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Gut Microbial Metabolites in Inflammatory, Metabolic, and Neurologic Disorders

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Abstract

The human gut microbiota transforms dietary and host-derived substrates into a diverse array of metabolites – including short-chain fatty acids (SCFAs), hydrogen sulfide (H_2S), bile acid derivatives, indoles, trimethylamine N-oxide (TMAO), branched-chain amino acid (BCAA) derivatives, and lipopolysaccharide (LPS) – that act as key signaling mediators along epithelial, immune, endocrine, and neural axes. These metabolites regulate barrier integrity, energy homeostasis, inflammation, and gut–brain communication and are increasingly implicated in the pathogenesis of inflammatory bowel disease (IBD), obesity, type 2 diabetes, metabolic dysfunction–associated steatotic liver disease (MASLD), and central nervous system (CNS) disorders. SCFAs, particularly butyrate, support colonocyte metabolism, reinforce tight junctions, promote regulatory T cells, and modulate enteroendocrine hormone release, whereas dysbiosis-associated depletion of SCFA-producing taxa correlates with barrier failure and heightened inflammation in IBD, metabolic disease, and neuroinflammatory conditions. Microbiota-derived H_2S exhibits dose-dependent effects, contributing to mucosal defense at physiological levels but impairing butyrate oxidation, damaging the epithelium, and worsening metabolic control when produced in excess. Microbial conversion of primary to secondary bile acids shapes FXR/TGR5 signaling, lipid and glucose metabolism, innate immunity, and microbial ecology, while perturbations in bile acid pools are linked to MASLD, insulin resistance, and IBD. In the microbiota–gut–brain axis, SCFAs and tryptophan catabolites influence microglial maturation, neuroinflammation, and neuroplasticity, with reduced SCFA levels and altered indole profiles described in Parkinson's disease, multiple sclerosis, and mood disorders. Emerging therapeutic strategies target these metabolite pathways using high-fiber and prebiotic diets, SCFA-producing probiotics, fecal microbiota transplantation, bile acid receptor modulators, and approaches to modulate H_2S and other metabolites, though current human evidence is largely associative and heterogeneous. Future work integrating multi-omics with longitudinal and interventional designs, alongside efforts toward standardization and personalization, will be essential to establish causality and safely harness microbial metabolites as precision targets in inflammatory, metabolic, and neurologic disorders.

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INTRODUCTION

The human gut harbors a dense and metabolically active microbial ecosystem that converts dietary and host-derived substrates into metabolites such as short-chain fatty acids (SCFAs), hydrogen sulfide (H_2S), and primary and secondary bile acids [1–4, 5, 6, 7]. These metabolites signal via epithelial, immune, endocrine, and neural pathways to regulate barrier integrity, energy homeostasis, inflammation, and gut–brain communication [2–4, 8, 9, 10]. Dysbiosis-associated shifts in microbial metabolite profiles are increasingly linked to inflammatory bowel disease

(IBD), obesity, type 2 diabetes, metabolic dysfunction–associated steatotic liver disease (MASLD), and central nervous system (CNS) disorders.

MAJOR CLASSES OF GUT MICROBIAL METABOLITES

Short-Chain Fatty Acids

SCFAs (mainly acetate, propionate, butyrate) are produced by anaerobic fermentation of dietary fibers by gut bacteria, especially Firmicutes such as *Faecalibacterium* and *Roseburia*. They act via GPR41/FFAR3, GPR43/FFAR2, and GPR109A and by inhibiting histone deacetylases to regulate epithelial energy supply, tight junctions, mucosal immunity, GLP-1 and PYY secretion, and systemic metabolism. Butyrate is the preferred energy source for colonocytes and has particularly strong barrier-protective and anti-inflammatory properties.

Hydrogen Sulfide

H_2S is generated in the gut by sulfate-reducing bacteria (e.g., *Desulfovibrio*, *Bilophila*) and by host enzymes. At physiological levels, H_2S regulates vascular tone, smooth muscle contractility, and mucosal defense, whereas excessive concentrations can inhibit colonocyte respiration, impair butyrate oxidation, and damage the epithelial barrier [3, 11, 12, 13, 14–16]. Recent work indicates that microbiota-derived H_2S can impair glucose homeostasis by suppressing GLP-1 secretion from enteroendocrine cells in high-fat-diet models.

Bile Acid Derivatives

Primary bile acids, synthesized from cholesterol, are secreted into the intestine and then deconjugated and transformed into secondary bile acids by microbial bile salt hydrolases and 7α-dehydroxylases. These bile acid pools signal via FXR, TGR5, and other receptors to influence lipid and glucose metabolism, intestinal barrier function, innate immunity, and enteroendocrine hormone release. Bile acids also exert direct antimicrobial effects that shape gut microbial community structure.

Other Relevant Metabolites

Other microbiota-derived metabolites implicated in health and disease include tryptophan catabolites (indoles, kynurenines), branched-chain amino acid (BCAA) derivatives, trimethylamine N-oxide (TMAO), and lipopolysaccharide (LPS). Indole and its derivatives can signal through the aryl hydrocarbon receptor (AHR) to modulate epithelial barrier and immune functions, whereas LPS from Gram-negative bacteria activates TLR4 and promotes low-grade systemic inflammation and insulin resistance (Table 1).

Table 1. Major gut microbial metabolites and their sources.

Metabolite class	Representative metabolites	Main microbial sources	Primary substrates	Key host targets/functions
Short-chain fatty acids	Acetate, propionate, butyrate	Firmicutes such as *Faecalibacterium*, *Roseburia*, and *Subdoligranulum*	Dietary fibers, resistant starch	Colonocyte fuel, GPCR signaling, HDAC inhibition, barrier support, immune modulation, GLP-1/PYY release.
Hydrogen sulfide	H_2S	Sulfate-reducing bacteria (*Desulfovibrio*, *Bilophila*)	Sulfate, sulfur, and amino acids	Control of motility, blood flow, barrier; context-dependent immune effects; at high levels, epithelial toxicity [3, 11, 14–16, 17].
Bile acid derivatives	Deconjugated and secondary bile acids (DCA, LCA)	BSH- and 7α-dehydroxylase-positive bacteria across major phyla	Primary bile acids	FXR/TGR5 signaling, lipid and glucose metabolism, antimicrobial activity, innate immunity.
Tryptophan metabolites	Indole, indole-3-propionic acid, indole-3-acetic acid	Multiple anaerobes	Dietary tryptophan	AHR signaling, epithelial protection, and anti-inflammatory effects.
LPS and others	LPS, TMAO, BCAA derivatives	Gram-negative bacteria; microbes metabolizing choline, carnitine, BCAA	Host and dietary lipids, choline, carnitine, BCAA	TLR4-mediated inflammation, atherogenesis, and insulin resistance.

MICROBIAL METABOLITES AND INTESTINAL HOMEOSTASIS

SCFAs, especially butyrate, enhance tight junction protein expression, stimulate mucus production, and provide energy to colonocytes, thereby maintaining epithelial barrier integrity. They also promote regulatory T cell differentiation, modulate Th17 responses, and influence innate lymphoid cells, supporting mucosal immune tolerance.

At physiological concentrations, H_2S can support mucosal blood flow, stimulate epithelial repair, and act as a gaseous signaling molecule [3, 11, 14–16]. However, excessive H_2S inhibits cytochrome c oxidase, impairs butyrate oxidation, and destabilizes the barrier, particularly in colitis [13]. Bile acids modulate antimicrobial peptide expression and interact with FXR/TGR5 in intestinal epithelial and immune cells, integrating signals from diet, host metabolism, and microbiota (Table 2).

Table 2. Mechanistic roles of key microbial metabolites in gut homeostasis.

Metabolite	Barrier integrity	Immune modulation	Motility and secretion	Microbiota composition
Butyrate	Fuels colonocytes, upregulates tight junctions, and enhances mucus	Promotes Treg differentiation, restrains pro-inflammatory cytokines via HDAC inhibition	Modulates motility and fluid transport via GPCRs	Supports beneficial anaerobes, suppresses pathogens.
Acetate/propionate	Support barrier and modest tight junction enhancement	Affect neutrophils, macrophages, and Th cells	Influence GLP-1 and PYY release, impacting transit	Act as substrates for cross-feeding, shaping communities.
H_2S	Low levels support mucosal blood flow and repair.	Dual pro-/anti-inflammatory effects depending on dose and context.	Regulates smooth muscle contraction and motility	H_2S donors can prevent NSAID-induced dysbiosis in models.
Secondary bile acids	Maintain barrier partly via FXR/TGR5 and tight junction regulation	Modulate innate immune receptors and inflammasomes	Influence bile flow and intestinal secretion via TGR5	Select for bile-tolerant species and inhibit others.

MICROBIAL METABOLITES IN INFLAMMATORY BOWEL DISEASE

IBD is associated with reduced microbial diversity, depletion of SCFA-producing taxa, and altered bile acid and tryptophan metabolite profiles. Lower fecal butyrate and reduced abundance of *Faecalibacterium prausnitzii* and other butyrate producers correlate with greater mucosal inflammation and compromised barrier function in IBD. Patients with IBD also show disrupted primary-to-secondary bile acid conversion, resulting in altered FXR/TGR5 signaling and dysregulated innate immunity.

In ulcerative colitis, increased sulfate-reducing bacteria and elevated luminal H_2S have been reported, suggesting that excessive H_2S contributes to epithelial damage and perpetuates colitis [3, 11, 14–16, 18]. Protective indole derivatives tend to be decreased, weakening AHR-mediated epithelial defense and anti-inflammatory pathways.

MICROBIAL METABOLITES IN METABOLIC DISORDERS

Metabolic disorders, such as obesity, insulin resistance, type 2 diabetes, and MASLD, are associated with alterations in gut microbiota composition and function, including deranged metabolite production. Dysmetabolic states often feature reduced SCFA-producing bacteria and altered SCFA levels, while SCFA supplementation in rodents improves glucose tolerance, energy expenditure, and adiposity [19]. MASLD is linked to decreased richness and depletion of genera, like *Faecalibacterium* and *Roseburia*, accompanied by SCFA and bile acid shifts that favor hepatic steatosis and inflammation (Table 3).

Table 3. Altered microbial metabolites in IBD.

Metabolite group	Direction of change	Microbial alterations	Putative consequences
SCFAs (esp. butyrate)	Decreased fecal levels	Reduced *Faecalibacterium*, *Roseburia*, and other butyrate producers	Impaired barrier, reduced Treg induction, heightened mucosal inflammation.
Secondary bile acids	Decreased or imbalanced	Loss of BSH/7α-dehydroxylase-positive taxa	Disturbed FXR/TGR5 signaling, altered innate immunity, and barrier function.
H_2S	Increased luminal levels in subsets (especially UC)	Enrichment of sulfate-reducing bacteria (*Desulfovibrio*)	Colonocyte toxicity, impaired butyrate oxidation, barrier disruption.
Tryptophan/indole metabolites	Decreased protective indoles	Reduced indole-producing commensals	Weaker AHR signaling, reduced epithelial defense, and anti-inflammatory tone.

Microbiota-derived H_2S has been shown to reduce GLP-1 secretion and worsen glucose homeostasis in high-fat-diet mice, implicating excessive H_2S in metabolic dysfunction. Other metabolites, such as BCAA derivatives and TMAO, also contribute to insulin resistance and cardiovascular risk (Table 4).

Table 4. Gut microbial metabolites and metabolic disorders.

Disorder	Key metabolite changes	Mechanisms implicated
Obesity	Reduced SCFA producers and altered SCFA levels	Decreased satiety hormones (GLP-1, PYY), low-grade inflammation, altered energy harvest.
Type 2 diabetes	Decreased butyrate/propionate and altered bile acids	Insulin resistance via LPS-driven inflammation; impaired incretin responses.
MASLD	Reduced *Faecalibacterium*, *Roseburia* and associated SCFAs; bile acid disturbances	Enhanced hepatic lipogenesis, inflammation, and fibrogenesis via gut–liver axis.
Metabolic syndrome with high H_2S	Increased H_2S-producing *Desulfovibrio* and luminal H_2S	Suppressed GLP-1 secretion, worsened glucose tolerance, and adiposity in models.

MICROBIAL METABOLITES AND NEUROLOGICAL DISEASES

The microbiota–gut–brain axis encompasses bidirectional communication between the gut and CNS via neural, endocrine, immune, and barrier mechanisms. SCFAs are key mediators in this axis; they can reach the brain at low concentrations and modulate microglial maturation, neuroinflammation, and neuroplasticity via GPCRs and epigenetic mechanisms. Butyrate is a potent HDAC inhibitor that influences gene expression in neurons and glial cells.

In Parkinson's disease (PD), decreased SCFA levels and reduced SCFA-producing bacteria have been reported, correlating with gastrointestinal dysmotility and possibly with α-synuclein pathology in the enteric nervous system. Multiple sclerosis (MS) cohorts show lower SCFA levels in feces and blood, and SCFA supplementation in experimental autoimmune encephalomyelitis reduces inflammation and demyelination. Emerging data link alterations in SCFAs and tryptophan metabolites with mood disorders and other CNS conditions [20].

THERAPEUTIC TARGETING OF MICROBIAL METABOLITES

Dietary, microbial, and pharmacological interventions that modulate microbial metabolites are being explored in IBD, metabolic, and neurological diseases [1, 2, 21]. High-fiber diets and prebiotics can increase SCFA production and improve metabolic and inflammatory parameters in animal models and early human trials. Probiotic formulations enriched in SCFA-producing strains and fecal microbiota transplantation (FMT) aim to restore beneficial metabolite profiles, though clinical results are heterogeneous (Table 5) [5, 6, 7, 8, 18, 22–26].

Table 5. Microbial metabolites in neurological diseases.

Condition	Metabolite alterations	Proposed CNS effects
Parkinson's disease	Reduced fecal and circulating SCFAs and SCFA producers	Enteric nervous system dysfunction, constipation, altered microglial activation, and neuroinflammation.
Multiple sclerosis	Lower acetate, propionate, butyrate in feces and serum	Impaired Treg induction, enhanced autoimmunity, and CNS demyelination.
Mood and stress-related disorders	Shifts in SCFAs and tryptophan metabolites	Changes in neurotransmission and neuroimmune signaling via gut–brain pathways.

Pharmacological strategies include bile acid receptor agonists/antagonists, SCFA or SCFA prodrug supplementation, and approaches to reduce excessive H_2S production such as targeting sulfate-reducing bacteria or using bismuth-containing compounds [3, 8, 11, 14–17]. Personalized approaches based on baseline microbiome and metabolome signatures may optimize responses and minimize adverse effects (Table 6).

Table 6. Metabolite-targeted interventions.

Approach	Primary target	Disease contexts	Evidence level
High-fiber/prebiotic diets	Increase SCFA production	IBD, obesity, T2D, MASLD, MS, PD	Animal and small human trials.
Probiotics (SCFA producers)	Restore butyrate and other SCFAs	IBD, MASLD, metabolic syndrome.	Preclinical and early clinical.
Fecal microbiota transplantation	Reestablish diverse SCFA and bile-transforming communities	Obesity, metabolic syndrome, IBD (selected)	Growing but mixed evidence.
Bile acid receptor modulators	Normalize FXR/TGR5 signaling	IBD, MASLD, T2D	Agents in clinical trials.
H_2S modulation	Reduce excessive microbial H_2S; preserve beneficial signaling	Metabolic syndrome, colitis models	Preclinical.
SCFA supplementation/prodrugs	Directly augment SCFAs	Obesity, insulin resistance, gut inflammation	Rodent and pilot human studies.

CHALLENGES, KNOWLEDGE GAPS, AND FUTURE DIRECTIONS

Most human data linking microbial metabolites to disease are associative, with limited longitudinal and interventional evidence for causality. Multi-omics approaches integrating metagenomics, metabolomics, transcriptomics, and clinical phenotypes are needed to dissect host–microbe–metabolite pathways. Inter-individual heterogeneity in microbiota composition and metabolite responsiveness will likely demand precision-medicine strategies with tailored diet, microbial, and drug interventions [1, 5, 6, 7, 8, 22–26].

Standardization of sampling, analytical pipelines, and reporting is required to compare metabolite data across studies. Long-term safety and off-target effects of bile acid and H_2S manipulation remain incompletely characterized [3, 5, 6, 7, 8, 11, 14–16]. Future priorities include mechanistic experiments linking specific microbes, defined metabolites, and host receptors to clinical outcomes in IBD, metabolic, and neurological diseases (Tables 7 and 8) [1–4, 5, 6, 7, 8, 10, 13, 18, 22–27].

Table 7. Key limitations and research priorities.

Area	Current limitation	Priority
Causality	Predominantly cross-sectional human data	Longitudinal and interventional studies with metabolite/receptor endpoints.
Metabolite coverage	Limited detection of low abundance/unstable metabolites	Advanced metabolomics and standardized pipelines.
Host–microbe signaling	Incomplete knowledge of cell-specific receptor networks.	Tissue-resolved receptor mapping and functional studies.
Personalization	Poorly defined microbiome "responders" vs "non-responders"	Stratification biomarkers and individualized algorithms.
Safety	Uncertain long-term safety of bile acid and H_2S modulation.	Chronic safety, off-target assessment, monitoring frameworks.

Table 8. Overview of roles of gut microbial metabolites in key disease groups.

Disease group	Dominant metabolite changes	Principal effects	Therapeutic angles
Inflammatory bowel disease	↓ SCFAs and indoles; altered bile acids; ↑ H_2S (subset)	Barrier failure, exaggerated immune activation, and mucosal injury	Fiber/prebiotics, SCFA-producing probiotics, bile acid, and H_2S modulation, FMT.
Metabolic disorders (obesity, T2D, MASLD, MetS)	↓ SCFA producers/SCFAs; altered bile acids; ↑ H_2S and other metabolites	Insulin resistance, steatosis, low-grade inflammation, reduced GLP-1	Diet, SCFAs, microbiota modulation, bile acid receptor drugs, H_2S lowering strategies.
Neurological diseases (PD, MS, mood disorders)	↓ SCFAs; altered tryptophan metabolites	Microglial dysregulation, impaired Treg function, neuroinflammation and ENS dysfunction	Diet and microbiota-based strategies to restore SCFAs and indoles.

CONCLUSION

Gut microbial metabolites constitute a central interface between environmental inputs, the intestinal ecosystem, and host physiology, and disturbances in their production or signaling emerge as shared mechanistic threads across IBD, metabolic disorders, and CNS diseases. Convergent evidence highlights SCFA depletion, disrupted bile acid transformation, excess or dysregulated H_2S, and imbalanced indole and other microbial products as key drivers of barrier breakdown, aberrant immune activation, metabolic dysfunction, and neuroinflammation. Interventions designed to restore a health-promoting metabolite milieu – through diet, live biotherapeutics, ecosystem-level strategies, such as fecal microbiota transplantation, and pharmacologic modulation of metabolite-responsive receptors – are promising but currently supported mainly by preclinical and early-phase clinical data. Major challenges include the predominantly cross-sectional nature of human studies, incomplete coverage of low-abundance and unstable metabolites, limited understanding of cell-specific receptor networks, and uncertain long-term safety of manipulating bile acid and H_2S pathways. Addressing these gaps will require standardized metabolomic pipelines, tissue-resolved mapping of host–microbe signaling circuits, and precision-medicine frameworks that account for inter-individual microbiome variability to identify responders and minimize off-target effects. Ultimately, linking defined microbes, specific metabolites, and their cognate receptors to clinical outcomes in well-controlled longitudinal and interventional studies will determine how best to translate microbiota-derived metabolite biology into safe and effective therapies for inflammatory, metabolic, and neurologic diseases.

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