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# GUT MICROBIOTA: COMPOSITION, INFLUENCING FACTORS, DYSBIOSIS AND THERAPEUTIC MODULATION IN HUMAN HEALTH AND DISEASE

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## ABSTRACT

The gut microbiota plays a vital role in maintaining human health by regulating immune responses, metabolism, and protection against pathogens. It supports nutrient digestion, produces short-chain fatty acids (SCFAs) and synthesizes essential vitamins such as biotin, folate, and vitamin K. The major microbial phyla include Firmicutes, Proteobacteria, Actinobacteria, Verrucomicrobia, and Archaea. The composition and diversity of the gut microbiome are influenced by factors such as age, diet, medication use, lifestyle and gender. Disruption of this balance, referred to as dysbiosis, is linked to various non-communicable diseases, including inflammatory bowel disease (IBD), colorectal cancer, obesity, and autoimmune disorders such as celiac disease and rheumatoid arthritis. Dysbiosis contributes to immune dysfunction, inflammation, and metabolic disturbances through altered SCFA production and increased intestinal permeability. Therapeutic strategies including dietary interventions, prebiotics, probiotics, antibiotics, and fecal microbiota transplantation (FMT) are being explored to restore microbial balance. Among these, FMT has shown notable success in treating recurrent *Clostridium difficile* infections, highlighting the potential of microbiota-based therapies. Overall, preservation of a healthy gut microbiome is essential for immune and metabolic homeostasis and may serve as a promising approach for the prevention and management of chronic diseases.

**Keywords: Colorectal cancer, Dysbiosis, Fecal microbiota transplantation, Probiotics**

## INTRODUCTION

The gut microbiota carries out multiple essential functions that help to maintain human health. These intestinal bacteria contribute to the maturation of the immune system, defend against harmful pathogens, participate in xenobiotic metabolism and assist in carbohydrate digestion **[1, 2]**. Fermentation of carbohydrates by gut microbes generates short-chain fatty acids (SCFAs), mainly propionate, butyrate and acetate **[3]**. The composition and quantity of these SCFAs depend on both the level of carbohydrate consumption and the microbial capacity to colonize the intestine. Among the SCFAs, butyrate plays a significant role: it serves as the primary energy source for colonocytes, supports the synthesis of vitamin K, biotin and folate, induces apoptosis, exerts anti-inflammatory effects and enhances insulin sensitivity by functioning as a potent histone deacetylase inhibitor **[4]**.

## COMPOSITION OF INTESTINAL MICROBIOTA

Colonization of the gastrointestinal tract by microorganisms commences immediately after birth and continues to undergo compositional shifts throughout life. Despite substantial inter-individual variation, a conserved subset of microbial taxa and genes referred to as the core gut microbiota and core microbiome is consistently observed across humans. High-throughput profiling of 16S rRNA gene sequences has revealed that the dominant bacterial phyla in the gut include Firmicutes, Actinobacteria, Bacteroidetes, Verrucomicrobia, Proteobacteria and Archaea with Bacteroidetes and *Firmicutes* constituting the majority of the community **[5]**. In contrast, phyla such as Cyanobacteria, Fusobacteria and Spirochaetes are detected at relatively low abundance**.** Age is one of the characteristics that may contribute to the dominance of bacteria at the genus level. *Bacteroides, Alistipes, Sporobacter, Dorea, Faecalibacterium, Roseburia* and *Ruminococcus* are more common in older individuals than in younger ones **[6]**. The gut microbiota is primarily classified into three enterotypes, each of which is dominated by a distinct species (*Bacteroides, Prevotella* or *Ruminococcus*) and functions of respective genera represented in **Table 1**. These enterotypes are independent of human race, gender, age or nationality **[7]**.

**Table 1: Major Gut Microbial Flora and Their Functions**

| Taxonomic Group | Representative Genera | Functional Role in the Gut |
|---|---|---|
| *Firmicutes* | *Clostridium, Ruminococcus, Roseburia, Faecal bacterium* | Production of short-chain fatty acids (e.g., butyrate) and maintenance of intestinal barrier integrity. |
| *Bacteroidetes* | *Prevotella, Bacteroides* | Breakdown of dietary polysaccharides and host-derived glycans. |
| *Proteobacteria* | *Escherichia, Desulfovibrio* | Participation in nitrate and sulfate reduction and opportunistic pathogenicity under dysbiosis. |
| *Actinobacteria* | *Bifidobacterium* | Carbohydrate fermentation and support of host immune modulation. |
| *Archaea* | *Methanobrevibacter* | Consumption of hydrogen and facilitation of methanogenesis to stabilize fermentation pathways. |
| *Verrucomicrobia* | *Akkermansia* | Regulation of mucus layer turnover and enhancement of mucosal health. |

## KEY FACTORS INFLUENCING THE HUMAN GUT MICROBIOTA

### 1. Gender

Evidence regarding sex-dependent variation in gut microbiota remains inconsistent. Some investigations report no appreciable compositional differences between males and females. In contrast, studies using gnotobiotic mice inoculated with human gut microbiota have shown that male and female hosts support distinct microbial assemblages. In Japanese subjects, *Prevotella*, *Megamonas* and *Fusobacteria* were more frequently detected in males, whereas *Bifidobacterium*, *Ruminococcus* and *Akkermansia* were more common in females. Comparable patterns were reproduced in gonadectomised and sex-hormone-supplemented mice, implicating sex hormones as a potential driver of these microbiota distinctions **[8]**.

### 2. Age

The gut microbiota exhibits substantial age-related dynamics. Initial colonisation begins at birth and proceeds through defined stages across infancy, childhood, adulthood and later life. In the early postnatal phase, the microbial community is relatively unstable and poorly diversified, but undergoes marked reorganisation during breastfeeding and again once solid foods are introduced. By about 2–3 years of age, the composition generally resembles a stable, adult-like profile. Perturbations during this early developmental window have been associated with an elevated risk of metabolic and immune-mediated diseases in later life. Although the microbiota remains amenable to change beyond this period, its capacity to re-establish equilibrium after disruptions largely depends on the functional stability of its core microbial members **[9]**.

### 3. Diet

Diet is a principal determinant of gut microbiota composition. Infants who are

breastfed typically exhibit a *Bifidobacterium*-dominated community whereas formula feeding yields a more heterogeneous microbiota enriched in Enterobacteriaceae and Enterococci. Population-level dietary habits further modulate microbial structure; fibre-rich, plant-based diets promote Bacteroidetes such as *Prevotella* and *Xylanibacter*, whereas protein- and fat-rich Western diets favour Enterobacteriaceae. Additionally, sustained dietary exposure can confer specialised metabolic functions, exemplified by the acquisition of seaweed-degrading enzymes by the Japanese gut microbiota from marine bacteria **[10]**.

**4. Medications**

Extended or recurrent administration of broad-spectrum antibiotics substantially decreases microbial richness and disrupts the ecological stability required for maintaining immune homeostasis. This antibiotic-induced dysbiosis can heighten susceptibility to inflammatory and autoimmune disorders. In parallel, the gut ecosystem can act as a niche for the horizontal dissemination of antibiotic-resistance determinants, contributing to the formation of multidrug-resistant microbial pools **[11]**.

**5. Lifestyle**

Advances in public health, including immunization, enhanced clinical care and the widespread implementation of handwashing measures, have substantially reduced the incidence of infectious diseases. However, the parallel adoption of Western lifestyle practices, characterized by the consumption of highly processed foods, reduced contact with environmental microbes, and predominantly indoor living, has been linked to an increased prevalence of immune-mediated disorders, such as allergic and inflammatory diseases. According to the hygiene hypothesis, insufficient microbial exposure during early developmental windows compromises immune system education, predisposing individuals to immune dysregulation and chronic inflammatory conditions in later life **[12]**.

**PATHOLOGICAL CONDITIONS ASSOCIATED WITH GUT MICROBIOTA ALTERATIONS**

- **Inflammatory Bowel Disease (IBD)**

Ulcerative colitis (UC) and Crohn's disease (CD) are major IBD subtypes in which gut dysbiosis contributes to disease in genetically predisposed individuals. Although mechanisms remain unclear. IBD is consistently associated with reduced microbial diversity and impaired anti-inflammatory regulation e.g., transforming growth factor-β, interleukin-10 (IL-10) resulting in sustained mucosal inflammation. Metagenomic profiles in IBD show loss of beneficial taxa such as

*Roseburia, Dorea*, *and Ruminococcus obeum* and enrichment of disease-associated species, including *Ruminococcus gnavus, Escherichia coli* and *Clostridium clostridioforme* in CD, and *Bifidobacterium breve* and *Clostridium symbiosum* in UC. Functional shifts include increased microbial enzymes linked to dysmetabolism compared with healthy microbiota. Serotonin (5-HT) released from enterochromaffin cells also modulates microbial composition. Elevated mucosal 5-HT enhances colitogenic states via direct microbial effects and β-defensin induction. Tryptophan hydroxylase1-deficient mouse models further demonstrate that serotonergic signalling shapes microbiota structure, implicating a 5-HT–microbiota axis in IBD pathogenesis **[13]**.

- **Malignancy**

Gut microbiota dysregulation is increasingly linked to colorectal cancer (CRC), with diet being a major contributing factor. Dietary influences can trigger DNA damage, inflammatory signalling, proto-oncogene activation and obesity. Thereby reshaping the microbiota and promoting carcinogenesis. CRC mucosa shows enrichment of Corio Bacteriaceae (e.g. *Slackia, Collinsella*) and depletion of Enterobacteriaceae such as *Citrobacter, Shigella, Serratia* and *Salmonella*. Probiotic interventions in animal models (e.g. *Bifidobacterium longum, B. breve, and Lactococcus lactis*) have demonstrated antitumor effects by reducing colonic oxidative stress and inflammation **[14]**.

- **Obesity**

The gut microbiota influences energy balance by fermenting indigestible carbohydrates into SCFA including butyrate, acetate, and propionate) which fuel hepatic gluconeogenesis/lipogenesis and provide energy to colonocytes. Microbial metabolites also modulate appetite and endocrine pathways (e.g., reduced GLP-1 in obesity promotes insulin resistance). Obese individuals typically exhibit a higher Firmicutes/Bacteroidetes (F/B) ratio enrichment of *Fusobacteria, Firmicutes, Proteobacteria and Lactobacillus reuteri*, and depletion of *Akkermansia, Methanobrevibacter smithii and Faecalibacterium prausnitzii*. An elevated F/B ratio (>3.0) is a consistent marker of obesity-associated dysbiosis and correlates with metabolic disorders including Type 2 diabetes, gout and dyslipidaemia. Enhanced energy extraction by Firmicutes and fibre-linked depletion of Bacteroidetes contribute to systemic inflammation and metabolic dysregulation underlying obesity **[15]**.

**AUTOIMMUNE DISEASES**

- **Celiac disease**

The condition arises from an abnormal immune response to gluten proteins

found in barley, wheat and rye in genetically predisposed individuals carrying Human leukocyte antigen-DQ2 or Human leukocyte antigen-DQ8 haplotypes. Alterations in gut microbial composition, including reduced levels of *Bifidobacterium*, *Lactobacillus*, and *Faecalibacterium prausnitzii*, along with increased *levels of Bacteroides, Prevotella, and E. coli, have been associated with celiac disease,* indicating a possible microbial contribution to its pathogenesis **[16]**.

- **Rheumatoid Arthritis**

  Gut microbiota alterations are increasingly recognized as potential contributors to rheumatoid arthritis (RA) a chronic autoimmune disorder of uncertain origin. Experimental studies in gnotobiotic mice and clinical observations suggest that imbalances in gut bacterial populations may promote inflammatory pathways involved in RA. Notably, patients with early untreated rheumatoid arthritis exhibit elevated levels of *Prevotella copri* and reduced *Bacteroides* species compared to healthy individuals, supporting a link between intestinal dysbiosis and disease onset **[17]**.

- **Non-alcoholic Fatty Liver Disease (NAFLD) and Non-alcoholic Steatohepatitis (NASH)**

  The human gut contains diverse microbial communities, primarily from the phyla *Firmicutes*, *Bacteroidetes*, *Proteobacteria* and *Archaea* with *Bacteroides* being one of the dominant genera. The liver and gut are closely linked through the portal circulation, allowing gut-derived toxins and endotoxins to influence liver function. Gut dysbiosis increased intestinal permeability, and endotoxemia contribute to liver inflammation and the development of NAFLD, which includes conditions such as steatosis, NASH, fibrosis, and cirrhosis. Obesity, type 2 diabetes, and high-fat diets play major roles in disease progression. In NASH microbial imbalance activates toll-like receptor 4 (TLR4), triggering hepatic inflammation. Western-style diets high in fat, animal protein and sugar as well as excessive fructose intake, further promote lipogenesis, triglyceride synthesis and hepatic fat accumulation, thereby enhancing the risk of steatosis and fibrosis **[18]**. various diseases and their respective mechanisms listed in **Table 2**.

**Table 2: Gut Microbiota Alterations in Major Non-Communicable Diseases**

| Category | Disease | Microbial Changes | Mechanism |
|---|---|---|---|
| **Vascular** | **Atherosclerosis** | **↑*Clostridia*, *Proteus*** | **Conversion of trimethylamine to trimethylamine N-oxide promotes plaque formation.** |
| **Metabolic** | **Obesity** | **↑ *Firmicutes*, ↓ *Bacteroidetes*** | **Altered energy harvest and low-grade inflammation.** |
| **Malignancy** | **Colorectal cancer** | **↑ *H. pylori*, *E. faecalis*** | **Pro-inflammatory and genotoxic microbial activity.** |

## THERAPEUTIC MODULATION OF GUT MICROBIOTA

- **Diet**

  A study conducted on mice fed a high-fat, carbohydrate-free diet demonstrated that antibiotic treatment resulted in significant alterations in gut microbiota and endotoxemia. Faecal analysis of antibiotic-treated mice revealed a reduction in endotoxin levels. High-fat feeding enhances intestinal permeability and reduces the expression of epithelial tight junction proteins such as Zonula Occludens-1 (ZO-1) and Occludin. Interestingly, the administration of antibiotics reversed these effects, particularly restoring Occludin expression, indicating that gut bacteria play a crucial role in maintaining intestinal barrier integrity. Moreover, lipid content was found to be elevated in these mice, suggesting that intestinal microflora contribute to energy harvest and metabolism. The high-fat diet also increased levels of inflammatory cytokines and oxidative stress both of which were markedly suppressed following antibiotic treatment. The reduction in adipocyte size and improvement in glucose tolerance further supported a strong relationship between gut microbiota, inflammation and metabolic endotoxemia under high-fat dietary conditions **[19]**.

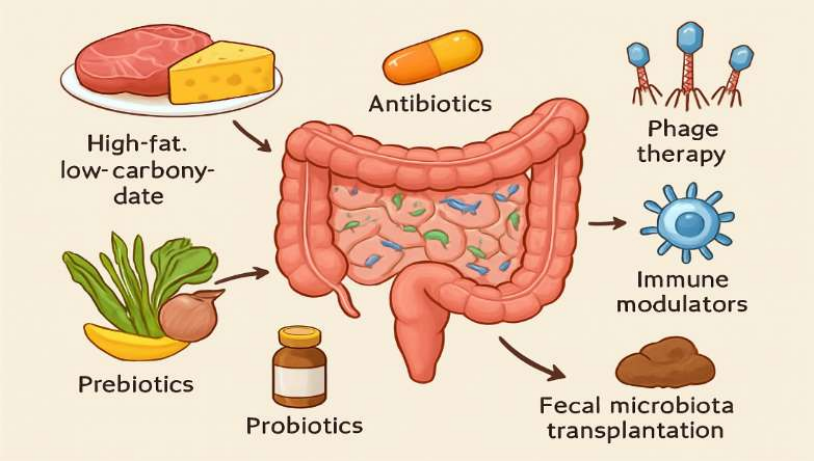


**Figure 1: Therapeutic Approaches to Modulate Gut Microbiota**

- **Prebiotics and Probiotics**

Prebiotics are non-digestible carbohydrates that serve as selective substrates for the fermentation and growth of beneficial gut microorganisms, leading to favourable alterations in intestinal microbiota composition and activity. They escape digestion in the small intestine and are fermented in the colon to produce SCFAs and gases. This fermentation process enhances the growth of *Bifidobacteria*, lowers colonic pH and inhibits the activity of pathogenic microbes. Common prebiotics include fructooligosaccharides (FOS) and glucooligosaccharides. Prebiotics are naturally present in foods such as asparagus, garlic, onion, chicory, wheat, banana, barley and rye. Other forms include isomaltose, Xylo oligosaccharides, galactooligosaccharides, cyclodextrins, raffinose oligosaccharides, soybean oligosaccharides and enzyme-resistant dextrins derived from various plant and starch sources **[20]**.

Recent studies have identified red kidney bean (*Phaseolus vulgaris*) as a potential prebiotic source. Its water-extractable polysaccharide (RKBWEP) promotes the growth of probiotic bacteria such as *Lactobacillus plantarum*, *Bifidobacterium and L. ferment* **[21]**. *In vitro* fermentation of RKBWEP resulted in elevated production of SCFAs, acetic, propionic, and butyric acid which peaked after 48 hours, indicating its strong prebiotic potential in modulating gut microbiota and supporting intestinal health depicted in **(Figure 1)**.

Probiotics are live microorganisms that, when administered in adequate amounts confer health benefits to the host. In contrast, prebiotics are non-digestible dietary components that selectively stimulate the growth or activity of beneficial gut bacteria, thereby enhancing host health. Both probiotics and prebiotics have demonstrated therapeutic potential in various gastrointestinal disorders, including traveller's diarrhoea, acute diarrhoea, radiation-induced diarrhoea and antibiotic-associated diarrhoea **[22]**. Additionally, their benefits extend beyond the gut, showing efficacy in managing non-alcoholic fatty liver disease, necrotising enterocolitis, hepatic encephalopathy and allergies. Most probiotics are derived from Gram-positive bacterial species, although a few Gram-negative strains are also employed **[23]**.

- **Antibiotic Therapy**

Antibiotic administration is a common approach to modify gut microbiota

composition. Studies show that antibiotics can improve insulin signalling in high-fat diet–fed mice and enhance anti-tumor responses in melanoma and lung cancer models by increasing $CD8^+$ T-cell infiltration and IFN-γ expression **[24]**. Targeted antibiotics such as metronidazole have been shown to eliminate *Fusobacterium* in CRC**,** reducing tumor growth and proliferation. These findings suggest that microbiota modulation through antibiotics may be useful in managing microbial-associated cancers**,** although broad-spectrum antibiotics can disrupt healthy gut flora. Therefore, selective antimicrobial approaches are preferred. Antibiotics also exhibit potential as adjuvants in immunotherapy. In animal models of pancreatic cancer, CRC, and melanoma, gut microbiota depletion inhibited tumor progression by enhancing anti-tumor immunity and promoting effector T-cell infiltration**.** Clinically, *H. pylori* eradication using clarithromycin and amoxycillin in early gastric cancer patients has been associated with a reduced risk of recurrent cancer and improved gastric mucosal health**,** highlighting the therapeutic relevance of antibiotic-based microbiota modulation **[25]**.

- **Immune modulators and phage therapy**

The immune system plays a fundamental role in regulating gut microbiota and influencing disease progression. In disorders like IBD, an immune imbalance significantly affects microbial composition. Experimental studies in mice lacking immune modulators such as IL-10 have revealed distinct alterations in gut microbiota, emphasizing the connection between immune regulation and microbial balance. While the application of immune modulators to modify gut microbiota is still under investigation, it shows therapeutic potential. Likewise, phage therapy employs bacteriophages to selectively target and lyse specific bacterial strains offering a promising but underexplored approach. However, issues such as phage resistance and limited experimental evidence remain major challenges for its clinical use **[26]**.

- **Fecal microbiota transplantation**

A recent study in England on patients with recurrent *Clostridium difficile* infection reported that faecal microbiota transplantation (FMT) achieved a 94% cure rate, markedly higher than that observed with antibiotic therapy alone or antibiotics combined with bowel lavage. Following FMT, there was a notable increase in beneficial bacterial groups particularly Bacteroidetes and Firmicutes

(Clostridium clusters IV and XIVa), and no relapse was observed. The results were so significant that the trial was terminated early, allowing all remaining participants to receive donor microbiota transplantation due to its superior therapeutic effect. Although FMT has demonstrated excellent clinical outcomes, its safe administration poses significant challenges, particularly regarding the potential transmission of infectious agents. In the referenced study, donor stool samples underwent thorough screening for parasites and enteropathogenic bacteria. Additionally, blood tests were performed to exclude infections such as human T-cell lymphotropic virus, Hepatitis A, B, and C, *Treponema pallidum*, *Entamoeba histolytica*, Epstein–Barr virus (EBV) and cytomegalovirus (CMV). To ensure continued safety, a donor pool was established and rescreened every four months **[27]**. Another major challenge concerns the method of administering faecal transplants. Due to the limited number of clinical studies, it remains uncertain which route of transplantation is the most suitable and how much donor stool is required to achieve optimal therapeutic benefits **[28]**.

The gut microbiota acts as a metabolically active "hidden organ" that maintains immune balance, regulates host metabolism, and defends against pathogens. Disruption of this ecosystem driven by diet, antibiotics, ageing, stress, and sedentary lifestyles, leads to dysbiosis, which is linked to non-communicable diseases such as inflammatory bowel disease, obesity, autoimmune disorders, and colorectal cancer. Although interventions including dietary changes, prebiotics, probiotics, antibiotics, and faecal microbiota transplantation may help restore microbial balance, individual variability and lack of standardized protocols limit clinical application. Future research should prioritize personalized microbiota-based therapies and the development of microbial biomarkers for early disease prediction and targeted treatment.

## CONCLUSION

The gut microbiota plays a vital role in maintaining health by supporting immune function, metabolism, and defense against pathogens. Its imbalance, influenced by diet, age, medications and lifestyle, is linked to several non-communicable diseases such as IBD, obesity, autoimmune disorders and CRC. Therapeutic modulation through diet, prebiotics, probiotics, antibiotics and faecal microbiota transplantation shows great potential in restoring microbial balance and improving health outcomes.

However, further research is needed to standardize treatment protocols, establish safe and effective dosages, and explore personalized microbiota-based therapies for disease prevention and management.

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