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Narrative Review Study of Gut Microbiota and Nutrition Connection: An Implications for Human Health

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ABSTRACT: The human gut microbiota is a complex ecological community of bacteria, archaea, fungi, viruses and other microorganisms that interacts continuously with the host and dietary environment. Nutrition is among the most influential modifiable determinants of microbial composition and function. Dietary substrates that escape digestion, including fermentable fibers and selected resistant starches, support microbial metabolism and the production of short-chain fatty acids (SCFAs), while protein, fats, polyphenols and fermented foods can influence microbial pathways and host responses. Through metabolite generation, maintenance of epithelial barrier function, immune education, bile-acid transformation and gut–brain signaling, the microbiota contributes to gastrointestinal physiology and broader metabolic, immune and neurological processes. Alterations in microbial ecology have been associated with obesity, type 2 diabetes, cardiovascular disease, inflammatory bowel disease, colorectal cancer and neuropsychiatric conditions; however, associations do not invariably establish causality, and microbiome features vary markedly between individuals. This study summarizes the relationship between diet and gut microbial ecology, outlines major mechanisms connecting microbial activity with human health, evaluates food-based and biotic interventions, and discusses evidence limitations and emerging precision-nutrition approaches. A diverse, minimally processed dietary pattern rich in plant foods and fiber is a pragmatic strategy that may support microbial functions, whereas routine use of supplements should be individualized and evidence-based. Standardized methods, longitudinal studies and adequately powered clinical trials are needed to clarify causal pathways and translate microbiome research into reliable clinical guidance.

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1. INTRODUCTION

The gastrointestinal tract is an interface between food, the host and a large microbial ecosystem. The gut microbiota refers to the microorganisms inhabiting this environment, while the microbiome is often used to describe their collective genetic material and, more broadly, their ecological context. The community is not static: it develops across the lifespan and is shaped by early-life exposures, diet, geography, medications, disease, physical activity and other environmental factors. Its functions include the metabolism of otherwise indigestible dietary components, synthesis or transformation of bioactive molecules, resistance to colonization by pathogens and modulation of host immune responses [1, 2, 3]. Diet provides both substrates and selective pressures. A change in food intake can alter microbial activity within a short period, while habitual dietary patterns can contribute to longer-term ecological differences. The relationship is reciprocal: microorganisms transform food-derived compounds into metabolites that may influence intestinal and extraintestinal physiology. This bidirectional interaction provides a plausible framework for understanding how nutrition affects health beyond the classical supply of calories, essential nutrients and micronutrients [2, 4, 5].

Interest in microbiota-informed nutrition has expanded with advances in sequencing, metabolomics and controlled dietary studies. Nevertheless, translating findings into clinical practice requires caution. Microbial diversity alone is not a universally valid marker of health, and a tax on associated with a favorable outcome in one setting may behave differently in another. Inter-individual variability, methodological heterogeneity and confounding make it difficult to infer causality from observational studies [6, 7]. The purpose of this narrative study is to summarize the principal diet–microbiota interactions and their implications for human health, with emphasis on mechanisms, dietary strategies, clinical associations and future directions [2, 6]. Evidence from controlled feeding studies, observational cohorts and mechanistic experiments answers different questions. For example, a short-controlled feeding study demonstrated rapid microbial responses to contrasting animal- and plant-based diets, while intensive daily sampling showed that relationships between specific foods and the microbiome vary among individuals. Both types of evidence help interpret dietary responsiveness, but clinical trials with relevant host outcomes are needed to establish the health effects of interventions [5, 6, 7].

2. COMPOSITION AND FUNCTIONAL CHARACTERISTICS OF GUT MICROBIOTA

The intestinal microbial ecosystem is dominated by bacteria, particularly members of the phyla Bacillota (historically Firmicutes), Bacteroidota (historically Bacteroidetes), Actinomycetota and Pseudomonadota, although taxonomic proportions differ among individuals and populations. Common genera include Bacteroides, Prevotella, Faecalibacterium, Roseburia, Ruminococcus, Bifidobacterium and Akkermansia. These labels should not be interpreted as a fixed roster of beneficial or harmful organisms: effects are strain-, species-, niche- and context-dependent [1, 3]. Microbial functions are often more informative than simple taxonomic abundance. Multiple organisms can perform overlapping functions, a phenomenon described as functional redundancy. For example, several bacterial groups may ferment carbohydrates and produce acetate, propionate or butyrate. Conversely, organisms with similar taxonomic labels may differ in genes and metabolic capabilities. Functional assessment may therefore combine sequencing with metabolite measurements and clinical outcomes [3, 4].

Stool sequencing provides a practical way to sample the distal gut, although fecal material primarily represents the luminal community and cannot fully characterize mucosa-associated organisms or the small intestine. In addition, an increase in the relative abundance of a taxon may reflect a decline in other taxa. When the question concerns microbial activity, pairing metagenomics with measurements of metabolites or gene expression helps distinguish a community's functional potential from what it is doing at the time of sampling [3, 4, 6]. The microbial density and environment change along the gastrointestinal tract. The stomach and proximal small intestine have relatively lower microbial loads, partly due to acidity, bile and rapid transit. The colon provides a more favorable environment for dense communities, with substrates from dietary fiber, resistant starch, mucus and endogenous compounds. Oxygen gradients, pH, transit time and local nutrient availability create distinct ecological niches. A stable host–microbe relationship depends on the integrity of the mucosal barrier and balanced immune surveillance [3]. Microbiota composition is influenced by age, birth-related exposures, breastfeeding and complementary feeding in early life, habitual diet, geography, medications especially antibiotics—and disease status. These influences make it inappropriate to define a single ideal microbial profile for all populations. Functional resilience, appropriate metabolite production and absence of harmful activity may be more relevant than achieving a specific taxonomic composition [6].

Table: 1 selected gut microorganisms and functions commonly investigated in human health [3, 4, 8, 9].

Microbial group / example	Function or metabolite	Interpretive note
Bifidobacterium spp.	Carbohydrate fermentation; acetate and lactate production	Effects vary by species and strain; often responsive to selected prebiotic substrates.
Faecalibacterium prausnitzii	Butyrate production; immunomodulatory potential	Frequently studied in relation to intestinal homeostasis; abundance is not a stand-alone health marker.

Microbial group / example	Function or metabolite	Interpretive note
Roseburia spp.	Butyrate production from fermentable carbohydrates	Potentially relevant to epithelial energy and barrier functions.
Akkermansia muciniphila	Mucin-associated metabolism; host–microbe interface	Context-dependent; mucus dynamics and strain characteristics matter.
Bacteroides spp.	Complex carbohydrate degradation; bile-acid and other metabolic pathways	Broad genus with diverse and sometimes contrasting functions.
Lactobacillus / Lacticaseibacillus spp.	Lactate production; strain-specific probiotic effects	Health claims require evidence for the exact strain and indication.
Fusobacterium nucleatum	Investigated in colorectal tumor-associated microbial ecology	Association does not establish causality or individual cancer risk.

3. NUTRITION AS A DETERMINANT OF GUT MICROBIAL ECOLOGY

3.1 Dietary fiber and resistant starch

Dietary fibers comprise carbohydrate polymers that resist digestion and absorption in the small intestine. Their physicochemical properties including solubility, viscosity, fermentability and structure determine how they affect the gut. Fermentable fibers and resistant starches serve as substrates for microbial metabolism, supporting production of SCFAs and other metabolites. Sources include legumes, whole grains, vegetables, fruits, nuts, seeds and foods containing cooled or structurally resistant starch. Different fibers nourish distinct microbial guilds; therefore, diversity of fiber sources may provide a broader range of substrates than reliance on a single ingredient [4, 10]. The chemical and physical form of a fiber can influence the response. In a two-week intervention comparing potato resistant starch, maize resistant starch and chicory inulin, potato resistant starch produced the largest average increase in fecal butyrate, with substantial person-to-person variation.

Microbes that first degrade a starch can make smaller carbohydrates available to other organisms, including butyrate producers. Consequently, findings for one resistant starch preparation cannot be generalized to all starches or to all individuals [11, 12]. Fermentation is not uniformly beneficial in every clinical context. Rapidly fermentable carbohydrates may provoke gas, bloating or symptoms in individuals with irritable bowel syndrome or sensitivity to fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs). A temporary low-FODMAP approach may be useful under professional guidance, but prolonged restriction can reduce intake of some prebiotic substrates and should generally be followed by structured reintroduction [10, 13].

3.2 Protein and amino-acid metabolism

Dietary protein is digested and absorbed predominantly in the small intestine. Protein that reaches the colon is metabolized by microbes into compounds such as branched-chain fatty acids, ammonia, phenols, indoles and sulfur-containing metabolites. Some metabolites, including selected indole derivatives, can support epithelial and immune signaling; others may be harmful at high concentrations or in particular contexts. The effects depend on protein source, quantity, digestibility, accompanying fiber and microbial capacity. Diets high in animal protein and low in plant-derived fiber may shift colonic metabolism toward proteolytic fermentation, although the clinical significance varies and should not be simplified into a universal rule [4, 5]. Microbial tryptophan metabolism illustrates why the consequences of protein fermentation depend on the pathway. Gut microorganisms convert tryptophan into several indole derivatives, some of which interact with host signaling systems involved in epithelial and immune regulation. The metabolites and their effects vary with the microbes present, the substrates available and the host environment. Measuring total protein intake alone therefore yields limited information about exposure to individual microbial metabolites [4, 14].

3.3 Dietary fats

Fat intake affects the gut environment through bile secretion, intestinal transit and delivery of fatty acids. Bile acids are modified by microbial enzymes, generating secondary bile acids that participate in signaling through receptors such as FXR and TGR5. These pathways influence lipid and glucose metabolism, inflammation and energy homeostasis. High-fat dietary patterns, particularly when combined with low fiber and excess energy intake, have been associated with altered microbial profiles and bile-acid pools. Unsaturated fats from nuts, seeds, olive oil and fish can be incorporated into dietary patterns associated with favorable cardiometabolic outcomes, but effects on microbiota depend on the overall diet rather than an isolated nutrient [2, 4]. In a six-month randomized controlled-feeding trial, diets providing different proportions of energy from fat produced differences in gut microbial composition, fecal metabolites and selected cardiometabolic markers. The diets also differed in carbohydrate content, so these findings describe a macronutrient substitution within a specific feeding context. Interpreting changes in microbial features requires specifying which foods or nutrients replaced one another and whether a corresponding clinical outcome was measured [4, 15].

3.4 Polyphenols and plant bioactives

Polyphenols occur in berries, apples, tea, coffee, cocoa, herbs, vegetables and other plant foods. Their bioavailability is often limited by intestinal absorption, and gut microorganisms transform many polyphenols into smaller metabolites. Polyphenols may influence microbial growth and activity, while microbial metabolism can affect the biological activity of the compounds. Findings from human intervention studies suggest that responses are heterogeneous and depend on habitual intake, compound structure, dose and baseline microbial features. Whole foods remain preferable to assuming that concentrated extracts reproduce the effects of dietary patterns [4, 10]. Microbial metabolism can generate different products from the same polyphenol exposure. For example, some gut communities convert ellagitannins and ellagic acid to urolithins, but the resulting metabolite profiles differ across individuals. Detecting these compounds can help characterize microbial conversion of a dietary substrate. Establishing that a particular metabolite contributes to a health benefit requires additional evidence on exposure, biological activity and clinical outcomes [16].

3.5 Fermented foods

Fermented foods—including yogurt, kefir, selected fermented vegetables and other traditionally fermented products—may provide live microorganisms, fermentation products and altered food matrices. Fermentation can improve preservation, modify flavor and sometimes increase nutrient availability or reduce antinutritional factors. Some controlled dietary research suggests fermented-food intake can influence microbial diversity or immune markers, but products differ substantially in organisms, processing and viable counts. Not all fermented foods qualify as probiotics, because probiotic status requires defined microorganisms and demonstrated health benefits at an adequate dose [8, 17, 18]. In a randomized dietary intervention in healthy adults, the fermented-food group showed increasing microbial diversity and reductions in several circulating inflammatory markers. The high-fiber group showed changes in microbial carbohydrate-degrading capacity without an overall increase in diversity during the study. These results illustrate that dietary interventions may affect distinct microbial and immune measures. Whether the short-term changes persist or reduce disease risk remains to be established [17].

3.6 Dietary patterns

Dietary patterns integrate food combinations and are often more meaningful than single nutrients. A Mediterranean-style pattern typically emphasizes vegetables, fruits, legumes, whole grains, nuts, olive oil and fish, with limited highly processed foods. Plant-rich and high-fiber patterns may promote SCFA-producing microbial functions and lower inflammatory markers in some studies. Conversely, Western-style patterns characterized by excess energy, refined carbohydrates, low fiber and frequent ultra-processed foods may contribute to unfavorable metabolic and microbial changes. Evidence does not support attributing all health effects of a dietary pattern to microbiota alone; host metabolism, energy balance and other pathways also contribute [2, 4, 7]. An eight-week isocaloric Mediterranean diet intervention in adults with overweight or obesity reported lower plasma cholesterol alongside changes in the gut microbiome and metabolome. Keeping energy intake stable helped assess the effects of dietary composition. Because the intervention changed several foods together, the trial cannot assign the cholesterol effect to a single food or establish that microbial changes mediated it [19].

Table: 2 Dietary components and their potential influence on gut microbial activity [4, 10, 11, 18, 20]

Dietary component	Common food sources	Potential microbiota-related effects
Fermentable dietary fiber	Legumes, oats, whole grains, vegetables, fruits	Substrate for fermentation; may support SCFA production and microbial cross-feeding.
Resistant starch	Legumes, some whole grains, cooled cooked starches	Colonic fermentation; may support butyrate-producing pathways.
Inulin and FOS	Chicory root, onion, garlic, asparagus; some supplements	Can selectively support certain bifidobacteria; may cause gas in sensitive individuals.
GOS	Legumes and selected supplements	Prebiotic substrate with variable response and tolerance.
Polyphenols	Berries, tea, cocoa, coffee, herbs	Microbial transformation into metabolites; selective effects on microbial ecology.
Unsaturated fats	Olive oil, nuts, seeds, fish	Influence host lipid and bile-acid pathways; effects depend on dietary pattern.
Fermented foods	Yogurt, kefir, fermented vegetables	May provide live organisms and fermentation metabolites; products are heterogeneous.
Excess ultra-processed, low-fiber pattern	Many refined and highly processed foods	May reduce substrate diversity and coincide with unfavorable metabolic and microbial features.

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4. MICROBIAL METABOLITES AND MECHANISMS LINKING NUTRITION TO HEALTH

4.1 Short-chain fatty acids

SCFAs are principally acetate, propionate and butyrate, generated by microbial fermentation of carbohydrates and, to a lesser extent, other substrates. Acetate is produced by many organisms and can enter systemic circulation; propionate is largely associated with hepatic metabolism; and butyrate is an important energy source for colonocytes. SCFAs can affect epithelial function, pH, immune responses and signaling through G-protein-coupled receptors, including FFAR2 and FFAR3. Butyrate can also influence gene expression through histone deacetylase inhibition. These mechanisms provide biologically plausible links between fermentable fiber and host physiology, although the magnitude of clinical benefit depends on context [12].

SCFA production is shaped by substrate availability and microbial cross-feeding. One organism may generate acetate or lactate that another converts into butyrate. Consequently, microbial interactions and community structure may matter as much as the presence of a single species. Fecal SCFA concentration is not a straightforward measure of production because absorption and utilization also determine the amount excreted [12]. Interpretation of SCFA measurements also depends on the sampling site. Butyrate is extensively used by colonocytes, whereas absorbed acetate and propionate can undergo additional metabolism in other tissues, including the liver. Stool and blood measurements therefore represent different compartments. Studies that infer a pathway from fecal concentrations should account for transit time and sample handling and interpret the metabolite profile alongside measures of microbial function [6, 12].

4.2 Intestinal barrier and mucosal defense

The intestinal barrier includes epithelial cells, tight-junction proteins, mucus, antimicrobial peptides and immune components. It allows nutrient absorption while limiting exposure to potentially harmful organisms and molecules. Microbial metabolites can support epithelial energy metabolism and influence mucus production and tight-junction regulation. A disrupted barrier may increase translocation of microbial products and contribute to local or systemic inflammation, but the concept of “leaky gut” should not be used as a diagnosis without appropriate clinical evaluation [3, 12]. Commensal microorganisms also contribute to colonization resistance by competing for nutrients and attachment sites, producing inhibitory compounds and shaping immune responses. Antibiotics and other disturbances may weaken these ecological functions, facilitating overgrowth of opportunistic organisms. Nutritional support of microbial ecology is one potential component of maintaining resilience, although it cannot replace indicated infection prevention or treatment [3, 4].

4.3 Immune regulation and inflammation

The microbiota participates in the development and regulation of innate and adaptive immunity. Microbial-associated molecular patterns interact with host receptors, while metabolites can influence regulatory T cells, cytokine production and immune-cell differentiation. SCFAs have been associated with regulatory pathways that can temper excessive inflammation, although their effects may vary with concentration, tissue and disease state. Diet can influence these processes by altering microbial substrates and metabolites, as well as through direct effects of vitamins, minerals and fatty acids [12, 21].

Mechanistic work describes routes by which SCFAs influence immune responses, including activation of G-protein-coupled receptors and inhibition of histone deacetylases. These pathways can affect epithelial and immune cell responses, with effects depending on the metabolite, concentration and tissue. Human studies that detect a change in an inflammatory marker after a dietary intervention can test whether these pathways are plausible in context, but a circulating marker alone does not demonstrate a specific mechanism or durable clinical benefit [12, 17].

4.4 Bile acids and metabolic signaling

Primary bile acids synthesized by the liver enter the intestine to facilitate fat digestion. Gut microbes deconjugate and transform bile acids into secondary forms. The bile-acid pool can signal through nuclear and membrane receptors involved in glucose regulation, lipid metabolism, energy expenditure and immune function. Diet and microbial activity influence the balance of these compounds. Some secondary bile acids have been associated with adverse outcomes, including potential carcinogenic effects in particular settings, while others participate in beneficial signaling. This illustrates why microbial metabolites cannot be classified uniformly as beneficial or harmful [4, 22].

4.5 Vitamins and nutrient bioavailability

Some gut microorganisms synthesize vitamins, including vitamin K and several B-group vitamins, and can modify minerals or food compounds in ways that affect bioavailability. However, microbial synthesis does not necessarily meet host requirements: production site, absorption, microbial utilization and dietary intake all matter. The gut microbiota should not be considered a substitute for a nutritionally adequate diet or clinically indicated supplementation [3, 4].

4.6 Gut–brain communication

The gut and brain communicate through neural, endocrine, immune and metabolic pathways. The vagus nerve, enteric

nervous system, hypothalamic–pituitary–adrenal axis, microbial metabolites and immune mediators may participate in this gut–brain axis. Microbes can metabolize tryptophan and produce compounds that influence host signaling, while diet may alter both substrate availability and microbial function. Associations between microbiota profiles and depression, anxiety, stress responses or cognition are increasingly reported, but findings remain heterogeneous. It is premature to claim that specific foods or probiotic products reliably treat psychiatric disorders; microbiome-related approaches should be considered investigational or adjunctive where appropriate [1, 21].

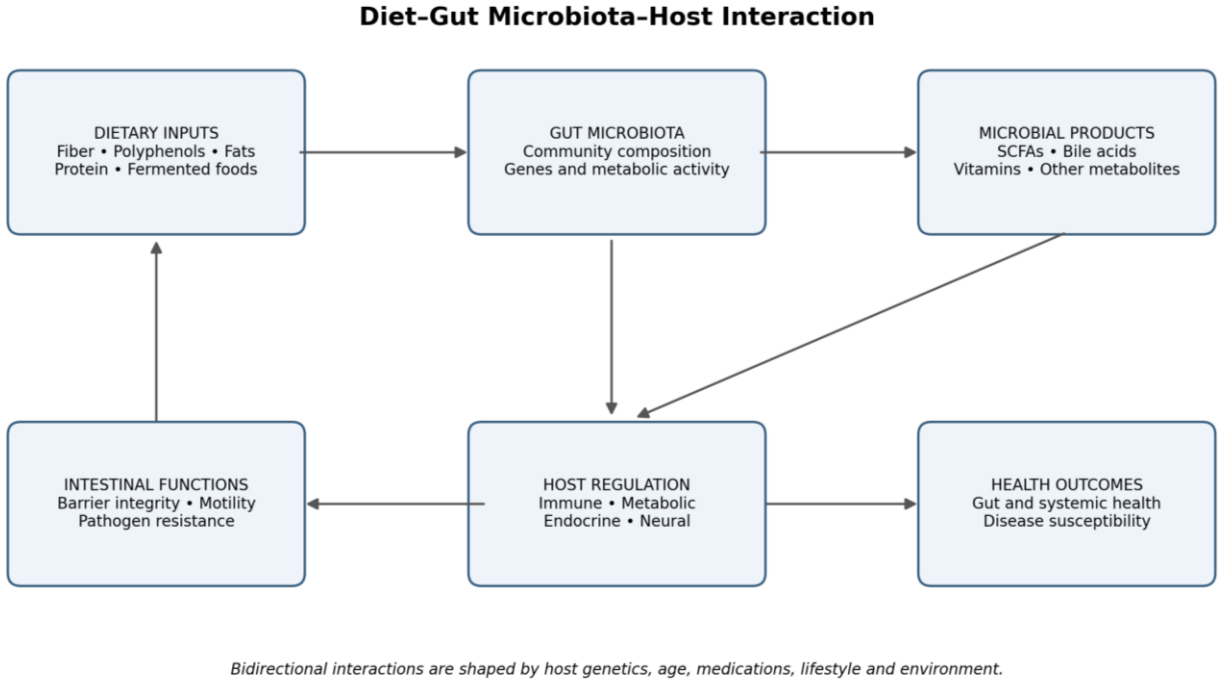


Figure 1. Conceptual overview of diet–gut microbiota–host interactions. Dietary substrates influence microbial composition and metabolism; microbial products and community functions affect intestinal and systemic host pathways. The relationship is bidirectional and modified by host and environmental factors [2, 4, 12, 21].

5. GUT MICROBIOTA AND HUMAN DISEASES

5.1 Obesity and metabolic syndrome

Obesity and metabolic syndrome involve energy balance, adipose tissue biology, insulin signaling, inflammation, genetics, behavior and environment. Gut microorganisms may influence these processes through energy harvest, SCFAs, bile-acid signaling, intestinal permeability and inflammatory pathways. Studies have reported associations between obesity and microbial features, but there is no single microbial signature consistently applicable to all individuals. Dietary interventions can modify microbiota and metabolic markers, yet the clinical effect is generally inseparable from changes in energy intake, weight and food quality. Microbiota modulation is therefore a plausible contributor, not a stand-alone explanation or treatment [1, 19].

5.2 Type 2 diabetes mellitus

Type 2 diabetes is associated with altered microbial composition and function in some populations, including changes in organisms involved in carbohydrate fermentation and bile-acid metabolism. Mechanistic hypotheses include effects on low-grade inflammation, intestinal barrier integrity, incretin signaling and SCFA production. Metformin itself can alter the microbiota, complicating interpretation of case-control studies. Fiber-rich dietary patterns may support glycemic management through several mechanisms, including slower carbohydrate absorption and satiety, alongside microbiota-derived effects. Clinical diabetes care remains based on evidence-based nutrition, physical activity, medication and monitoring rather than microbiome testing alone [1, 23]. A randomized study in adults with type 2 diabetes found that a high-fiber dietary intervention promoted a subset of SCFA-producing bacteria. Greater representation of these organisms was associated with greater improvement in glycated hemoglobin. A separate study in treatment-naive adults showed that starting metformin altered the gut microbiome. Drug exposure should therefore be recorded when comparing microbiomes in diabetes, especially when interpreting microbial signals as possible mediators of diet or markers of disease severity [23, 24].

5.3 Cardiovascular disease

Microbial metabolism of dietary choline, phosphatidylcholine and carnitine can produce trimethylamine, which the liver converts to trimethylamine-N-oxide (TMAO). Elevated TMAO has been associated with cardiovascular risk in several observational studies, but causality and the influence of renal function, diet and other confounders remain debated. Gut microbial transformation of bile acids and production of SCFAs may also influence blood pressure, lipid metabolism and vascular inflammation. A heart-healthy dietary pattern is recommended based on established clinical evidence; microbiome mechanisms may help explain part of its effects but do not yet justify routine TMAO-based management [25]. In a human phosphatidylcholine challenge, temporary suppression of intestinal microbes with antibiotics reduced TMAO generation, supporting a microbial contribution to its formation. In the same report, higher fasting TMAO concentrations were associated with subsequent major cardiovascular events in patients undergoing cardiac evaluation. The challenge and cohort findings address distinct questions. A trial would be needed to establish whether lowering TMAO itself reduces cardiovascular events [25, 26].

5.4 Inflammatory bowel disease

Crohn's disease and ulcerative colitis are chronic inflammatory disorders involving genetic susceptibility, immune dysregulation, environmental factors and microbiota. Dysbiosis, reduced abundance of selected butyrate-producing organisms and altered microbial metabolism have been observed, although changes may be consequences as well as drivers of inflammation. Nutritional care is important for maintaining energy and nutrient adequacy, managing symptoms and addressing deficiencies. Specific dietary therapies, including exclusive enteral nutrition in selected Crohn's disease settings, may be used under specialist supervision. Generalized microbiota-targeted diets should not replace established medical therapy [27]. A longitudinal multi-omics study of inflammatory bowel disease documented temporal changes in microbial composition and metabolic profiles. Repeated sampling helped capture variation that a single stool specimen would miss. In clinical cohorts, disease activity, treatment, dietary adaptation and intestinal transit can all influence the measured microbial profile. These influences complicate the use of microbial signatures as diagnostic or dietary response markers [27].

5.5 Irritable bowel syndrome

Irritable bowel syndrome is a disorder of gut–brain interaction characterized by abdominal pain and altered bowel habits. Microbiota findings are variable, and no validated microbial test reliably diagnoses IBS or selects treatment for all patients. Some patients benefit from a time-limited low-FODMAP diet followed by reintroduction and personalization. Fiber type matters: soluble fiber such as psyllium may help some patients, while abrupt increases in fermentable fiber can worsen bloating. Dietetic guidance can help maintain nutritional adequacy and avoid unnecessary restriction [13]. The American College of Gastroenterology guideline supports a limited trial of a diet low in fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) for global IBS symptoms. It also supports soluble fiber for symptom management. Symptom improvement is the clinically relevant outcome, and evidence for these strategies does not establish that changing microbial diversity mediates symptom relief [13].

5.6 Colorectal cancer

The gut microbiota may influence colorectal carcinogenesis through inflammation, genotoxic metabolites, bile-acid transformation and interactions with the mucosal immune system. Certain organisms, including enterotoxigenic Bacteroides fragilis and strains of Fusobacterium nucleatum, have been investigated in relation to tumor biology, but their presence is not sufficient to establish an individual's cancer risk. Dietary fiber, whole grains and plant foods are associated with lower colorectal cancer risk in epidemiologic evidence, with possible contributions from fermentation and stool transit. Screening and clinical prevention remain essential; microbiome-based cancer screening or treatment requires validation [9, 28]. A supervised two-week exchange of diets differing in fiber and fat among African American and rural South African adults altered microbial metabolites and colonic mucosal markers linked to cancer risk. Because the study measured short-term markers, it could not determine effects on cancer incidence. Separately, pooled analyses of fecal metagenomes identified microbial signatures associated with colorectal cancer across cohorts, supporting further validation of diagnostic performance in screening populations [9, 28].

5.7 Liver disease

The gut–liver axis links intestinal microbial products and bile acids with hepatic immune and metabolic activity through the portal circulation. Changes in barrier function and microbial metabolism have been associated with metabolic dysfunction-associated steatotic liver disease (MASLD), alcohol-related liver disease and cirrhosis. Diet, obesity, alcohol exposure and medications also influence these conditions. Microbiota-targeted interventions are being studied, but management should prioritize established measures such as weight management when indicated, dietary quality, alcohol-related risk reduction and medical care [22]. Portal circulation exposes the liver to intestinal products, while hepatic bile secretion shapes the intestinal environment. This reciprocal relationship can complicate interpretation of cross-sectional microbiome findings in

metabolic dysfunction-associated steatotic liver disease. Demonstrating a therapeutic effect of a microbiota-directed intervention requires measures of liver disease and sufficient follow-up to distinguish sustained clinical changes from short-term shifts in microbial profiles [22].

5.8 Neurological and mental health conditions

Research has explored microbial associations with Parkinson's disease, autism spectrum conditions, depression, anxiety and cognitive impairment. Potential pathways include inflammatory signaling, metabolites, vagal communication and altered tryptophan metabolism. Most evidence is associative, and reverse causality is plausible because neurological conditions can affect diet, activity, medication use and bowel function. At present, microbiota-directed nutrition is not a replacement for evidence-based neurological or psychiatric treatment [21].

Table: 3 selected disorders, microbiota-related mechanisms and nutritional considerations [1, 9, 13, 22, 24, 26, 27]

Condition	Potential microbiota-related pathways	Nutrition perspective / limitation
Obesity and metabolic syndrome	Energy harvest, SCFAs, bile-acid signaling, inflammation	Plant-rich dietary patterns and energy balance; no universal obesity microbiome signature.
Type 2 diabetes	SCFAs, barrier function, bile acids, incretin and inflammatory signaling	Fiber-rich foods support overall glycemic management; microbiome effects are one component.
Cardiovascular disease	TMAO formation, bile-acid signaling, SCFAs and inflammation	Heart-healthy dietary pattern has established benefits; microbiome biomarkers not routine clinical tools.
Inflammatory bowel disease	Dysbiosis, reduced selected SCFA pathways, mucosal immune interactions	Individualized nutrition and specialist care; dietary therapy does not replace medication.
Irritable bowel syndrome	Variable microbial patterns, fermentation and gut–brain signaling	Soluble fiber or supervised low-FODMAP approach may help selected patients.
Colorectal cancer	Microbial genotoxic metabolites, inflammation, bile-acid transformation	Fiber-rich foods and screening are important; microbial markers remain investigational.
MASLD / liver disease	Gut–liver axis, barrier disruption, bile-acid and inflammatory pathways	Dietary quality and metabolic risk management; microbiome-targeted therapies remain under study.

6. NUTRITIONAL STRATEGIES FOR SUPPORTING A HEALTHY GUT MICROBIOTA

6.1 Food-first, plant-diverse dietary patterns

A practical approach is to consume a broad variety of minimally processed plant foods, including vegetables, fruits, legumes, whole grains, nuts and seeds, while ensuring adequate protein and essential fats. Variety introduces diverse fibers and phytochemicals that can support multiple microbial pathways. Dietary changes should be gradual, particularly for people with low habitual fiber intake, to reduce discomfort. Adequate hydration is important when increasing fiber [2, 10].

6.2 Prebiotics

A prebiotic is a substrate selectively utilized by host microorganisms that confers a health benefit. Commonly studied prebiotic substrates include inulin, fructooligosaccharides (FOS), galactooligosaccharides (GOS) and certain resistant starches. They can increase the activity or abundance of selected microbes and may influence SCFA generation. Response depends on the substrate, dose, baseline microbiota and tolerance. Prebiotic supplements may cause gas or bloating and are not necessary for everyone; food sources can be incorporated into a balanced diet [10, 20]. Fermentability by itself is insufficient to classify a substrate as a prebiotic. The consensus definition requires selective microbial utilization and a demonstrated health benefit in the host. For example, an increase in bifidobacteria can support evidence that a substrate is selectively utilized, but assessment of a relevant health outcome is also needed to substantiate a prebiotic claim [20].

6.3 Probiotics

Probiotics are live microorganisms that, when administered in adequate amounts, confer a health benefit on the host. Evidence is strain-specific and indication-specific. A result obtained with one Lactobacillus or Bifidobacterium strain cannot be assumed to apply to another strain or a generic product. Some probiotic preparations have evidence for selected outcomes, such as reducing the risk or duration of certain forms of diarrhea, but evidence is inconsistent across indications. Product quality, viable count through expiry, storage and dose are relevant. Probiotics are generally well tolerated in healthy individuals, yet caution is appropriate in critically ill or severely immunocompromised patients, and medical advice should be sought in such contexts [8]. The American Gastroenterological Association guideline evaluates specific probiotic formulations by clinical indication. Its recommendations vary by condition, and it limits probiotic use for several gastrointestinal disorders to clinical trials. Interpreting a study requires knowing the strain or formulation, dose, treatment duration, patient population and outcome. Products that list only a genus or species provide too little information to determine whether a published result applies to them [8, 29].

6.4 Synbiotics and postbiotics

Synbiotics combine live microorganisms with substrates intended to be utilized by host microorganisms, with the goal of providing a health benefit. Postbiotics are preparations of inanimate microorganisms and/or their components that confer a health benefit. Definitions and product formulations have evolved, and clinical efficacy should be demonstrated for the specific preparation. Neither category should be presumed effective merely because it is marketed as microbiome-supportive [30, 31]. The synbiotic consensus distinguishes complementary products, which combine a probiotic and a prebiotic, from synergistic products, in which the substrate is designed to be selectively utilized by the co-administered microorganism. A postbiotic preparation contains inanimate microorganisms or their components and must confer a health benefit. Purified microbial metabolites on their own fall outside that definition. Evidence should correspond to the formulation and population for which a benefit is claimed [30, 31].

6.5 Fermented foods and dietary implementation

Fermented foods can be included according to culture, tolerance and food safety. Unsweetened yogurt or kefir, fermented vegetables and traditional fermented foods may contribute to dietary variety. Salt content, added sugars, pasteurization after fermentation and viable microbial content vary by product. A fermented food does not automatically have a proven therapeutic effect, and live cultures are not appropriate for every patient [17, 18].

6.6 Caution with restrictive diets and supplements

Highly restrictive diets can reduce nutritional adequacy and potentially narrow microbial substrate availability. Gluten-free diets are medically indicated for celiac disease and selected conditions, but unnecessary gluten avoidance is not an established microbiome-health strategy. Similarly, ketogenic, low-FODMAP or elimination diets may be indicated in specific circumstances but should be individualized and periodically reassessed. Commercial microbiome tests and personalized supplement plans often lack validated clinical utility; interpretation should not lead to treatment without evidence [13, 32].

7. CHALLENGES, EVIDENCE GAPS AND FUTURE PERSPECTIVES

A major challenge is the lack of a universally accepted definition of a healthy microbiome. High diversity is often discussed as favorable, but diversity metrics do not capture functional activity, and some healthy states may have relatively low diversity. The relevant features may depend on the host, age, disease, diet and the outcome being studied. Clinical translation should therefore focus on validated functional measures and health outcomes rather than simplistic labels such as “good” or “bad” bacteria [6, 7]. Study design is another limitation. Dietary assessment methods, intervention duration, sample collection, sequencing platforms, bioinformatic pipelines and statistical analyses vary widely. Stool samples do not fully represent microbial communities or metabolites along the gastrointestinal tract, and fecal metabolite concentrations may not reflect production or host exposure. Diet is also difficult to isolate from medications, physical activity, socioeconomic factors and other behaviors. Standardized reporting and reproducible analytical approaches are necessary for meaningful comparisons [6].

Researchers should report how diet was assessed, the timing of sample collection in relation to dietary exposure, storage and sequencing procedures, medication use and the statistical approach used for multiple comparisons. In intervention studies, documenting adherence and analyzing prespecified clinical outcomes alongside microbial measures helps identify whether apparent responses reflect the intended dietary change and whether they matter for health [6]. Longitudinal cohorts and randomized controlled trials can improve causal inference, particularly when they combine detailed dietary assessment with microbial sequencing, metagenomics, metabolomics and clinically meaningful endpoints. Trials should be adequately powered, preregistered where appropriate and transparent about adherence, adverse effects and analysis plans. Researchers should avoid overinterpreting exploratory taxa or subgroup findings and should distinguish microbial changes from actual health benefits [6].

Precision nutrition is an emerging concept in which dietary recommendations account for individual variation in physiology, microbiota, genetics, lifestyle and preferences. Some studies demonstrate heterogeneity in postprandial responses and microbial changes after similar dietary exposures. Nonetheless, microbiome-based personalization is not yet sufficiently validated for broad routine use. Future applications require external validation, cost-effectiveness assessment, accessible testing and evidence that personalized advice improves outcomes compared with standard dietary guidance [2, 6]. A proof-of-concept study used clinical and dietary variables together with microbiome data to predict postprandial glycemic responses and tested algorithm-guided dietary advice over a short period. Translation requires validation across populations and comparison with advice based on readily available predictors. Longer trials should assess whether adding stool profiling improves sustained outcomes, such as glycated hemoglobin or quality of life, enough to justify its cost and burden [6, 32, 33].

Other areas of investigation include targeted prebiotic substrates, defined microbial consortia, next-generation probiotics, postbiotics, microbial metabolite therapeutics and fecal microbiota-based interventions. These approaches may hold promise for specific diseases, but safety, product consistency, durability and long-term consequences need careful evaluation. Fecal microbiota transplantation, for example, has established use in selected recurrent Clostridioides difficile infection settings under regulated clinical practice; it is not a general wellness intervention and carries potential risks of

transmitting pathogens or other undesirable traits [2, 30]. Public health implementation should emphasize accessible dietary patterns rather than expensive supplements or commercial microbiome tests. Food systems, affordability, cultural preferences and local availability affect adherence. Nutrition guidance can be adapted to regional diets by including a variety of vegetables, fruits, pulses, whole grains, nuts and fermented foods as tolerated. Such advice should be integrated with established preventive and clinical nutrition recommendations [2, 4].

8. CLINICAL AND PUBLIC HEALTH IMPLICATIONS

For most generally healthy adults, a pragmatic microbiota-supportive approach is to consume a diverse diet rich in plant foods and fiber, limit excessive intake of highly processed foods, maintain adequate hydration, and include fermented foods when acceptable and safe. Fiber should be increased gradually, and recommendations should be individualized for gastrointestinal symptoms, chronic disease, allergies, swallowing difficulties or other clinical constraints. Supplements should be selected for a defined indication, with attention to evidence and product quality [2, 10, 18]. Healthcare professionals should communicate that the microbiota is one component of a complex biological system. A dietary change may alter microbial features without producing a clinically meaningful benefit, and a favorable clinical outcome may occur through pathways unrelated to microbiota.

Patients should be discouraged from stopping prescribed treatment or adopting restrictive diets based solely on commercial microbiome reports. Nutrition care is best delivered through shared decision-making and, when needed, qualified dietetic or medical supervision [6, 32]. An international consensus statement on microbiome testing identified limited evidence for routine clinical use and discouraged patient-requested testing without a clinical recommendation. Microbiome-based dietary advice needs standardized analytical methods, interpretable results and evidence that acting on those results improves patient outcomes. In routine care, dietary intake, symptoms, medication use and established risk markers can guide nutritional assessment [2, 32].

CONCLUSION

The gut microbiota is an important intermediary between nutrition and human physiology. Dietary fiber, resistant starch, polyphenols, fats, proteins and fermented foods influence microbial ecology and metabolic activity, while microbial products—including SCFAs and transformed bile acids participate in intestinal barrier function, immune regulation, metabolic signaling and gut–brain communication. Microbiota alterations have been linked with several chronic and gastrointestinal disorders, but causal relationships and therapeutic implications differ by condition and remain incompletely resolved. A varied, plant-rich, minimally processed dietary pattern is a reasonable food-first strategy that supports overall health and may foster beneficial microbial functions. Probiotics, prebiotics and other microbiota-targeted interventions should be used selectively, guided by strain- or product-specific evidence and individual context. Future progress depends on standardized research methods, robust clinical trials and validated precision-nutrition tools that demonstrate meaningful health benefits [2, 4, 6, 12].

Declarations

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Author contributions

The original author and the corresponding author’s supervisor prepared the initial drafts of the manuscript. Both contributed to data collection, edited the manuscript, designed the tables, and approved the final version for submission to the journal.

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