

# Dietary influence on gut microbial balance transitioning from dysbiosis to eubiosis



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## Abstract

Diet is one of the strongest and most modifiable determinants of gut microbial composition and function. Shifts from a diverse, resilient microbial ecosystem (eubiosis) toward a disturbed state dominated by pathobionts and reduced metabolic capacity (dysbiosis) are linked to inflammatory, metabolic, and even neuropsychiatric disorders. This review summarizes current evidence on how dietary patterns, macronutrient composition, specific food groups, and targeted interventions (prebiotics, probiotics, fermented foods, and synbiotics) influence the microbiome and the pathways bridging diet, microbes, and host health. We outline mechanisms — substrate availability, microbial metabolite production (notably short-chain fatty acids, bile acid modification), mucosal barrier integrity, and immune modulation — that explain how diet drives the dysbiosis eubiosis continuum. We close with clinical implications, limitations of current evidence, and practical dietary strategies to restore or maintain microbial eubiosis.

**Keywords:** Dysbiosis, Eubiosis, Synbiotics

## 1. Introduction

Gut Microbiota also termed as intestinal microbiota is a group of micro-organisms consisting of eukaryote, prokaryote and viruses that colonizes the gastrointestinal tract, and found in the higher portions of the human cells. Gut microbes are key to human health including immune, metabolic and neurobehavioral traits. A healthy gut microbiome is typically characterized by taxonomic diversity, functional redundancy, and stable production of beneficial metabolites such as short-chain fatty acids (SCFAs) (1,2). In contrast, dysbiosis broadly defined as reduced diversity, loss of beneficial taxa, and expansions of potentially harmful microbes is associated with diseases ranging from inflammatory bowel disease (IBD) and obesity to cardiovascular disease and mood disorders (3). Diet is one of the most powerful external modulators of the gut microbiome, even short-term dietary shifts can alter microbial composition and metabolite output, while habitual dietary patterns shape long-term community structure. Recent systematic reviews and mechanistic studies have emphasized dietary fibre, plant-based patterns for e.g., Mediterranean diet, ultra-processed foods, and high-fat Western diets as major drivers of microbial states.

“Eubiosis” is best considered a functional construct: a community that supports host homeostasis through nutrient metabolism, colonization resistance, and immune regulation. “Dysbiosis” is not a single taxonomic signature but a shift in structure and function that

undermines those services — reduced microbial diversity, depletion of keystone SCFA producers e.g., Faecal bacterium, Roseburia, enrichment of pathobionts e.g., some Proteobacteria, and altered metabolite profiles. Contemporary reviews emphasize that resilience (ability to return to baseline after perturbation) and functional outputs are as important as taxonomic lists when assessing health.

## **2. Substrate availability and niche selection**

Dietary fibres and complex polysaccharides reach the colon and serve as primary substrates for saccharolytic bacteria. High-fibre diets enrich SCFA producers and increase butyrate production, which nourishes colonocytes and supports barrier function. Conversely, low-fibre, high-simple-sugar diets favor fast-growing, mucin-degrading species that can erode the mucus layer.

## **3. Microbial metabolite signalling**

SCFAs (acetate, propionate, butyrate) influence immune responses, gut motility, and metabolic signalling (e.g., GLP-1 secretion). Microbial bile acid transformations produce secondary bile acids that regulate host lipid metabolism and the intestinal immune environment; dietary fat quantity and composition strongly influence bile acid pools and thus microbiome structure.

## **4. Mucosal barrier and inflammatory cascades**

Diets promoting harmful taxa or depleting SCFA production can compromise tight junctions and mucus integrity, permitting microbial-associated molecular patterns (MAMPs) such as LPS to access immune receptors and trigger low-grade systemic inflammation. This inflammation further perturbs the niche, creating a feed-forward loop towards dysbiosis.

## **5. Antimicrobial exposures and processing**

Ultra-processed foods, food additives (emulsifiers, some artificial sweeteners), and frequent antibiotic use directly or indirectly perturb gut communities and reduce resilience. Processed diets are often low in fiber and high in additives that may alter mucous properties or microbial growth directly (4).

## **6. Dietary Patterns and Microbial Outcomes**

### **6.1 Mediterranean and Plant-Rich Diets**

The Mediterranean diet (MD) — high in whole grains, legumes, fruits, vegetables, nuts, olive oil, and moderate fish — consistently correlates with increased microbial diversity and enrichment of SCFA-producing taxa in both observational and interventional studies. Increased intake of dietary polyphenols and fibres in MD also supports beneficial metabolites and reduces systemic inflammation. Several 2024–2025 reviews confirm MD’s favorable modulation of microbiota and downstream metabolic markers.

### **6.2 High-Fat, Western-Style Diet**

A Western dietary pattern (high in saturated fat, refined carbohydrates, and ultra-processed foods) is repeatedly associated with lower diversity, loss of butyrate producers, increased mucin-degraders, and higher endotoxin translocation — mechanisms implicated in metabolic endotoxemia and insulin resistance. Animal models and human cohorts link this pattern to chronic low-grade inflammation and metabolic disease risk.

### **6.3 Fiber, Resistant Starch, and Polysaccharides**

Soluble and insoluble fibers, resistant starches, and other fermentable carbohydrates selectively increase saccharolytic bacteria and SCFA output. Interventions boosting fiber intake show

rapid increases in SCFA levels and beneficial taxa, though baseline microbiome composition may influence individual responsiveness.

#### **6.4 Protein and Fat Quality**

High animal protein diets may shift microbiota toward proteolytic fermentation products (e.g., branched-chain fatty acids, ammonia) linked to colonic mucosal stress, especially when combined with low fiber. The type of fat matters: saturated fats more strongly promote bile acid changes and dysbiosis than unsaturated fats.

#### **6.5 Fermented Foods**

Fermented foods (yogurt, kefir, kimchi, sauerkraut) introduce live microbes and fermentation-derived metabolites that can transiently alter gut communities and enhance certain beneficial functions. Recent trials suggest fermented foods can increase microbial diversity and reduce inflammatory markers even without large shifts in core taxa, although effects may be strain- and product-specific (5).

### **7. Targeted Interventions: Prebiotics, Probiotics, Synbiotics, and Beyond**

#### **7.1 Prebiotics**

Prebiotics are select fermentable substrates like inulin, FOS, GOS selectively stimulate beneficial microbes e.g., Bifidobacterium and increase SCFA production. Clinical trials show improvements in bowel function and metabolic markers in certain populations, though responses vary by baseline microbiome.

#### **7.2 Probiotics and Synbiotics**

Probiotic interventions can be useful for specific indications for e.g., antibiotic-associated diarrhea, certain IBS subsets. However, meta-analyses reveal heterogeneous effects: strain choice, dose, and host baseline microbiota strongly determine outcomes. Synbiotics are the composition of prebiotic and probiotic may offer additive benefits, but high-quality, large RCTs with standardized endpoints are still needed. Recent systematic reviews caution against overgeneralized claims and emphasize targeted, evidence-based applications.

#### **7.3 Fermented Foods and Diet-First Approaches**

Rather than relying solely on supplements, increasing whole-food fermented products and dietary fibers can foster sustained ecological shifts. Food-based interventions change substrate landscapes and support native beneficial microbes, often better than transient colonization by supplement strains.

#### **7.4 Focal Microbiota Transplantation (FMT) and Emerging Therapies**

FMT is established for recurrent *C. difficile* but remains experimental for many other conditions. Precision microbiome therapeutics defined microbial consortia, postbiotics are in development and may offer safer, more controllable approaches to restore functional eubiosis.

#### **7.5 Clinical and Public Health Implications**

Disease prevention and management. Diets that support SCFA production and microbial diversity have high in fiber and plant diversity are promising adjuncts to prevent metabolic and inflammatory diseases. For patients with IBD, individualized strategies balancing fermentable fibers with symptom control are required; in metabolic disease, MD-type patterns show consistent benefits.

#### **7.6 Personalized nutrition caveats**

Inter-individual variability in microbial response to diet is large. Baseline microbiome, host genetics, age, medication use (especially antibiotics and proton pump inhibitors), and lifestyle

affect outcomes. Predictive algorithms are improving but not yet fully mature for routine clinical translation.

## **8. Practical Dietary Strategies to Promote Eubiosis (Evidence-based)**

**8.1 Increase dietary fibre intake:** Whole grains, legumes, fruits, vegetables, nuts. Variety supports a broader set of saccharolytic microbes.

**8.2 Adopt a Mediterranean-style pattern:** Emphasize plant foods, olive oil, fish, and limited red/processed meat — linked to higher diversity and beneficial metabolites.

**8.3 Limit ultra-processed foods and added sugars:** These displace fiber and include additives that may disrupt mucosal integrity.

**8.4 Include fermented foods regularly:** Yogurt, kefir, fermented vegetables can provide live microbes and fermentation products that support microbiome function.

**8.5 Prefer unsaturated fats over saturated fats:** olive oil, nuts, and seeds rather than excessive saturated fats to favor healthier bile acid profiles and microbial composition.

**8.6 Prudent probiotic use:** Use strain-specific probiotics for indicated conditions (e.g., *Saccharomyces boulardii* for some antibiotic-associated diarrhea) rather than generic “immune-boosting” claims (6).

## **9. Conclusion**

In conclusion, maintaining gut microbial balance is fundamental to overall health and disease prevention. The human gut microbiome plays a pivotal role in maintaining metabolic, immune, and neurobehavioral homeostasis. An imbalance in this complex microbial ecosystem—termed dysbiosis—has been linked to a wide range of pathological conditions, including obesity, diabetes, inflammatory bowel disease, and even neuropsychiatric disorders. Restoration of gut balance through dietary modulation, probiotic and prebiotic supplementation, faecal microbiota transplantation, and emerging post-biotic formulations represents a promising therapeutic frontier.

Achieving a healthy gut microbiome is not a single intervention but a multifactorial process influenced by diet, lifestyle, environment, and host genetics. Recent advances in omics technologies and personalized nutrition have opened new avenues for targeted microbiome modulation. However, standardization of probiotic strains, dosage forms, and long-term safety assessments remain key challenges for clinical translation.

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