and cognition	Fibre-rich dietary patterns; Mediterranean-style diet; selected multi-strain probiotics (exploratory); FMT experimental
Parkinson's disease (PD)	SCFAs ↓; ↑ mucin-degrading bacteria (e.g., Akkermansia muciniphila); ↑ Enterobacteriaceae; barrier dysfunction	LPS/leaky-gut–driven neuroinflammation; α-synuclein aggregation (preclinical links); vagal signalling	Higher fibre intake; diet quality (HEI) associated with SCFA producers; probiotics under study; FMT experimental (non-motor symptoms)
Schizophrenia	Altered community structure; signatures linked to metabolic risk; polyamine pathway changes reported	Immune activation and neurotransmitter modulation (e.g., GABA/tryptophan pathways)	Nutritional optimisation; early-stage probiotic evidence; monitor metabolic comorbidity
Multiple sclerosis (MS)	Reduced butyrate producers; disease-modifying therapies (DMTs) alter composition; pro-inflammatory taxa shifts	SCFAs → Treg support and anti-inflammatory tone; barrier integrity; cytokine signalling	Dietary fibre/quality patterns; Lactobacillus/Bifidobacterium formulations with immunomodulatory signals; need for robust RCTs
Autism spectrum disorder (ASD)	Lower diversity with shifts in Clostridia/Bacteroides reported; GI symptoms frequent; microbial metabolite differences	Vagal/ENS signalling; neurotransmitter (GABA) and tryptophan pathways; immune–barrier cross-talk	Dietary strategies; pre/probiotics (selected strains) under study; FMT experimental (strict safety/ethics)
Depression (MDD)	Dysbiosis with SCFA-producer depletion and ↑ opportunistic taxa; diet quality correlates with profiles	Cytokine-mediated inflammation; HPA axis; neurotransmitter modulation (GABA/serotonin)	Mediterranean-style/anti-inflammatory diets; psychobiotics (multi-strain) show symptom reduction in meta-analyses

Note: SCFAs=short-chain fatty acids; LPS=lipopolysaccharides; ENS=enteric nervous system; HPA=hypothalamic–pituitary–adrenal; DMTs=disease-modifying therapies; FMT=faecal microbiota transplantation; HEI=Healthy Eating Index

Modification of the microbiome-dietary and pharmacobiotic interventions

Clinical data indicate a significant impact of diet and supplementation on the microbiome and the course of neurological diseases (Frej-Mądrzak et al., 2021). A diet rich in fiber and anti-inflammatory components (e.g., prebiotic fibers, polyphenols) promotes the growth of beneficial SCFA-producing bacteria. In a study of PD patients, higher fiber intake and a better HEI-2015 diet score were associated with a greater abundance of Butyricicoccus and Coprococcus, which produce butyrate, and with fewer pro-inflammatory bacteria. Conversely, higher intake of simple sugars increased pathogenic bacteria (Kwon et al., 2024). In summary, a"healthy" diet can support the microbiome's anti-inflammatory and neuroprotective actions.

Probiotics and prebiotics are live bacterial strains or substrates that promote the development of beneficial bacteria. Systematic reviews indicate that multi-strain probiotics have a significant clinical effect in reducing inflammatory symptoms, depression, and anxiety. Administration of specific strains (e.g., Bifidobacterium, Lactobacillus) reduced inflammation in MS patients (Tankou et al., 2018) and, in rats, reduced anxiety-like behaviors and improved social functioning (Szklany et al., 2020). In multiple sclerosis, probiotic supplementation lowered CRP and TNF-α levels, improved neurological parameters, and subjective quality-of-life ratings.

Faecal microbiota transplantation (FMT) is gaining interest as an experimental therapy. In PD patients, it alleviated non-motor symptoms (insomnia, depression, anxiety) (Park et al., 2021). However, clinical trials are few due to ethical and safety issues.

In mice with a PD model, FMT alleviated blood-brain barrier damage and suppressed neuroinflammation in the substantia nigra, which further reduced dopaminergic neuron injury (Zhao et al., 2021).

Implications for technology & society

Digitalisation is reshaping how microbiome science translates into care. Here we outline opportunities and safeguards that matter for patients, clinicians and policymakers.

Precision nutrition and AI-enabled decision support

Integrating dietary logs, wearable-derived behaviour/sleep data and multi-omics with machine-learning can help identify subgroups who benefit from fibre-rich patterns, specific prebiotics or well-characterised probiotic strains. Evaluation should follow reporting standards for AI interventions to ensure transparent design, external validation and prespecified outcomes (e.g., inflammatory markers, SCFAs, cognitive measures) (Liu et al., 2020; Rivera et al., 2020). Models should incorporate uncertainty estimates and be audited for dataset shift and bias across age, sex and socioeconomic strata.

Consumer testing and mHealth

Direct-to-consumer microbiome tests and mobile apps can motivate behaviour change, yet analytical and clinical validity remain inconsistent across providers. An international consensus cautions against routine clinical use outside defined indications, and independent benchmarks highlight large inter-service variability (Porcari et al., 2024; Servetas et al., 2024). Reports to lay users should avoid deterministic language, communicate uncertainty and link advice to evidence-based dietary patterns rather than opaque"scores". In research, microbiome studies should follow STORMS reporting to improve reproducibility and comparability (Mirzayi et al., 2021).

Data governance, standards and equity

Microbiome profiles are identifiable and longitudinal; consent, data minimisation and secure storage are essential. FAIR data principles support responsible sharing while preserving privacy and reusability (Wilkinson et al., 2016). To avoid widening disparities, development and evaluation of tools should include under-represented populations and address affordability, language and digital access. Interoperability with electronic health records and cleardata provenance are necessary for clinical deployment.

Ethics and stewardship of FMT

Faecal microbiota transplantation remains experimental in neurology. Regulatory safety alerts underscore risks of pathogen transmission and the need for rigorous donor screening, traceability and informed consent (Food and Drug Administration, 2019; Food and Drug Administration, 2020). Current gastroenterology guidance recommends FMT primarily for recurrent Clostridioides difficile infection, not for neuropsychiatric indications (Cammarota et al., 2017; Peery et al., 2024). Any off-label use should be embedded in registries with active pharmacovigilance and transparent reporting of adverse events and benefit-risk.

Implementation pathways

Health systems can pilot"microbiome-aware" neurology workflows: brief evidence-based dietary counselling; pharmacist-supported probiotic stewardship; and decision-support prompts that translate SCFA-oriented advice into practical meal patterns. Minimum reporting sets (dietary intake, medications, sequencing method, targeted outcomes) would enable meta-research and real-world synthesis. Co-design with patients is critical to align tools with preferences and literacy.

Research agenda

Priorities include adequately powered, preregistered trials comparing dietary patterns and multi-strain formulations; harmonised laboratory pipelines; integration of mechanistic endpoints (SCFAs, cytokines, neuroimaging) with clinical outcomes; and head-to-head evaluations of digital decision-support. Pragmatic trials and registries can quantify effectiveness, safety and cost-utility in routine care.

Practical takeaway

Technology can help personalise diet-and microbe-based care, but only within robust clinical governance, privacy-preserving data practices and cautious communication to the public (Liu et al., 2020; Rivera et al., 2020; Mirzayi et al., 2021; Wilkinson et al., 2016; Porcari et al., 2024; Servetas et al., 2024; Food and Drug Administration, 2019; Food and Drug Administration, 2020; Cammarota et al., 2017; Peery et al., 2024).

Discussion

This discussion interprets the evidence summarised in the Results and situates it within the current literature; operational, digital and ethical aspects are addressed separately in Implications for technology & society.

Summary of evidence

Across neurological and psychiatric conditions, a recurring pattern of dysbiosis emerges: reduced short-chain-fatty-acid (SCFA)–producing taxa, impaired barrier integrity and low-grade immune activation. Mechanistic strands converge on SCFA-mediated immunomodulation, tryptophan/kynurenine metabolism and neural-endocrine signalling (vagal and HPA-axis), providing a biologically coherent link between diet, microbiota and brain outcomes. While effect sizes vary by condition and study design, these signals are directionally consistent and most apparent in outcomes related to inflammation and selected mood/cognitive domains.

Interventions: what is known

Dietary patterns rich in fibre and polyphenols and well-characterised multi-strain probiotics/prebiotics show the clearest-though modest-clinical benefits on inflammatory activity and selected mood/quality-of-life outcomes. Cognitive effects remain mixed. Psychobiotics provide preliminary signals that align with neurotransmitter-focused mechanisms, but require larger trials. Faecal microbiota transplantation remains experimental in neurology; any potential benefits must be weighed against safety, regulatory and donor-screening constraints. Strain specificity, baseline diet and medications likely mediate heterogeneity and should be reported explicitly.

Strengths and limitations of the evidence

Most available studies are small and heterogeneous in populations, laboratory pipelines (sampling, sequencing, bioinformatics) and outcome measures. Confounding by diet, medications (e.g., antibiotics, PPIs), comorbidities and lifestyle is common; reverse causation cannot be excluded in cross-sectional designs. Selective reporting and limited strain-level characterisation further reduce comparability and certainty.

Limitations of this review

This is a structured narrative review with PRISMA-style transparency but without meta-analysis. We synthesised heterogeneous designs narratively and did not apply a single formal risk-of-bias tool across all study types. Searches were limited to English and Polish within a defined timeframe, which may have missed grey literature or non-indexed sources. Our PRISMA-style transparency (Figure 1; Supplement S2) and provision of full search strings (Supplement S1) and study-level tables (Supplement S3–S4) aim to support reproducibility and auditability of the review."

Research priorities

Preregistered, adequately powered randomised trials should compare dietary patterns and multi-strain formulations with harmonised wet-lab and bioinformatic pipelines. Trials ought to integrate mechanistic endpoints (SCFAs, cytokines, metabolites, neuroimaging) with clinical and patient-reported outcomes, and report strain composition and dose. Pragmatic trials and registries-with standardised minimum datasets on diet, medications and sequencing methods-are needed to quantify effectiveness, safety and cost-utility in routine care, and to enable subgroup analyses for equitable implementation.

Operational Pathways, Digital Decision-Support, Consumer Testing, Data Standards and Stewardship Of Faecal Microbiota Transplantation are Detailed in Implications for Technology & Society

Conclusions

The gut microbiome is a modifiable contributor to neurological and psychiatric health. Across conditions, the most consistent signal is the loss of SCFA-producing taxa with downstream immune and barrier dysfunction; convergent mechanistic evidence supports SCFA-mediated, neural and endocrine pathways linking gut and brain. Among interventions, high-fibre/polyphenol dietary patterns and multi-strain probiotics/prebiotics show the clearest, yet modest clinical benefits on inflammatory activity and selected mood/cognitive outcomes, whereas faecal microbiota transplantation remains experimental in neurology. Evidence is heterogeneous and often small-scale, so translation should be cautious.

For practice and policy, low-risk dietary optimisation is justified, while probiotic use should follow strain-specific stewardship. Looking ahead, precision-nutrition and data-driven decision support may help match diets and strains to patient subgroups, but require transparent models, robust validation and privacy-preserving data governance. Next steps include adequately powered, preregistered trials with harmonised microbiome pipelines and mechanistic endpoints, comparative evaluations of digital tools, and equitable implementation so that microbiome-informed care enhances (rather than widens) access and outcomes.

No conflicts of interest to declare.

Figure and table credits. All figures and Table 1 are original and created by the authors.

Data availability. No new data were generated. All data supporting this review are contained within the article and its supplementary materials (Supplement S1–S4; PRISMA CCV available on request)

REFERENCES

1. Agus, A., Planchais, J., & Sokol, H. (2018). Gut microbiota regulation of tryptophan metabolism in health and disease. *Cell Host & Microbe, 23*(6), 716–724. https://doi.org/10.1016/j.chom.2018.05.003
2. Alli, S. R., Gorbovskaya, I., Liu, J. C. W., et al. (2022). The gut microbiome in depression and potential benefit of prebiotics, probiotics and synbiotics: A systematic review of clinical trials and observational studies. *International Journal of Molecular Sciences, 23*(9), 4494. https://doi.org/10.3390/ijms23094494
3. Cadilhac, D. A., & Mahal, A. (2024). Costs of neurological disorders. *Neuroepidemiology, 58*(6), 409–411. https://doi.org/10.1159/000539370
4. Camacho, M., Macleod, A. D., Maple-Grødem, J., et al. (2021). Early constipation predicts faster dementia onset in Parkinson's disease. NPJ Parkinson's Disease, 7(1), 45. https://doi.org/10.1038/s41531-021-00191-w
5. Cammarota, G., Ianiro, G., Tilg, H., Rajilić-Stojanović, M., Kump, P., Satokari, R., … Malfertheiner, P. (2017). European consensus conference on faecal microbiota transplantation in clinical practice. *Gut, 66*(4), 569–580. https://doi.org/10.1136/gutjnl-2016-313017
6. Chandra, S., Sisodia, S. S., & Vassar, R. (2023). The gut microbiome in Alzheimer's disease: What we know and what remains to be explored. *Molecular Neurodegeneration, 18*(1), 9. https://doi.org/10.1186/s13024-023-00595-7
7. Choi, J. G., Kim, N., Ju, I. G., et al. (2018). Oral administration of *Proteus mirabilis* damages dopaminergic neurons and motor functions in mice. *Scientific Reports, 8*(1), 1275. https://doi.org/10.1038/s41598-018-19646-x
8. Cryan, J. F., O'Riordan, K. J., Cowan, C. S. M., et al. (2019). The microbiota–gut–brain axis. *Physiological Reviews, 99*(4), 1877–2013. https://doi.org/10.1152/physrev.00018.2018
9. David, L. A., Maurice, C. F., Carmody, R. N., et al. (2014). Diet rapidly and reproducibly alters the human gut microbiome. *Nature, 505*(7484), 559–563. https://doi.org/10.1038/nature12820
10. Dinan, T. G., & Cryan, J. F. (2017). Brain–gut–microbiota axis and mental health. *Psychosomatic Medicine, 79*(8), 920–926. https://doi.org/10.1097/PSY.0000000000000519

11. Duan, W. X., Wang, F., Liu, J. Y., et al. (2024). Relationship between short-chain fatty acids and Parkinson's disease: A review from pathology to clinic. *Neuroscience Bulletin, 40*(4), 500–516. https://doi.org/10.1007/s12264-023-01123-9
12. U.S. Food and Drug Administration. (2019, June 13). *Fecal microbiota for transplantation: Safety communication—Risk of serious adverse reactions due to transmission of multidrug-resistant organisms.* https://www.fda.gov/safety/medical-product-safety-information/fecal-microbiota-transplantation-safety-communication-risk-serious-adverse-reactions-due
13. U.S. Food and Drug Administration. (2020, March 12). *Fecal microbiota for transplantation: Safety alert—Risk of serious adverse events likely due to transmission of pathogenic organisms.* https://www.fda.gov/safety/medical-product-safety-information/fecal-microbiota-transplantation-safety-alert-risk-serious-adverse-events-likely-due-transmission
14. Frej-Mądrzak, M., Gębarowska, J., Choroszy-Król, I., i wsp. (2021). Wpływ mikrobioty jelitowej na występowanie zaburzeń psychicznych oraz chorób neurodegeneracyjnych. *Postępy Higieny i Medycyny Doświadczalnej, 75*, 620–633. https://doi.org/10.2478/ahem-2021-0021
15. Fung, T. C., Olson, C. A., & Hsiao, E. Y. (2017). Interactions between the microbiota, immune and nervous systems in health and disease. *Nature Neuroscience, 20*(2), 145–155. https://doi.org/10.1038/nn.4476
16. Góralczyk-Bińkowska, A., Szmajda-Krygier, D., & Kozłowska, E. (2022). The microbiota–gut–brain axis in psychiatric disorders. *International Journal of Molecular Sciences, 23*(19), 11245. https://doi.org/10.3390/ijms231911245
17. Hsiao, E. Y., McBride, S. W., Hsien, S., et al. (2013). Microbiota modulate behavioral and physiological abnormalities associated with neurodevelopmental disorders. *Cell, 155*(7), 1451–1463. https://doi.org/10.1016/j.cell.2013.11.024
18. Jangi, S., Gandhi, R., Cox, L. M., et al. (2016). Alterations of the human gut microbiome in multiple sclerosis. *Nature Communications, 7*, 12015. https://doi.org/10.1038/ncomms12015
19. Jiang, X., Zheng, Y., Sun, H., et al. (2025). Fecal microbiota transplantation improves cognitive function of a mouse model of Alzheimer's disease. *CNS Neuroscience & Therapeutics, 31*(2), e70259. https://doi.org/10.1111/cns.70259
20. Kelly, J. R., Borre, Y., O'Brien, C. E., et al. (2016). Transferring the blues: Depression-associated gut microbiota induces neurobehavioral changes in the rat. *Journal of Psychiatric Research, 82*, 109–118. https://doi.org/10.1016/j.jpsychires.2016.07.019
21. Kochalska, K., Oakden, W., Słowik, T., et al. (2020). Dietary supplementation with *Lactobacillus rhamnosus* JB-1 restores brain neurochemical balance and mitigates the progression of mood disorder in a rat model of chronic unpredictable mild stress. *Nutrition Research, 82*, 44–57. https://doi.org/10.1016/j.nutres.2020.06.019
22. Kwon, D., Zhang, K., Paul, K. C., *et al.* (2024). Diet and the gut microbiome in patients with Parkinson's disease. *NPJ Parkinson's Disease, 10*(1), 89. https://doi.org/10.1038/s41531-024-00681-7
23. Li, S., Zhuo, M., Huang, X., et al. (2020). Altered gut microbiota associated with symptom severity in schizophrenia. *PeerJ, 8*, e9574. https://doi.org/10.7717/peerj.9574
24. Liu, X., Cruz Rivera, S., Moher, D., Calvert, M. J., Denniston, A. K., & SPIRIT-AI and CONSORT-AI Working Group. (2020). CONSORT-AI extension: Reporting guidelines for clinical trials evaluating artificial-intelligence interventions. *Nature Medicine, 26*, 1364–1374. https://doi.org/10.1038/s41591-020-1034-x
25. Liu, R. T., Lieberman, A. F., Musci, R. J., et al. (2022). Psychobiotics for mental health: A systematic review and meta-analysis. *Critical Reviews in Food Science and Nutrition, 62*(3), 630–652. https://doi.org/10.1080/10408398.2020.1790920
26. Li, Z., Lai, J., Zhang, P., et al. (2022). Multi-omics analyses of serum metabolome, gut microbiome and brain function reveal dysregulated microbiota–gut–brain axis in bipolar depression. *Molecular Psychiatry, 27*(10), 4123–4135. https://doi.org/10.1038/s41380-022-01569-9
27. Lorenc, J., Bramorska, A., Zarzycka, W., et al. (2020). Wpływ mikrobiomu jelitowego na mózg i psychikę. *Kosmos, 69*(1), 45–58. https://doi.org/10.36921/kos.2020_263
28. Maier, L., Pruteanu, M., Koepsell, S., et al. (2018). Extensive impact of non-antibiotic drugs on human gut bacteria. *Nature, 555*(7698), 623–628. https://doi.org/10.1038/nature25979
29. Mirzayi, C., Renson, A., Zohra, F., Elsafoury, S., Geistlinger, L., Kasselman, L. J., … The STORMS Consortium. (2021). Reporting guidelines for human microbiome research: The STORMS checklist. *Nature Medicine, 27*(11), 1885–1892. https://doi.org/10.1038/s41591-021-01552-x
30. Modesto-Lowe, V., Chaplin, M., & Sgambato, D. (2023). Major depressive disorder and the gut microbiome: What is the link? *General Psychiatry, 36*(1), e100973. https://doi.org/10.1136/gpsych-2022-100973
31. Nassar, S. T., Tasha, T., Desai, A., et al. (2022). Fecal microbiota transplantation in the treatment of Alzheimer's disease: A systematic review. *Cureus, 14*(10), e29968. https://doi.org/10.7759/cureus.29968
32. Nguyen, T. T., Kosciolek, T., Eyler, L., et al. (2019). Fecal microbiota signatures of metabolic risk in schizophrenia. *Molecular Psychiatry, 24*(3), 1–9. https://doi.org/10.1038/s41380-019-0536-x

33. Otaru, N., Ye, K., Mujezinovic, D., et al. (2021). GABA production by human intestinal *Bacteroides* spp.: Prevalence, regulation, and role in acid stress tolerance. *Frontiers in Microbiology, 12*, 656895. https://doi.org/10.3389/fmicb.2021.656895
34. Park, S. H., Lee, J. H., Shin, J., et al. (2021). Cognitive function improvement after fecal microbiota transplantation in an Alzheimer's dementia patient: A case report. *Current Medical Research and Opinion, 37*(10), 1739–1744. https://doi.org/10.1080/03007995.2021.1957807
35. Peery, A. F., Kelly, C. R., et al. (2024). AGA clinical practice guideline on fecal microbiota–based therapies for gastrointestinal disease. *Gastroenterology*. Advance online publication. https://www.gastrojournal.org/
36. Porcari, S., et al. (2024). International consensus statement on microbiome testing in clinical practice. *The Lancet Gastroenterology & Hepatology*. https://doi.org/10.1016/S2468-1253(24)00311-X
37. Rivera, S. C., Liu, X., Chan, A.-W., Denniston, A. K., Calvert, M. J., & SPIRIT-AI and CONSORT-AI Working Group. (2020). Guidelines for clinical trial protocols for interventions involving artificial intelligence: The SPIRIT-AI extension. *BMJ, 370*, m3210. https://doi.org/10.1136/bmj.m3210
38. Salim, S., Ahmad, F., Banu, A., et al. (2023). Gut microbiome and Parkinson's disease: Perspective on pathogenesis and treatment. *Journal of Advanced Research, 50*, 83–105. https://doi.org/10.1016/j.jare.2022.10.013
39. Sarkar, A., Lehto, S. M., & Harty, S., et al. (2016). Psychobiotics and the manipulation of bacteria–gut–brain signals. *Trends in Neurosciences, 39*(11), 763–781. https://doi.org/10.1016/j.tins.2016.09.002
40. National Institute of Standards and Technology. (2024). *Evaluating the analytical performance of direct-to-consumer gut microbiome testing services using a standardized NIST human fecal material.* https://www.nist.gov/publications/evaluating-analytical-performance-direct-consumer-gut-microbiome-testing-services
41. Strandwitz, P. (2018). Neurotransmitter modulation by the gut microbiota. *Brain Research, 1693*, 128–133. https://doi.org/10.1016/j.brainres.2018.03.015
42. Szklany, K., Wopereis, H., de Waard, C., et al. (2020). Supplementation of dietary non-digestible oligosaccharides from birth onwards improves social and reduces anxiety-like behaviour in male BALB/c mice. *Nutritional Neuroscience, 23*(11), 896–910. https://doi.org/10.1080/1028415X.2019.1576362
43. Sánchez-Villegas, A., Galbete, C., Martinez-González, M. A., et al. (2013). Effect of a Mediterranean diet on the incidence of depression: Results from the PREDIMED trial. *BMC Medicine, 11*, 208. https://doi.org/10.1186/1741-7015-11-208
44. Tankou, S. K., Regev, K., Healy, B. C., et al. (2018). Disease-modifying therapies alter gut microbial composition in multiple sclerosis. *Annals of Neurology, 83*(6), 797–811. https://doi.org/10.1002/ana.25244
45. Vascellari, S., Palmas, V., Melis, M., et al. (2020). Gut microbiota and metabolome alterations associated with Parkinson's disease. *mSystems, 5*(5), e00561-20. https://doi.org/10.1128/mSystems.00561-20
46. Vogt, N. M., Kerby, R. L., Dill-McFarland, K. A., et al. (2017). Gut microbiome alterations in Alzheimer's disease. *Scientific Reports, 7*, 13537. https://doi.org/10.1038/s41598-017-13601-y
47. Vrijsen, S., Houdou, M., Cascalho, A., et al. (2023). Polyamines in Parkinson's disease: Balancing between neurotoxicity and neuroprotection. *Annual Review of Biochemistry, 92*, 435–464. https://doi.org/10.1146/annurev-biochem-071322-021330
48. Wilkinson, M. D., Dumontier, M., Aalbersberg, I. J., Appleton, G., Axton, M., Baak, A., … Mons, B. (2016). The FAIR guiding principles for scientific data management and stewardship. *Scientific Data, 3*, 160018. https://doi.org/10.1038/sdata.2016.18
49. Zajac, D. J., Shaw, B. C., Braun, D. J., et al. (2022). Exogenous short chain fatty acid effects in APP/PS1 mice. *Frontiers in Neuroscience, 16*, 873549. https://doi.org/10.3389/fnins.2022.873549
50. Zeng, B., Li, G, Yuan, J., et al. (2018). The microbial community of the human gut is altered in schizophrenia. *Scientific Reports, 8*(1), 15775. https://doi.org/10.1038/s41598-018-34035-0
51. Zhao, Z., Ning, J., Bao, X.-Q., et al. (2021). Fecal microbiota transplantation protects rotenone-induced Parkinson's disease mice via suppressing inflammation mediated by the lipopolysaccharide–TLR4 signalling pathway through the microbiota–gut–brain axis. *Microbiome, 9*(1), 226. https://doi.org/10.1186/s40168-021-01107-9