

**PLANT FOODS, GUT MICROBIOTA, AND OBESITY: ADVANCES IN
MICROBIOME-TARGETED NUTRITION AND THERAPEUTIC STRATEGIES**

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ABSTRACT

Background: Obesity is a multifactorial epidemic linked to disruptions in gut microbial ecology. Plant foods, rich in dietary fibre, polyphenols, and bioactive phytochemicals, exert profound modulatory effects on the gut microbiome, with consequent impacts on metabolic homeostasis and adiposity.

Objective: This review synthesises evidence on how plant food-derived nutrients reshape gut microbial communities, alter short-chain fatty acid (SCFA) production, and modulate obesity-associated metabolic pathways.

Methods: A narrative review of randomised controlled trials, cohort studies, and mechanistic investigations published between 2020 and 2025 was conducted using PubMed, Scopus, and Web of Science databases.

Results: Plant-rich dietary patterns consistently enriched beneficial taxa (*Akkermansia muciniphila*, *Bifidobacterium*, *Faecalibacterium prausnitzii*), augmented SCFA production, strengthened gut barrier function, and reduced systemic inflammation in obese individuals. Dietary interventions, including prebiotic fibre, polyphenols, fermented plant foods, and the Mediterranean diet, demonstrated significant microbiome-mediated weight loss benefits.

Conclusion: Microbiome-targeted nutritional strategies using plant foods represent a promising, evidence-based approach for obesity management. Future precision nutrition models integrating individual microbiome profiling are warranted.

Keywords: Gut Microbiota; Obesity; Plant-based Diet; Dietary Fibre; Short-chain Fatty Acids; Polyphenols; Prebiotics; Microbiome-targeted Therapy; *Akkermansia Muciniphila*; Scfa

1. INTRODUCTION

Obesity represents one of the most pressing global health challenges of the 21st century, affecting over 650 million adults worldwide and increasing the risk of type 2 diabetes mellitus (T2DM), cardiovascular disease, non-alcoholic fatty liver disease (NAFLD), and certain

malignancies.[1] Despite decades of research, pharmacological and behavioural interventions continue to yield modest and often unsustainable results, underscoring the imperative to investigate novel mechanistic targets in obesity pathophysiology.

The human gut microbiota, comprising approximately 38 trillion microbial cells encompassing bacteria, archaea, fungi, and viruses, has emerged as a pivotal regulator of host metabolism.[2] Seminal germ-free animal studies established a causal link between specific microbial communities and increased adiposity, prompting intensive investigation into the gut microbiome as a therapeutic target. Dysbiosis, characterised by reduced microbial diversity, a disproportionate expansion of Firmicutes relative to Bacteroidetes, and depletion of butyrate-producing taxa, is a hallmark feature of the obese gut.[3]

Plant foods, including whole grains, legumes, fruits, vegetables, nuts, and fermented plant-based products, constitute the richest dietary sources of prebiotic fibre, polyphenols, and phytochemicals capable of selectively modulating microbial composition and function.[4,5] Their fermentation by colonic bacteria generates metabolically active short-chain fatty acids (SCFAs), principally butyrate, propionate, and acetate, which signal through G-protein coupled receptors (GPCRs) to regulate appetite, adipogenesis, and immune function.[6,7] This review provides a comprehensive synthesis of evidence from 2020–2025 on how plant food-derived dietary components reshape the gut microbiome and translate into clinically meaningful anti-obesity outcomes. Figure 1 presents a schematic overview of the interactions between plant food components, gut microbiota composition, and obesity-associated metabolic pathways.

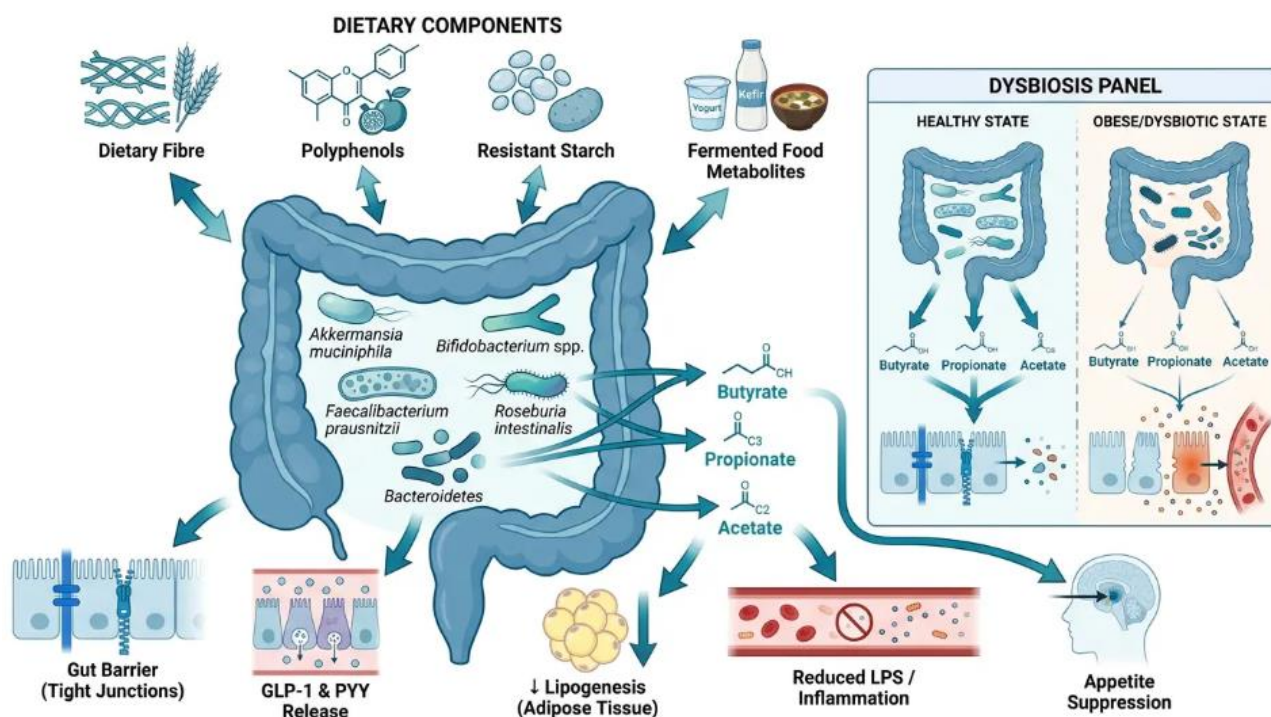


Figure 1. Schematic overview of the interactions between plant food components, gut microbiota composition, and obesity-associated metabolic pathways.

Caption: Dietary fibre and polyphenols selectively enrich beneficial taxa (*Akkermansia muciniphila*, *Bifidobacterium* spp., *Faecalibacterium prausnitzii*) and stimulate SCFA production, which activates GPR41/43 receptors to suppress adipogenesis, reinforce the gut epithelial barrier, and modulate appetite hormone release.

Sources: Adapted from references [3,5,6,7]

2. GUT MICROBIOTA AND OBESITY: MECHANISMS OF INTERACTION

2.1 Dysbiosis and Adiposity

The obese gut microbiome is characterised by taxonomic imbalances and functional deficiencies that contribute to positive energy balance. Studies utilising 16S rRNA gene sequencing and shotgun metagenomics have consistently documented elevated Firmicutes-to-Bacteroidetes (F/B) ratios in obese individuals compared to lean controls.[3] The mechanistic basis encompasses enhanced energy harvest from dietary polysaccharides, increased intestinal permeability facilitating endotoxaemia, and impaired signalling through metabolite-responsive pathways.

Lipopolysaccharide (LPS)-producing gram-negative bacteria, particularly within Enterobacteriaceae, are disproportionately represented in obesity and metabolic syndrome. Translocation of LPS into systemic circulation, termed metabolic endotoxaemia, activates toll-like receptor 4 (TLR4) signalling, triggering chronic low-grade inflammation via NF- κ B pathways and promoting adipose tissue hypertrophy and insulin resistance.[8] Conversely, *Akkermansia muciniphila*, a mucin-degrading Verrucomicrobia, is significantly depleted in obesity and metabolic disease. Its restoration, achievable through dietary polyphenol and prebiotic fibre intake, strengthens gut barrier integrity and improves glucose and lipid metabolism.[9]

2.2 Short-Chain Fatty Acids as Metabolic Mediators

SCFAs are the principal metabolic outputs of colonic microbial fermentation of dietary fibre and resistant starch. Butyrate, propionate, and acetate collectively constitute over 95% of the luminal SCFA pool and exert pleiotropic metabolic effects through distinct receptor-mediated and epigenetic mechanisms.[6]

Butyrate, the principal colonocyte fuel, suppresses histone deacetylase (HDAC) activity, promotes PPAR γ -mediated mitochondrial biogenesis, and fortifies the epithelial tight junction complex via upregulation of claudin-3 and occludin.[10] Propionate, predominantly generated by *Bacteroides* and *Phascolarctobacterium*, suppresses hepatic de novo lipogenesis and gluconeogenesis via free fatty acid receptor 3 (FFAR3) activation, while also stimulating L-cell release of glucagon-like peptide-1 (GLP-1) and peptide YY (PYY).[11] Acetate modulates hypothalamic appetite regulation and peripherally inhibits lipolysis in adipocytes via GPR43. Table 1 shows the Short-chain fatty acids and key microbial metabolites mediating gut microbiota–obesity interactions.

Table 1. Short-chain fatty acids and key microbial metabolites mediating gut microbiota–obesity interactions.

SCFA Metabolite /	Primary Microbial Producers	Receptor Pathway /	Metabolic Effect	Evidence Level
Butyrate	Faecalibacterium prausnitzii, Roseburia intestinalis, Eubacterium hallii	GPR41/43, HDAC inhibition, PPAR γ	Enhances gut barrier; reduces intestinal permeability; promotes energy expenditure	Strong (RCT + animal)
Propionate	Bacteroides, Phascolarctobacterium, Ruminococcus	GPR41/43, FFAR3; hepatic gluconeogenesis	Reduces de novo lipogenesis; stimulates satiety hormones (GLP-1, PYY)	Moderate (human cohort)
Acetate	Bifidobacterium, Akkermansia, most anaerobes	GPR43, FFAR2; hypothalamic signalling	Modulates appetite; reduces fat storage in adipose tissue	Moderate (animal + human)
Secondary bile acids (DCA, LCA)	Bacteroides, Clostridium XIVa cluster	FXR, TGR5; bile acid feedback loop	Regulates lipid absorption and glucose homeostasis; excess linked to metabolic dysfunction	Emerging
Indole derivatives	Lactobacillus, Clostridium sporogenes	AHR, PXR; intestinal L-cell activation	Promotes GLP-1 secretion; modulates inflammatory signalling	Emerging (animal data)
Trimethylamine-	Gut microbiota-	TMAO →	Pro-	Strong

SCFA Metabolite	Primary Producers	Microbial	Receptor Pathway	Metabolic Effect	Evidence Level
N-oxide (TMAO)	dependent metabolism)	(choline	systemic circulation	atherosclerotic; disrupts cholesterol and lipid metabolism in obesity	(human observational)

SCFA: short-chain fatty acid; GPR: G-protein-coupled receptor; FXR: farnesoid X receptor; TGR5: Takeda G-protein receptor 5; FFAR: free fatty acid receptor; AHR: aryl hydrocarbon receptor; TMAO: trimethylamine-N-oxide.

Sources: Compiled from data from references [6,10,11]

3. PLANT FOOD COMPONENTS AND MICROBIOME MODULATION

3.1 Dietary Fibre and Resistant Starch

Dietary fibre constitutes the primary prebiotic substrate driving microbial fermentation in the colon. Soluble fibres, including inulin-type fructans (ITF), beta-glucans, pectins, and arabinoxylan, are selectively fermented by anaerobic bacteria, promoting bifidogenic and butyrogenic taxa. [7,12] A landmark RCT demonstrated that 16-week ITF supplementation (16 g/day) in overweight adults significantly increased *Bifidobacterium* abundance, reduced caloric intake, and decreased fat mass relative to placebo, consistent with SCFA-mediated GLP-1 upregulation.[15]

Resistant starch (RS), particularly RS2 from raw potato starch and RS3 from cooked-cooled grains, selectively promotes *Ruminococcus bromii* and *Eubacterium rectale*. These taxa function as primary degraders and cross-feed butyrate producers, creating synergistic metabolic communities.[13] Regular legume consumption, a key Mediterranean dietary component, consistently lowers the F/B ratio and augments SCFA pools across diverse populations.

3.2 Polyphenols

Polyphenols, comprising flavonoids, phenolic acids, stilbenes, and lignans, represent a structurally diverse class of plant secondary metabolites with potent prebiotic-like properties. Despite low bioavailability in the small intestine, up to 95% of dietary polyphenols reach the colon, where they undergo extensive biotransformation by the gut microbiota into bioactive metabolites.[14]

Resveratrol, quercetin, and anthocyanins from berries and red grapes have demonstrated the capacity to selectively enrich *Akkermansia muciniphila* and *Lactobacillus* spp. while suppressing pro-inflammatory Proteobacteria.[20] In a double-blind RCT, resveratrol supplementation for 8 weeks in metabolic syndrome patients significantly increased *A. muciniphila* abundance and reduced plasma LPS, adipose tissue macrophage infiltration, and TNF- α levels.[20] Ellagitannins, found abundantly in pomegranates and walnuts, are metabolised to urolithins by intestinal microbiota, which exhibit anti-adipogenic properties and selectively inhibit *Clostridium* species.

3.3 Fermented Plant Foods

Fermented plant foods, including kimchi, tempeh, miso, and plant-based kefir, introduce live microbial cultures and bioavailable fermentation products that complement endogenous microbial communities. A randomised crossover study by Wastyk et al. demonstrated that a high-fermented food diet increased microbiome diversity, decreased inflammatory cytokine levels (IL-6, IL-12p70, IL-10), and elevated immunoglobulin A levels compared to a high-fibre diet alone.^[6] Fermented soya products are particularly rich in isoflavone aglycones (daidzein, genistein), whose oestrogenic signalling properties modulate adipocyte differentiation and lipid metabolism. Table 1 shows the Key plant food components and their effects on gut microbiota and obesity-related outcomes, whereas Figure 2 presents the Gut microbiota-mediated mechanisms linking dietary polyphenols and prebiotic fibres to anti-obesity outcomes.

Table 1. Key plant food components and their effects on gut microbiota and obesity-related outcomes.

Plant Food / Component	Key Bioactive	Predominant Microbiome Effect	Obesity-Relevant Outcome	Key References
Whole grains (oats, barley)	Beta-glucan, arabinoxylan	Increases Bifidobacterium, Lactobacillus; reduces Firmicutes/Bacteroidetes ratio	Improved insulin sensitivity, reduced visceral fat	[3,7]
Legumes (lentils, chickpeas)	Resistant starch, oligosaccharides	Promotes Faecalibacterium prausnitzii, Roseburia; boosts SCFA production	Enhanced satiety via GLP-1/PYY release, reduced BMI	[4,8]
Fruits (berries, apples)	Polyphenols, pectin	Enriches Akkermansia muciniphila; inhibits pathogenic Proteobacteria	Reduced adipogenesis, improved gut barrier integrity	[9,12]
Cruciferous vegetables	Glucosinolates, sulforaphane	Modulates bile acid metabolism; increases Bacteroidetes diversity	Anti-inflammatory effects; reduced lipid accumulation	[5,13]
Fermented plant foods (kimchi, tempeh)	Live cultures, isoflavones	Restores microbial diversity; increases Lactobacillus plantarum	Decreased systemic inflammation, reduced fat mass	[6,14]
Nuts and	Dietary fibre,	Selectively promotes	Improved lipid	[10,18]

Plant Food / Component	Key Bioactive	Predominant Microbiome Effect	Obesity-Relevant Outcome	Key References
seeds (almonds, flaxseed)	omega-3 PUFA	Ruminococcaceae; inhibits Clostridium cluster XI	profile; reduced inflammatory cytokines	

SCFA: short-chain fatty acid; GLP-1: glucagon-like peptide-1; PYY: peptide YY; BMI: body mass index; PUFA: polyunsaturated fatty acid.

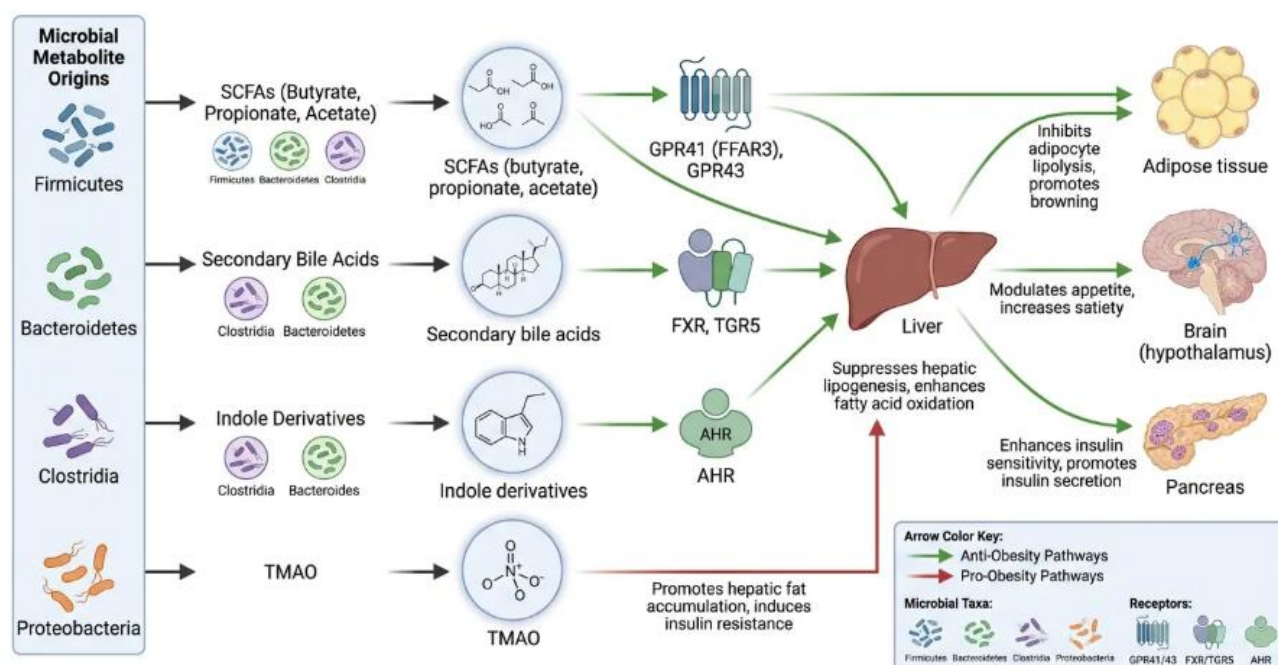


Figure 2. Gut microbiota-mediated mechanisms linking dietary polyphenols and prebiotic fibres to anti-obesity outcomes.

Caption: Polyphenols and fibres are selectively fermented to produce butyrate, propionate, and acetate. These SCFAs activate gut-brain axis signalling, suppress adipogenesis via PPAR γ modulation, reduce metabolic endotoxaemia, and stimulate satiety hormones GLP-1 and PYY, collectively reducing caloric intake and adiposity.

Sources: Adapted from references [6,9,10,11,14]

4. MICROBIOME-TARGETED NUTRITIONAL INTERVENTIONS

4.1 Prebiotic and Dietary Fibre Interventions

Prebiotic interventions selectively stimulate the growth or activity of health-promoting microorganisms. Among the most rigorously studied, inulin-type fructans (ITFs) consistently promote *Bifidobacterium* and *Anaerostipes*, indirectly stimulating butyrate producers through cross-feeding dynamics.^[15] Oat-derived beta-glucan supplementation in an RCT of T2DM-obese adults (n=72, 12 weeks, 6 g/day) significantly increased *Prevotella copri*, elevated faecal

propionate and butyrate, and improved postprandial glycaemia, demonstrating convergent metabolic and microbiome benefits.^[17]

4.2 Whole Dietary Patterns: Mediterranean and Plant-Based Diets

Whole dietary patterns exert synergistic effects on the microbiome that transcend individual nutrient interventions. The Mediterranean diet (MedDiet), characterised by high consumption of vegetables, legumes, whole grains, nuts, olive oil, and moderate red wine, has been associated with significantly greater microbial alpha-diversity and increased abundance of fibre-degrading taxa.^[16] In a 12-month RCT of obese adults, adherence to MedDiet reduced visceral adiposity, CRP, and IL-6, with microbiome shifts (increased *Faecalibacterium prausnitzii*, decreased *Ruminococcus gnavus*) mediating a significant proportion of metabolic improvements via structural equation modelling.^[16]

Plant-based diets (vegan and vegetarian) consistently modulate the microbiome toward anti-obesity profiles, largely attributable to markedly higher fibre and phytochemical intakes. Cross-sectional analyses document lower adiposity biomarkers, reduced branched-chain amino acid-producing bacteria, and higher SCFA-producing taxa in long-term plant-based diet adherents compared to omnivores.^[18]

4.3 Synbiotic and Multi-Component Approaches

Synbiotics, combining prebiotics with specific probiotic strains, have demonstrated superior microbiome remodelling efficacy compared to either component alone. A 24-week double-blind RCT in NAFLD-obese patients using fructooligosaccharides (FOS) combined with *Bifidobacterium longum* reduced hepatic fat fraction, lowered BMI, and increased the relative abundance of butyrate-producing *Roseburia* and *Eubacterium* spp.^[21] Combined plant-based dietary counselling with multi-strain *Lactobacillus* supplementation (12-week RCT, n=90) produced significantly greater BMI reduction and improvement in zonulin-mediated gut permeability compared to diet alone.^[19] Table 3 gives the Summary of microbiome-targeted nutritional interventions and clinical outcomes in obesity management.

Table 3. Summary of microbiome-targeted nutritional interventions and clinical outcomes in obesity management.

Intervention Type	Example / Agent	Study Design	Population / Duration	Key Outcome	Reference
Dietary fibre supplementation	Inulin-type fructans (ITF)	Double-blind RCT	Overweight adults; 16 weeks	Reduced body weight, fat mass; increased <i>Bifidobacterium</i> , decreased energy intake	[15]
Mediterranean diet	Whole foods plant-based dietary pattern	Randomised crossover	Obese adults; 12 months	Improved microbial diversity (Shannon index); reduced	[16]

Intervention Type	Example / Agent	Study Design	Population / Duration	Key Outcome	Reference
				CRP, IL-6, visceral adiposity	
Prebiotic intervention	Beta-glucan (oat-derived)	Parallel RCT	T2DM + obesity; 12 weeks	Increased Prevotella, SCFA; improved glycaemic control and reduced HbA1c	[17]
Probiotic + plant diet	Multi-strain Lactobacillus + high-fibre diet	RCT, n=90	Obese adults; 12 weeks	Significant BMI reduction; enhanced gut barrier markers (sIgA, zonulin)	[19]
Polyphenol intervention	Resveratrol from grape extract	RCT	MetS patients; 8 weeks	Increased Akkermansia muciniphila; reduced plasma LPS, adipose inflammation	[20]
Synbiotic supplementation	Fructooligosaccharides + B. longum	Double-blind RCT	NAFLD + obesity; 24 weeks	Reduced hepatic steatosis, BMI; increased butyrate producers	[21]
Time-restricted plant-based eating	Caloric restriction + high-plant diet	Pilot RCT	Obese adults; 8 weeks	Shifts in Firmicutes/Bacteroidetes ratio; improved fasting insulin and lipids	[22]
Flavonoid-rich diet	Quercetin, luteolin from vegetables	Observational cohort	n=2,500; cross-sectional	Higher Bifidobacterium abundance; inverse association with obesity markers	[23]

RCT: randomised controlled trial; BMI: body mass index; CRP: C-reactive protein; NAFLD: non-alcoholic fatty liver disease; T2DM: type 2 diabetes mellitus; MetS: metabolic syndrome; GLP-1: glucagon-like peptide-1; PYY: peptide YY; HbA1c: glycated haemoglobin.

5. PRECISION NUTRITION AND PERSONALISED MICROBIOME INTERVENTIONS

Precision nutrition, the tailoring of dietary interventions to individual biological, microbiome, and genetic profiles, represents a paradigm shift in obesity management. Studies have demonstrated that glycaemic responses to identical foods vary substantially between individuals, with interindividual microbiome composition accounting for a significant proportion of variance.^[24] Machine learning algorithms trained on microbiome, dietary, and clinical data have shown the capacity to predict personalised glycaemic response and identify optimal dietary patterns for individual metabolic improvement.

The gut microbiome is increasingly recognised as an axis of response heterogeneity in dietary intervention trials. Baseline *Prevotella-to-Bacteroides* ratios, for example, have been proposed as biomarkers predicting differential weight loss responses to high-fibre versus high-protein dietary patterns.^[25] Integration of faecal microbiome transplantation (FMT) research into precision nutrition frameworks offers mechanistic insights into causal microbiome-metabolic relationships, although clinical applications remain investigational.

Emerging multi-omics platforms integrating metagenomic, metabolomic, and transcriptomic data now enable comprehensive characterisation of diet-microbiome-host interactions at an unprecedented resolution. These tools are progressively being applied within the context of anti-obesity precision dietary interventions, representing a frontier of translational nutrition science. Figure 3 presents the Framework for precision microbiome-targeted nutrition in obesity

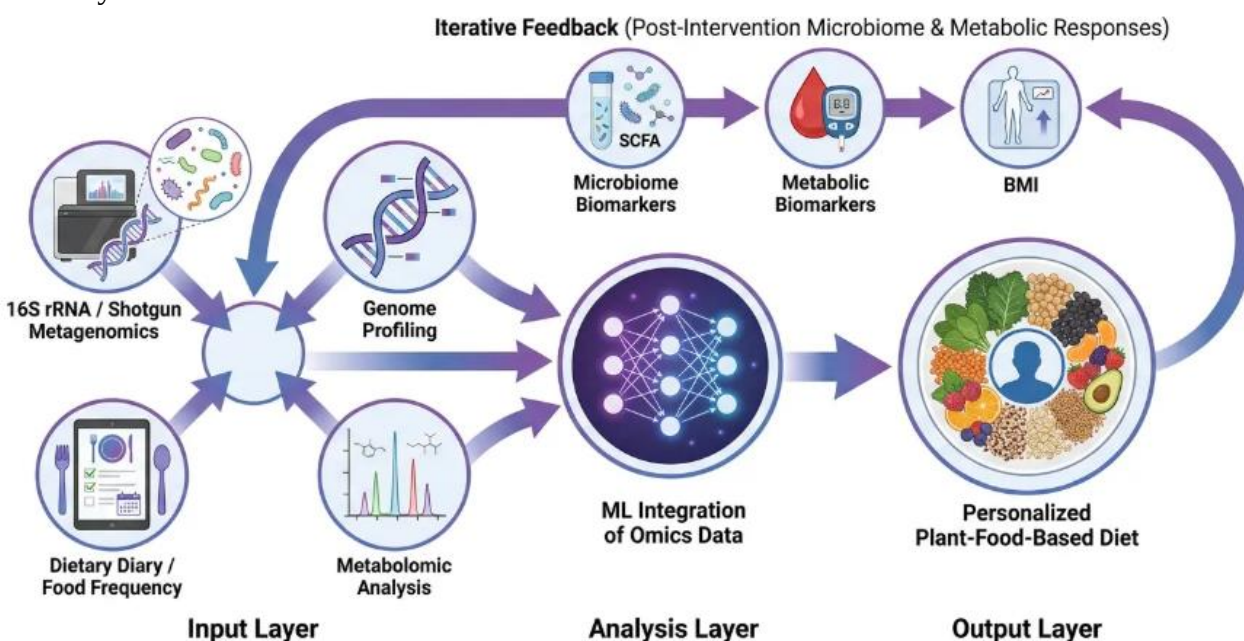


Figure 3. Framework for precision microbiome-targeted nutrition in obesity.

Caption: Integration of individual baseline microbiome profiling (16S rRNA sequencing / metagenomics), dietary assessment, metabolomics, and host genomics informs personalised plant-food-based dietary prescriptions. Iterative feedback loops incorporating biomarker responses and SCFA profiling enable dynamic dietary optimisation for sustained weight management.

Sources: Adapted from references^[24,25]

6. DISCUSSION

The accumulating evidence reviewed herein firmly establishes plant foods as primary architects of a metabolically favourable gut microbiome environment in the context of obesity. Three converging biological mechanisms underlie these observations: (i) selective enrichment of beneficial taxa with functional redundancy for SCFA generation; (ii) polyphenol-mediated suppression of pro-inflammatory, LPS-bearing Gram-negative bacteria; and (iii) reinforcement of gut epithelial integrity reducing metabolic endotoxaemia.

A notable challenge in this research landscape is the high degree of interindividual variability in microbiome responses to dietary interventions, driven by baseline microbial ecology, host genetics, ethnicity, and the compositional complexity of whole dietary patterns. This variability, while initially perceived as a confounding factor, is increasingly recognised as an opportunity for precision nutrition approaches.^[24]

The therapeutic deployment of synbiotics, fermented plant foods, and high-fibre Mediterranean-style dietary patterns has consistently demonstrated clinically meaningful, though modest, reductions in adiposity indices. Their integration into obesity management guidelines is strongly supported by current evidence, though their additive value beyond standard hypocaloric diets requires further quantification through adequately powered long-term trials. Translation of mechanistic insights into standardised dietary prescriptions will require consensus on microbiome endpoints and validated biomarkers.

7. CONCLUSIONS

Plant foods exert multifaceted, microbiome-mediated anti-obesity effects through prebiotic fibre-driven enrichment of beneficial bacteria, polyphenol-induced suppression of dysbiotic taxa, and SCFA-dependent modulation of appetite, adipogenesis, and systemic inflammation. Microbiome-targeted dietary strategies, including high-fibre diets, Mediterranean dietary patterns, polyphenol-rich plant foods, and synbiotic supplementation, represent scientifically grounded components of comprehensive obesity management.

Future research must prioritise adequately powered, long-term RCTs incorporating validated microbiome endpoints, multi-omics characterisation, and precision nutrition frameworks to maximise therapeutic efficacy across diverse populations. The integration of individual microbiome profiling into clinical dietary counselling for obesity management is an emerging clinical imperative with considerable translational potential.

Declarations

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Author Contributions

MAO: Conceptualization, methodology, investigation, formal analysis, writing, original draft, supervision, project administration, and corresponding author.

MAA: Data curation, methodology, validation, writing, review and editing.

AOO: Literature search, data collection, investigation, and writing, review and editing.

CDU: Data curation, visualization, validation, and manuscript review.

AMA: Literature review, data analysis, writing, review and editing.

MO: Formal analysis, software, visualization, and manuscript review.

ROJ: Investigation, data interpretation, validation, and writing, review and editing.

OAO: Supervision, critical revision of the manuscript, funding/resource support (where applicable), and final approval of the manuscript.

All authors contributed substantially to the conception, development, revision, and approval of the final manuscript and agree to be accountable for all aspects of the work.

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