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Dietary Modulation of Gut Microbiota in Obesity and Type 2 Diabetes: A Narrative Review

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ABSTRACT: Obesity and type 2 diabetes mellitus (T2DM) are major metabolic disorders shaped by interactions among diet, host physiology, and the gut microbiota. Growing human evidence indicates that dietary fiber, prebiotics, probiotics, synbiotics, polyphenols, fermented foods, and Mediterranean-style dietary patterns can alter microbial composition and function, with potential effects on short-chain fatty-acid production, intestinal barrier integrity, inflammatory signaling, and glucose homeostasis. This narrative review synthesizes human clinical and mechanistic evidence on dietary modulation of the gut microbiota in obesity and T2DM, with a structured literature update through September 2026. Recent systematic reviews and meta-analyses suggest that microbiota-targeted dietary interventions can produce modest improvements in glycemic and inflammatory outcomes, but effects on microbial diversity, body weight, and specific taxa remain heterogeneous. Inter-individual variation in baseline microbiota, intervention composition and dose, medication use, geographic dietary patterns, sequencing methods, and study duration limits direct comparison across trials. Emerging evidence supports a shift from universal microbiome-targeted recommendations toward food-based and precision-nutrition approaches that account for microbial and metabolic phenotype. Well-powered, longer-duration randomized trials using harmonized microbiome and metabolomic methods are needed before specific microbial signatures can be routinely used to guide clinical nutrition practice.

1. INTRODUCTION

Obesity and T2DM are complex metabolic disorders driven by interactions among dietary quality, physical activity, genetic susceptibility, medication exposure, and environmental factors. The intestinal microbiota has emerged as an additional metabolic interface because microbial communities contribute to nutrient transformation, immune regulation, mucosal barrier integrity, bile-acid metabolism, and production of metabolites that can influence systemic physiology [1,2].

Microbial alterations reported in obesity and T2DM include changes in diversity, functional capacity, and the relative abundance of taxa involved in short-chain fatty-acid (SCFA) production and barrier maintenance. However, a single reproducible 'dysbiotic signature' has not been established across populations. Differences in host phenotype, geography, diet, medication use, sequencing platforms, and bioinformatic pipelines contribute substantially to between-study heterogeneity [3,4,5].

Diet is among the most modifiable determinants of microbial ecology. Dietary fiber and prebiotics provide fermentable substrates; fermented foods and probiotics introduce or support potentially beneficial microorganisms; polyphenols undergo extensive microbial biotransformation; and dietary fat quality and overall dietary patterns influence microbial metabolites,

intestinal permeability, and inflammatory signaling. Recent evidence has increasingly moved beyond taxonomic descriptions toward functional outcomes, including SCFAs, bile acids, endotoxemia, glycemic control, and host inflammatory responses.

This narrative review therefore evaluates how major dietary components and dietary patterns modulate the gut microbiota in obesity and T2DM, integrates evidence from human interventions with mechanistic findings, critically examines inconsistent results and methodological limitations, and highlights implications for clinical nutrition and future precision-nutrition research.

2. MATERIALS AND METHODS

2.1 Review design and search scope

This narrative review integrates human clinical findings with mechanistic literature on dietary modulation of the gut microbiota in obesity and type 2 diabetes mellitus (T2DM). The literature search used PubMed/MEDLINE, Scopus, and Web of Science and covered January 2010 through September 2026. The aim was a focused, explanatory synthesis rather than an exhaustive systematic review or a new meta-analysis.

The search concepts combined gut microbiota or intestinal microbiome with obesity or T2DM and dietary exposures, including dietary fiber, prebiotics, probiotics, synbiotics, fermented foods, polyphenols, dietary fat, and Mediterranean-style diets. Microbial composition and function, glucose metabolism, inflammation, and related metabolic outcomes provided the outcome context.

2.2 Literature selection principles

Priority was given to human randomized or controlled interventions, systematic reviews and meta-analyses, and human observational studies reporting relevant diet–microbiota–metabolism relationships. Clinical evidence in obesity, T2DM, or related dysglycemia was considered most directly applicable. Studies in healthy adults were retained when informative about dietary effects on microbial or inflammatory endpoints, with their population limitations identified. Mechanistic reviews and experimental evidence were used to explain biological pathways rather than to establish clinical efficacy.

Case reports, non-peer-reviewed material, reports without a relevant diet–microbiota–metabolism relationship, and studies with insufficient methodological detail were excluded. Selection emphasized relevance to the review question, study design, population, exposure, and reported outcomes. The selected literature is representative rather than exhaustive.

2.3 Evidence categories and synthesis

Evidence is organized into dietary fiber and prebiotics; probiotics, synbiotics, and fermented foods; polyphenols; and dietary fat quality and whole-diet patterns. Human intervention evidence informs the assessment of clinical and microbial outcomes. Observational associations provide context but do not establish causation. Mechanistic evidence provides biological plausibility and is discussed separately from demonstrated clinical benefit. Table 1 summarizes representative human studies and evidence syntheses; Figure 1 presents a conceptual pathway rather than a validated causal model.

2.4 Interpretation of systematic reviews and meta-analyses

Interpretation considers the population and comparator, intervention composition and dose, duration, outcome definitions, and the consistency and magnitude of reported effects. Where reported by the source reviews, confidence intervals, between-study heterogeneity, risk-of-bias assessments, sensitivity analyses, and evidence-certainty ratings inform the strength of interpretation. Statistical significance is distinguished from clinically meaningful benefit; changes in microbial diversity, individual taxa, or metabolites are not assumed to demonstrate improvement in body weight or glycemic control.

Greater interpretive weight is placed on directly relevant human controlled evidence than on observational associations or mechanistic plausibility. Potential overlap of primary trials among reviews limits treating concordant pooled results as independent replication. This narrative review uses the cited reviews' risk-of-bias and certainty assessments and does not undertake new formal grading or pooled analysis.

3. RESULTS AND DISCUSSION

3.1 Gut microbiota patterns in obesity and T2DM

Obesity and T2DM have frequently been associated with altered microbial community structure and reduced representation of organisms linked with butyrate production or mucosal health, including *Faecalibacterium prausnitzii*, selected *Bifidobacterium* species, and *Akkermansia muciniphila*. These observations are biologically plausible because microbial metabolites can affect epithelial integrity, immune tone, incretin signaling, and insulin sensitivity. Nevertheless, recent syntheses emphasize that taxonomic findings are not uniform and that functional readouts may be more informative than individual taxa. A 2026 systematic review of 87 randomized trials involving 6,086 adults with overweight or obesity found substantial methodological and outcome heterogeneity; most interventions did not consistently alter alpha- or beta-diversity, although prebiotic and probiotic interventions showed selected taxonomic and body-composition effects [5]. This cautions against treating microbial diversity alone as a universal marker of metabolic improvement.

3.2 Dietary fiber and prebiotics

Dietary fiber is a major substrate for microbial fermentation. Fermentable fibers can increase production of acetate, propionate, and butyrate, which participate in epithelial energy metabolism, gut-barrier maintenance, immune regulation, appetite signaling, and glucose homeostasis [6,7]. Prebiotics such as inulin-type fructans can enrich *Bifidobacterium* and other saccharolytic organisms, but clinical responses vary according to fiber structure, dose, baseline diet, microbial functional capacity, and intervention duration. The 2026 obesity systematic review reported a positive association between prebiotic consumption and *Actinobacteria*/*Bifidobacterium* abundance, while also concluding that evidence linking microbiome changes to durable weight or metabolic improvement remains limited [5]. These findings support fiber-rich foods as a broadly appropriate dietary strategy while arguing against assuming that a specific microbial response will occur in every individual.

3.3 Probiotics, synbiotics, and fermented foods

Probiotic and synbiotic interventions have been studied extensively in dysglycemia. A 2026 GRADE-assessed systematic review and dose-response meta-analysis of 48 randomized trials reported reductions in fasting glucose, fasting insulin, HbA1c, and HOMA-IR with probiotic or synbiotic supplementation, although substantial heterogeneity was present across outcomes [8]. A separate 2025 systematic review of microbiome-targeted interventions in T2DM and related dysglycemia found generally modest glycemic improvements, particularly in interventions lasting at least 12 weeks, but highlighted inconsistency in mechanistic readouts and study designs [9]. Conversely, network meta-analytic evidence has judged the certainty or clinical importance of several effects to be limited, demonstrating that statistically significant changes should not automatically be interpreted as strong evidence for routine supplementation [10]. Fermented-food interventions may also increase microbial diversity and reduce inflammatory signaling in some populations [11], but food matrix, baseline diet, and product-specific microbial composition remain important sources of variability. The fermented-food findings cited here arose in healthy adults [11]; they should not be interpreted as direct evidence of treatment efficacy in obesity or T2DM. Likewise, clinical changes after probiotic or synbiotic supplementation do not by themselves establish a microbiota-mediated mechanism.

3.4 Polyphenols and microbiota-derived metabolites

Polyphenols from fruits, vegetables, tea, cocoa, berries, and other plant foods interact bidirectionally with the gut microbiota. Microorganisms transform poorly absorbed polyphenols into smaller bioactive metabolites, while polyphenols and their metabolites can alter microbial ecology and barrier-related pathways. A 2026 review emphasized microbiota-derived metabolites as key mediators linking polyphenols with obesity, T2DM, and other metabolic diseases [12]. A 2026 systematic review and meta-analysis of 32 randomized trials in adults with overweight or obesity found that intervention dose, duration, and food matrix influenced microbiota and inflammatory outcomes; whole polyphenol-rich foods appeared particularly relevant for selected inflammatory markers and beneficial taxa [13]. A 2025 meta-analysis of 13 randomized trials involving 670 adults with overweight or obesity reported lower circulating lipopolysaccharide and higher butyrate and acetate, without significant changes in body weight or BMI [14]. These findings concern intermediate biomarkers and do not establish durable weight loss or prove that clinical effects are mediated by the microbiota.

3.5 Dietary fat quality and whole-diet patterns

High-saturated-fat Western dietary patterns have been associated with adverse microbial and inflammatory profiles, whereas diets rich in plant foods, unsaturated fats, fiber, and polyphenols tend to support microbial functions associated with SCFA production and metabolic health. Mediterranean-style diets are especially relevant because they integrate multiple microbiota-active components rather than relying on a single supplement. A systematic review and meta-analysis published in 2026 evaluated Mediterranean-diet interventions reporting both glucose-metabolism and gut-microbiota outcomes in adults at increased cardiometabolic risk, reinforcing the potential for whole-diet patterns to influence metabolic and microbial endpoints while also demonstrating heterogeneity across intervention studies [15]. Earlier human work similarly linked Mediterranean-diet adherence with SCFA-producing organisms and metabolically favorable microbial functions [16].

3.6 Mechanistic integration: from food substrates to metabolic outcomes

The most plausible model is functional rather than taxon-specific. Fermentable carbohydrates can increase SCFA production; microbial transformation of polyphenols generates bioactive metabolites; dietary patterns can modify bile-acid pools and intestinal permeability; and changes in barrier integrity can influence exposure to microbial products such as lipopolysaccharide. These pathways may converge on inflammatory signaling, incretin biology, hepatic glucose production, peripheral insulin sensitivity, and appetite regulation. The same food or supplement may produce different metabolite profiles in individuals with different baseline microbial communities. This provides a biological basis for variable clinical response and for emerging precision-nutrition approaches. This pathway integrates biological explanations from mechanistic literature [2,6,7,12] with observations from human studies. It does not establish that every step has been demonstrated within the same clinical trial. Human observational associations, including diet-associated microbial signatures [17], can be influenced by confounding and reverse causation. Stronger causal inference requires temporally aligned dietary, microbial, metabolite, and clinical measurements in controlled interventions, with appropriate assessment of mediation. Experimental models can help identify pathways, but their findings require confirmation in relevant human populations.

3.7 Clinical interpretation and precision nutrition

The accumulated evidence supports diet as a practical means of influencing the gut microbial ecosystem, but current data do not justify prescribing a universal probiotic strain, prebiotic dose, or microbial target for all patients with obesity or T2DM. Food-first dietary patterns rich in diverse plant foods, fermentable fiber, polyphenols, and unsaturated fats have the advantage of simultaneously affecting multiple microbial and host pathways. Supplements may be useful in selected contexts, but strain specificity, dose, duration, medication use, and baseline phenotype should be considered. Future clinical nutrition research should determine whether baseline microbial or metabolomic signatures can reproducibly identify responders and non-responders before microbiome-guided precision nutrition becomes routine practice.

3.8 Methodological limitations and research priorities

Interpretation is limited by major differences in sequencing technology, taxonomic resolution, bioinformatics pipelines, stool collection and storage, dietary assessment, intervention duration, medication control, and outcome reporting. Many studies are small and short, and microbiome outcomes are often secondary endpoints. Recent systematic reviews continue to report substantial heterogeneity despite growth in the number of trials [5,8]. Future trials should pre-specify microbiome outcomes, use harmonized collection and analytical protocols, report absolute as well as relative microbial measures where feasible, integrate metagenomics with metabolomics, control important medications such as metformin and antibiotics, and include longer follow-up. Clinical outcomes should be prioritized over isolated taxonomic shifts. Alpha-diversity, beta-diversity, relative taxon abundance, microbial gene pathways, and metabolite concentrations represent different endpoints and are not interchangeable measures of benefit. A higher relative abundance may reflect changes in other organisms rather than an absolute increase, while metabolite measurements depend on sampling compartment and host absorption. These differences complicate comparison across studies and weaken attempts to define a universal clinically meaningful microbial signature.

Long-term randomized evidence remains limited, leaving uncertainty about persistence of microbial changes, sustained glycemic or weight outcomes, safety, and adherence after intervention withdrawal. Cohort-derived signatures require external validation across geographic populations, medication exposures, and analytical platforms before they can guide treatment. Trials should harmonize sample collection, sequencing, bioinformatics, and metabolomic methods; specify clinically relevant outcomes and follow-up; and account for diet, metformin, antibiotics, and other important covariates.

This review also has limitations arising from its narrative design. Selection is not exhaustive, and emphasis on representative studies may introduce selection bias. Published reviews may contain overlapping trials, so agreement between them is not necessarily independent confirmation. No new quantitative pooling or formal evidence-certainty grading was undertaken. Mechanistic plausibility, short-term biomarker shifts, and statistically significant pooled findings therefore remain distinct from established, durable clinical benefit.

Table 1. Representative recent human evidence on diet–microbiota–metabolic outcomes

Study	Design / population	Intervention / exposure	Key microbiota or metabolic finding	Interpretation
Wastyk et al., 2021 [11]	Randomized dietary intervention; healthy adults	High-fiber vs fermented-food diets	Fermented-food arm increased microbiota diversity and reduced several inflammatory markers	Supports food-based microbiota modulation; population not limited to metabolic disease
Asnicar et al., 2021 [17]	Large human cohort	Habitual diet patterns	Diet-associated microbial signatures correlated with cardiometabolic markers	Demonstrates personalized diet–microbiome relationships
Lee et al., 2026 [5]	Systematic review; 87 RCTs, 6,086 adults with BMI ≥ 25 kg/m ²	Lifestyle and microbiota-targeted interventions	Most interventions did not consistently change alpha/beta diversity; selected prebiotic/probiotic effects observed	Strong evidence of heterogeneity; diversity alone is not a sufficient endpoint
Setayesh et al., 2026 [8]	Systematic review/meta-analysis; 48 RCTs in prediabetes/T2DM	Probiotics and synbiotics	Modest pooled improvements in FBG, insulin, HbA1c and HOMA-IR; substantial heterogeneity	Potential adjunctive benefit, but effect size and certainty vary
Shalbah et al., 2025 [9]	Systematic review; 30 studies	Probiotics, prebiotics, synbiotics, FMT	Generally modest glycemic effects; mechanistic readouts inconsistent	Supports cautious clinical interpretation
Lauria et al., 2026 [15]	Systematic review/meta-analysis of intervention trials	Mediterranean diet	Evaluated combined glucose-metabolism and gut-microbiota outcomes	Supports whole-diet approach; heterogeneity remains
Wang et al., 2026 [13]	Systematic review/meta-analysis; 32 RCTs in overweight/obesity	Polyphenol interventions	Dose, duration and food matrix influenced inflammatory and microbial outcomes; beneficial taxa increased in subgroups	Whole polyphenol-rich foods may provide synergistic effects

4. CONCLUSION

Dietary modulation of the gut microbiota is biologically plausible and increasingly supported by human evidence in obesity and T2DM. Fiber-rich foods, prebiotics, fermented foods, probiotics/synbiotics, polyphenol-rich foods, and Mediterranean-style dietary patterns can influence microbial composition or function and may improve selected metabolic and inflammatory outcomes. However, recent evidence through 2026 also shows substantial heterogeneity, inconsistent changes in microbial

diversity, and variable translation from microbial biomarkers to clinically meaningful outcomes. Current evidence therefore favors a diverse, plant-rich, food-based dietary pattern while microbiome-targeted supplements should be interpreted as potentially useful adjuncts rather than universal therapies. Standardized, adequately powered, longer-term trials integrating metagenomic, metabolomic, dietary, medication, and clinical data are required to establish reproducible microbiome-informed precision-nutrition strategies.

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Author Contributions

Bi Bi Mariam: Conceptualization, methodology, literature synthesis, writing – original draft, writing – review and editing, and final approval of the manuscript.

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Conflict of Interest

The author declares no conflict of interest.

Ethics Statement

Ethical approval was not required because this review did not involve new research with human participants or animals.

Data Availability Statement

Not applicable. This narrative review did not generate a new dataset.

Informed Consent

Not applicable.

Clinical Trial Registration

Not applicable.

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Figure legend: Dietary components can influence gut microbial ecology and microbial metabolic outputs. The direction and magnitude of response vary according to baseline microbiota, host phenotype, medications, food matrix, dose, duration, and analytical methods.

Figure 1. Conceptual pathway linking diet, gut microbiota, and metabolic health

