Advances in Obesity, Endocrinology, and Diabetes

Volume 3 Issue 2 (July-Dec 2026) | **ISSN**: 3049-0715

Gut Microbiota as a Determinant of Interindividual Variability in Response to Dietary Interventions: Mechanistic Insights and Clinical Implications

Running head: Microbiota and Dietary Response

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Funding

No external funding was received.

Conflict of Interest Statement

The authors declare no conflict of interest.

Ethics Statement

Ethical approval was not required because this work is a review based exclusively on published literature.

AI Use Disclosure

An AI-assisted language and editorial support tool was used to improve English style and manuscript organization. The authors reviewed the final text and took full responsibility for the content.

Abstract:

Introduction: Dietary interventions are central to the management of obesity, insulin resistance, type 2 diabetes and related cardiometabolic disorders, yet clinical responses vary substantially between individuals exposed to apparently similar nutritional prescriptions. The gut microbiota may explain part of this variability because it transforms dietary substrates into bioactive metabolites and influences intestinal, immune and endocrine pathways. Methods: A narrative review informed by a structured literature search was conducted using PubMed, ScienceDirect and Google Scholar. Search terms combined concepts related to gut microbiota, intestinal microbiota, dietary intervention, nutrition, fiber, prebiotics, probiotics,

metabolic response, obesity, insulin sensitivity and glycemic response. Forty records were initially identified, and 21 references were retained for qualitative synthesis after duplicate removal, screening and full-text eligibility assessment. Results: The reviewed literature indicates that baseline microbial diversity, community structure and functional capacity are associated with differential metabolic responses to calorie restriction, fiber-rich dietary patterns, probiotics, prebiotics and glycemia-oriented personalized nutrition. Key taxa and communities discussed in the evidence include Bacteroides, Prevotella, Akkermansia muciniphila, Bifidobacterium, Lactobacillus and Christensenella-associated communities. Mechanistic pathways include short-chain fatty acid generation, bile acid transformation, tryptophan-derived signaling, incretin-related pathways, epithelial barrier integrity and low-grade inflammation. Conclusion: The gut microbiota should be regarded as both a biomarker and a potential mediator of response to dietary interventions. Microbiota-informed nutrition is promising for precision diabetes and endocrinology care, but standardized prospective studies integrating microbiome profiling with robust metabolic outcomes remain necessary before routine clinical implementation.

Keywords:

Gut microbiota; personalized nutrition; dietary interventions; obesity; insulin resistance; type 2 diabetes; short-chain fatty acids; prebiotics; probiotics

Introduction:

Response heterogeneity remains a major limitation of dietary therapy in obesity, insulin resistance, type 2 diabetes and related metabolic disorders. Even when patients receive similar advice and report comparable adherence, clinical trajectories may differ markedly in terms of body weight, postprandial glycemia, insulin sensitivity, appetite regulation and inflammatory status. This observation suggests that treatment response is shaped by determinants beyond calorie intake or macronutrient distribution alone (1,2).

The gut microbiota is increasingly recognized as a dynamic interface between dietary exposure and host physiology. Intestinal microorganisms transform non-digestible dietary substrates, participate in bile acid metabolism, produce short-chain fatty acids and other signaling molecules, and interact with epithelial, immune and endocrine pathways (3,12,19). Because diet can remodel microbial communities within days while the pre-existing microbiota also influences nutrient processing, a reciprocal diet-microbiota-host axis offers a biologically plausible explanation for variable responses to nutritional interventions (3,4).

Interindividual variability in gut microbial composition is also shaped by host genetics, environmental exposure, geography, medication use and baseline metabolic status (2,11). These sources of variation may explain why a diet that improves glycemic control or promotes weight loss in one patient has a smaller or different effect in another. The objective of this review is to synthesize mechanistic and clinical evidence supporting the role of gut microbiota composition and function as determinants of dietary response, with emphasis on obesity, insulin resistance, type 2 diabetes and metabolic syndrome.

Methods:

This manuscript was developed as a narrative review informed by a structured literature search. PubMed, ScienceDirect and Google Scholar were searched using combinations of the following terms: “gut microbiota”, “intestinal microbiota”, “microbiome”, “dietary intervention”, “nutrition”, “diet”, “fiber”, “prebiotic”, “probiotic”, “metabolic response”, “glycemic response”, “insulin sensitivity”, “obesity” and

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"type 2 diabetes". English- and French-language publications were considered because the final evidence base included both international biomedical literature and relevant French reviews.

The search strategy was designed to identify publications addressing at least one of the following themes: (i) the association between microbiota composition or function and metabolic response to diet; (ii) mechanistic pathways linking microbial metabolites with glucose-lipid metabolism, appetite, inflammation or intestinal barrier function; (iii) clinical or experimental evidence supporting microbiota-targeted interventions, including probiotics, prebiotics or fecal microbiota transfer; and (iv) methodological or translational issues relevant to microbiota-informed nutrition.

Inclusion criteria were: published articles or academic reports directly relevant to gut microbiota and dietary/metabolic response; human observational, interventional, mechanistic animal, in vitro, review or perspective publications; and availability of sufficient bibliographic and content information for qualitative synthesis. Exclusion criteria were: duplicate citations; publications not focused on diet, microbiota or metabolic/endocrine outcomes; inaccessible full texts; and records with limited relevance after full-text assessment. Forty records were initially identified. After removal of duplicates, title/abstract screening and full-text eligibility assessment, 21 references were retained. The selection process is summarized in Figure 1.

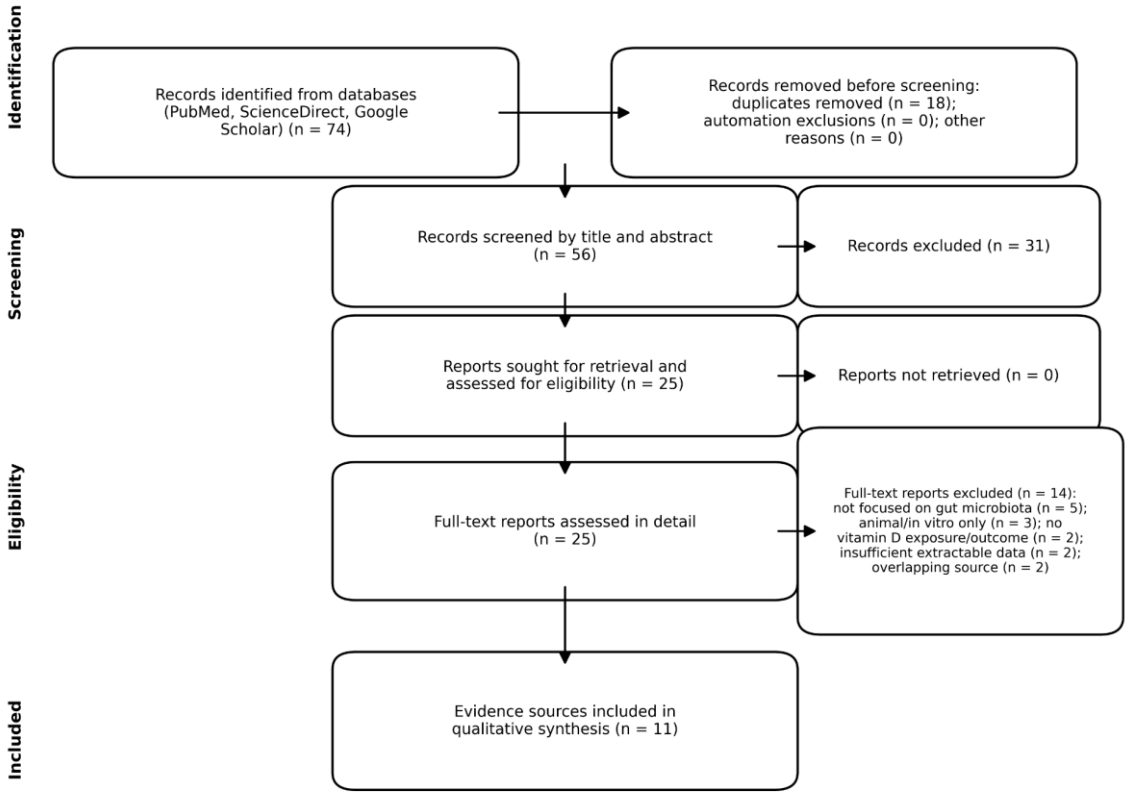

Figure 1. PRISMA-style flow diagram of the literature search and study selection process. The diagram shows records identified, duplicates removed, records screened, records excluded, full-text reports assessed, full-text reports excluded with principal reasons, and evidence sources included in the qualitative synthesis.

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Results:

The 21 retained publications comprised human cohort studies, short-term dietary intervention studies, microbiota-transfer evidence, mechanistic animal or cellular studies, metagenomic resource studies, narrative or critical reviews and one doctoral thesis focused on prebiotic-bacteria interactions. A structured synthesis of all retained articles is provided in Table 1. Together, the evidence supports a model in which baseline microbial ecology, microbial functional capacity and host context determine the metabolic benefit derived from dietary interventions.

Table 1. Summary of the 21 reviewed articles and their relevance to the review.

Article Title	Authors & Year	Study Design	Sample Size & Population	Key Findings	Relevance to Our Study
Are We Really Vastly Outnumbered? Revisiting the Ratio of Bacterial to Host Cells in Humans	Sender, Fuchs, Milo (2016)	Quantitative review/commentary	Not applicable; literature-based human host/microbial cell estimates	Revised the widely cited 10:1 microbial-to-human cell ratio toward an approximate 1:1 estimate.	Frames the microbiota as a large but biologically integrated metabolic interface rather than a numerically overwhelming entity.
Human Genetics Shape the Gut Microbiome	Goodrich et al. (2014)	Human twin cohort microbiome study	>1,000 fecal samples from the TwinsUK cohort, including 416 twin pairs	Host genetics contributed to microbial variation; heritable taxa, including Christensenellaceae, were associated with leanness-related traits.	Supports the concept that baseline microbiota is individualized before dietary intervention begins.
Diet Rapidly and Reproducibly Alters the Human Gut Microbiome	David et al. (2014)	Short-term controlled dietary intervention	10 healthy adult volunteers exposed to plant-based and animal-based dietary patterns	Diet altered microbial composition and activity within days, with effects on bile-tolerant taxa and microbial gene expression.	Shows that diet can rapidly remodel the microbiome, a core premise for microbiota-guided nutrition.

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Article Title	**Authors & Year**	**Study Design**	**Sample Size & Population**	**Key Findings**	**Relevance to Our Study**
Personalized Nutrition by Prediction of Glycemic Responses	Zeevi et al. (2015)	Prospective cohort, machine-learning model, validation and dietary intervention	800 participants; independent 100-person validation cohort; individualized dietary intervention subgroup	Identical meals produced divergent postprandial glycemic responses; microbiome features improved personalized response prediction.	Directly supports microbiota-informed precision nutrition for glycemic control.
Richness of Human Gut Microbiome Correlates with Metabolic Markers	Le Chatelier et al. (2013)	Metagenomic association study	292 Danish adults, including non-obese and obese participants	Low microbial gene richness was associated with adiposity, insulin resistance, dyslipidemia and low-grade inflammation.	Identifies microbial richness as a potential determinant of metabolic responsiveness.
The Firmicutes/Bacteroidetes Ratio: A Relevant Marker of Gut Dysbiosis in Obese Patients?	Magne et al. (2020)	Critical narrative review	Not applicable; review of obesity-associated microbiome studies	The Firmicutes/Bacteroidetes ratio is inconsistent and insufficient alone as a marker of obesity-related dysbiosis.	Adds caution against oversimplified taxonomic markers in clinical interpretation.

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Article Title	**Authors & Year**	**Study Design**	**Sample Size & Population**	**Key Findings**	**Relevance to Our Study**
Characterization of Gut Microbiomes in Nonalcoholic Steatohepatitis Patients	Zhu et al. (2013)	Pediatric observational 16S ribosomal RNA study	Children with nonalcoholic steatohepatitis, obese controls and healthy controls	Disease status clustered with distinct microbial profiles; alcohol-producing bacteria and elevated blood ethanol were linked to nonalcoholic steatohepatitis.	Connects dysbiosis, microbial metabolites and metabolic liver disease relevant to dietary response.
Transfer of Intestinal Microbiota from Lean Donors Increases Insulin Sensitivity in Individuals with Metabolic Syndrome	Vrieze et al. (2012)	Randomized clinical microbiota-transfer intervention	18 male participants with metabolic syndrome receiving allogenic or autologous microbiota infusion	Lean-donor microbiota transfer improved insulin sensitivity and altered intestinal microbial composition.	Provides causal human evidence that microbiota can modulate metabolic phenotype.
Cross-talk between Akkermansia muciniphila and Intestinal Epithelium Controls Diet-Induced Obesity	Everard et al. (2013)	Experimental animal/mechanistic study	Diet-induced obese and diabetic mouse models; complementary mechanistic experiments	Akkermansia muciniphila abundance was linked with improved barrier function, reduced endotoxemia and improved metabolic parameters.	Identifies a candidate microbiota target relevant to obesity and dietary modulation.

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Article Title	**Authors & Year**	**Study Design**	**Sample Size & Population**	**Key Findings**	**Relevance to Our Study**
A Catalog of the Mouse Gut Metagenome	Xiao et al. (2015)	Metagenomic resource study	184 mice from diverse genetic, housing and dietary backgrounds	Established a mouse gut metagenomic gene catalog useful for functional analysis of diet-microbiota interactions.	Supports mechanistic translation from mouse models to diet-responsive microbial functions.
Regional Variation Limits Applications of Healthy Gut Microbiome Reference Ranges and Disease Models	He et al. (2018)	Population microbiome modeling study	7,009 individuals from 14 districts within one Chinese province	Geographic variation strongly shaped microbiota and reduced generalizability of disease models across locations.	Highlights why microbiota-based nutrition models require local validation.
From Dietary Fiber to Host Physiology: Short-Chain Fatty Acids as Key Bacterial Metabolites	Koh et al. (2016)	Mechanistic review	Not applicable; synthesis of experimental and clinical evidence	Short-chain fatty acids generated from fiber fermentation influence epithelial integrity, immunity, appetite and glucose-lipid metabolism.	Provides the principal mechanistic bridge between fiber intake, microbiota function and host metabolism.
Commensal Microbe-Derived Butyrate Induces the Differentiation of Colonic Regulatory T Cells	Furusawa et al. (2013)	Experimental mechanistic study	Mouse models and cellular/metabolomic experiments	Butyrate promoted colonic regulatory T-cell differentiation and contributed to anti-inflammatory immune regulation.	Links microbial fermentation products to immune pathways that may influence metabolic inflammation.

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Article Title	**Authors & Year**	**Study Design**	**Sample Size & Population**	**Key Findings**	**Relevance to Our Study**
Probiotics in Prevention and Treatment of Obesity: A Critical View	Kobyliak et al. (2016)	Critical review	Not applicable; synthesis of animal and human probiotic evidence	Probiotics may influence appetite, adiposity, barrier function and inflammatory tone, but effects are strain- and context-dependent.	Supports cautious interpretation of probiotic adjuncts in obesity care.
Dietary Fiber and Body Weight	Slavin (2013)	Narrative review	Not applicable; review of human studies on dietary fiber and body weight	Dietary fiber may support weight management through satiety, lower energy density and metabolic effects.	Connects fiber intake to clinically relevant outcomes that may be mediated partly by microbiota.
Probiotics and Prebiotics in Intestinal Health and Disease: From Biology to the Clinic	Sanders et al. (2019)	Clinical and mechanistic review	Not applicable; synthesis of probiotic and prebiotic evidence	Clinical effects depend on strain, prebiotic substrate, host context and target outcome.	Reinforces the need for precise intervention definitions in microbiota-targeted nutrition.
Probiotics and Personalized Nutrition	Suez et al. (2019)	Perspective/commentary	Not applicable	Empiric probiotic effects vary among individuals and require host- and microbiome-aware personalization.	Supports individualized rather than universal probiotic recommendations.

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Article Title	**Authors & Year**	**Study Design**	**Sample Size & Population**	**Key Findings**	**Relevance to Our Study**
Human Gut Microbes Impact Host Serum Metabolome and Insulin Sensitivity	Pedersen et al. (2016)	Human metagenomic and metabolomic association study	277 non-diabetic Danish individuals	Microbial functional potential was associated with serum branched-chain amino acid patterns and insulin resistance.	Links microbial function to systemic metabolic markers relevant to dietary outcomes.
Le rôle de l'intestin et du microbiote dans les fonctions métaboliques	Vily-Petit, Mithieux (2024)	Mechanistic review	Not applicable	Describes intestinal energy harvesting, bacterial metabolite handling and intestinal gluconeogenesis as metabolic regulatory mechanisms.	Updates the mechanistic interpretation of gut-derived signals in metabolic disease.
Histoire du microbiote, déterminant des maladies métaboliques	Schlienger (2024)	Narrative review	Not applicable	Summarizes the historical evolution of microbiota research and its links with obesity, diabetes and metabolic diseases.	Provides contemporary clinical context for the microbiota-metabolic disease axis.
Exploration approfondie des interactions entre des prébiotiques et une sélection de bactéries intestinales pertinentes pour la santé	Biscarrat (2023)	Doctoral thesis; experimental/ in vitro microbiology	Selected health-relevant intestinal bacteria and prebiotic substrates; no human population	Explores interactions between prebiotic substrates and selected gut bacteria, including cooperative microbial dynamics.	Supports the biological plausibility of targeted prebiotic strategies.

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Baseline microbial diversity and gene richness appear to be important markers of metabolic resilience. Low microbial gene richness has been associated with adiposity, insulin resistance, dyslipidemia and inflammatory activation, whereas richer microbial ecosystems tend to coexist with more favorable metabolic profiles (5). However, single taxonomic indicators should be interpreted cautiously. For example, the Firmicutes/Bacteroidetes ratio is frequently mentioned in obesity research, but critical reviews emphasize that it is inconsistent and insufficient as a stand-alone marker of dysbiosis or treatment response (6).

Diet itself can rapidly reshape the gut microbiome. Controlled feeding data show that plant-based and animal-based dietary patterns can induce reproducible microbial shifts within a short time frame, supporting the concept that nutritional exposure acts as a strong ecological pressure on the intestinal ecosystem (3). At the same time, host genetics and geography constrain microbial variability and may limit the generalizability of microbiome-based prediction models across populations (2,11). These findings are clinically important because they suggest that microbiota-informed recommendations may require local validation and patient-specific interpretation.

Evidence linking microbiota to glycemic response is particularly relevant for diabetes and endocrinology. In personalized nutrition research, identical meals produced highly variable postprandial glycemic responses, and microbiome features improved prediction of individual glycemic trajectories when combined with clinical and dietary variables (4). In another human metabolic study, gut microbial functional potential was associated with serum metabolomic patterns and insulin resistance in non-diabetic individuals (18). Clinical causality is suggested by fecal microbiota transfer data, where microbiota from lean donors increased insulin sensitivity in individuals with metabolic syndrome (8).

Several microbial taxa and functions recur across the literature. Akkermansia muciniphila has been linked in experimental models to improved epithelial barrier function, reduced metabolic endotoxemia and improved metabolic phenotype during diet-induced obesity (9). Bifidobacterium and Lactobacillus are commonly discussed in probiotic research, although strain specificity, host context and background diet strongly influence clinical effects (14,16,17). Studies of nonalcoholic steatohepatitis also show that microbial profiles and metabolite production may differ according to metabolic disease state, further supporting a role for dysbiosis in cardiometabolic complications (7,20).

Microbial metabolites provide a mechanistic bridge between dietary substrates and host metabolic outcomes. Fermentation of dietary fiber generates short-chain fatty acids that influence epithelial integrity, immune regulation, appetite-related signaling and glucose-lipid metabolism (12,13,15,19). Experimental and translational evidence also supports roles for bile acid transformation, tryptophan-derived metabolites, inflammatory signaling and intestinal permeability. These pathways are summarized conceptually in Figure 2. Prebiotic studies and experimental work on bacterial interactions suggest that targeted substrates may selectively promote beneficial microbial functions, although clinical translation remains incomplete (16,21).

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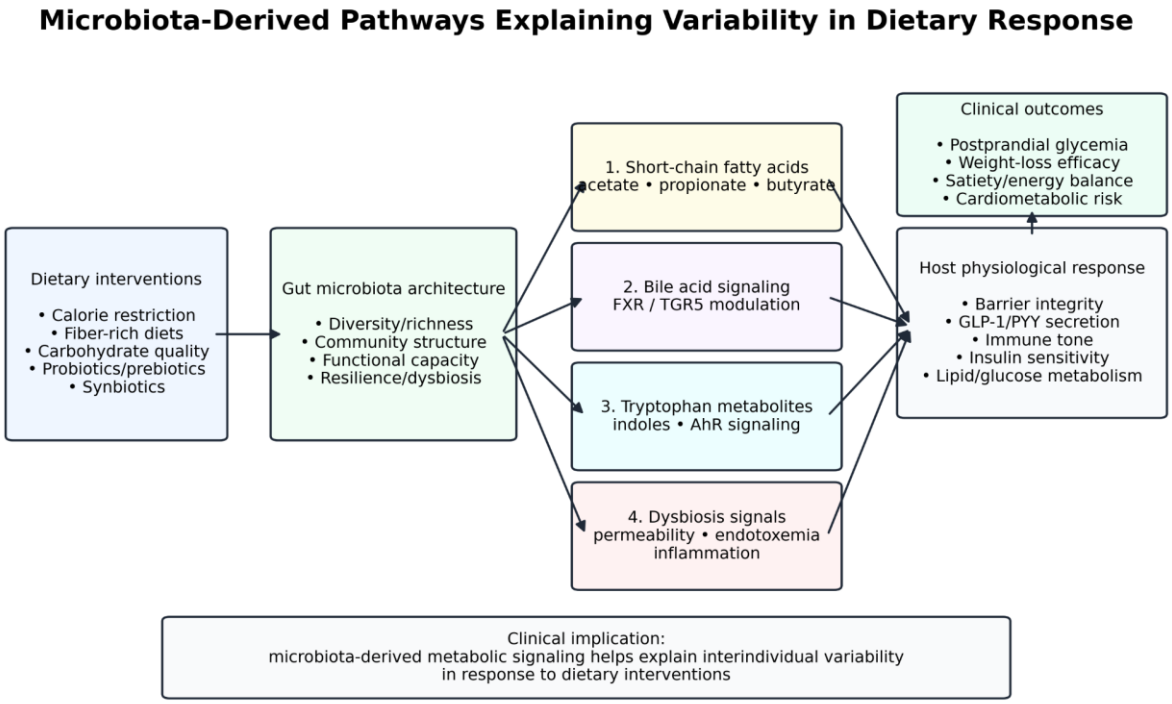

Figure 2. Conceptual framework of microbiota-derived metabolic pathways relevant to variability in dietary response. Dietary exposures reshape microbial diversity, community structure and functional capacity. Microbiota-derived pathways, including short-chain fatty acid production, bile acid signaling, tryptophan-derived metabolites and dysbiosis-associated inflammatory signaling, influence epithelial barrier integrity, incretin-related pathways, immune tone, insulin sensitivity and glucose-lipid metabolism.

Discussion:

This review supports the view that the gut microbiota is not merely a passive marker of diet quality but an active determinant of dietary response. The same nutritional intervention may yield divergent outcomes because individuals differ in microbial richness, dominant community structure, fermentative capacity, metabolite production, epithelial barrier status and inflammatory tone. This helps explain why conventional one-size-fits-all dietary recommendations often produce heterogeneous results in obesity and dysmetabolic disease.

The strongest clinical implications concern precision nutrition. Baseline microbiome profiling could, in principle, help identify patients more likely to respond to fiber enrichment, carbohydrate-quality modification, probiotic or prebiotic adjuncts, or personalized glycemic-response algorithms. Such strategies are especially attractive in prediabetes, type 2 diabetes and metabolic syndrome, where early improvement in glycemic variability, insulin sensitivity and adherence may influence long-term outcome. Nevertheless, current evidence is not sufficient to justify routine microbiome-guided prescribing for all patients.

Several limitations should be emphasized. Studies differ in population characteristics, dietary definitions, sample collection, sequencing methodology, bioinformatic pipelines and metabolic endpoints. Taxonomic

signatures are context dependent, and findings from one region or cohort may not transfer directly to another. Many probiotic and prebiotic studies are limited by strain heterogeneity, small sample size, short duration or inconsistent clinical endpoints. In addition, fecal microbiota does not fully represent mucosal microbiota, and taxonomic profiling may miss functional differences that are clinically important.

Future research should prioritize prospective interventional trials that combine standardized microbiome sampling with detailed dietary assessment, validated metabolic outcomes and multi-omics approaches. Function-based biomarkers, including microbial gene content, metabolomics and bile acid or short-chain fatty acid profiles, may be more informative than broad taxonomic ratios alone. Integrating microbiome data with clinical variables, medication exposure, lifestyle factors and geography will be essential for developing prediction models that are clinically useful and reproducible.

Conclusion:

The gut microbiota is a major biological determinant of interindividual variability in response to dietary interventions. By regulating microbial metabolite production, intestinal barrier integrity, immune signaling, incretin-related pathways and systemic metabolic tone, it helps explain why similar diets may produce divergent clinical outcomes. Microbiota-informed nutrition has strong potential for future precision care in diabetes and endocrinology, but standardized prospective studies and validated clinical algorithms are required before routine implementation.

Statements

Ethical Statement: Hence, the research is exclusively based on published literature, Ethical Approval is not required.

Artificial Intelligence (AI) Disclosure Statement: During the preparation of this study the author(s) used OpenAI ChatGPT in writing assistance, manuscript organization, table preparation and figure generation. After using this AI tool/service, the author(s) revised and edited the content and take(s) full responsibility for the publication.

Data Sharing Statement: All Data sets are included in the published article and the accompanying table file.

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