Jurnal Biota Prodi Biologi Universitas Islam Negeri Raden Fatah Palembang

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To cite this article before publication: Yusfarani. D. *et al* (2025) The Role of Gut Microbiota in Metabolism and Immune Response: A Literature Review on Metabolic Health. Jurnal Biota. In press
http://jurnal.radenfatah.ac.id/index.php/biota/article/view/25315

The Role of Gut Microbiota in Metabolism and Immune Response: A Literature Review on Metabolic Health

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Abstract

The gut microbiota, a community of microorganisms within the digestive tract, plays a pivotal role in human health by regulating both immune function and metabolism. This review examines the contributions of the gut microbiota to immune responses and metabolic health, as well as its role in preventing chronic diseases. We employed a mixed-methods (qualitative and quantitative) literature review spanning the last 50 years and analyzed 100 articles related to *Gut Microbiota in Metabolism and Immune Response*. The inclusion criteria encompassed all studies focusing on the gut microbiota's impact on metabolic and immune functions, while non-relevant studies were excluded. Our analysis highlights recent findings on the microbiota's ability to produce metabolites such as short-chain fatty acids (SCFAs), which exhibit anti-inflammatory properties and reinforce mucosal integrity. A balanced gut microbiota supports immune regulation, inhibits pathogen colonization, and optimizes metabolic processes. However, lifestyle factors—including high-sugar, low-fiber diets, antibiotic misuse, stress, and insufficient sleep—can disrupt this microbial equilibrium and lead to dysbiosis. Such imbalances can precipitate metabolic and inflammatory disorders, including diabetes, autoimmune diseases, and cardiovascular conditions. This review underscores the importance of maintaining a balanced gut microbiota for disease

prevention and long-term health, as well as the need to raise awareness about the impact of lifestyle choices on gut health.

Keywords: *Dysbiosis; Gut Microbiota; Immune System; Metabolic Health; SCFA.*

Introduction

The gut microbiota, composed of trillions of microorganisms within the digestive tract, plays a fundamental role in maintaining human health [1]. Since advances in next-generation sequencing and metagenomics have allowed for a deeper exploration of gut microbial diversity, it has become evident that these microbial communities are involved not only in nutrient digestion and metabolism but also in modulating immune responses [2,3]. Numerous studies have demonstrated that the gut microbiota produces bioactive metabolites, such as short-chain fatty acids (SCFAs), that help regulate the immune system, maintain mucosal barrier integrity, and limit inflammatory processes [2]. This interaction between the microbiota and the host immune system is crucial for sustaining immunological homeostasis and preventing overgrowth of pathogens.

Despite of the growing body of research indicating the gut microbiota's integral part in overall health, major gaps remain. While previous studies have confirmed the gut microbiota's role in disease etiology, ranging from autoimmune disorders to metabolic syndromes, there is less clarity on how multifactorial lifestyle elements collectively influence microbiota balance over the long term. For instance, it has been established that diets high in sugar and low in fiber can reduce beneficial microbial diversity, yet the precise mechanisms by which these dietary patterns lead to chronic dysbiosis and inflamation remain partially unexplored [4,5]. Similarly, although antibiotic misuse and inadequate sleep have been implicated in disrupting the microbiome, comprehensive investigations that integrate these factors alongside stress and extreme dietary habit are relatively scarce [6,7].

Current evidence also points to the gut microbiota as a mediating factor in mental health via the gut-brain axis, underscoring the broader systemic implications of dysbiosis [8,9]. However, state-of-the-art research highlights the need for further elucidation of how specific microbial shifts contribute to metabolic and immunological dysfunction. Emerging reviews call for integrative approaches, combining metagenomic, metabolomic, and clinical data, to map the causal pathways linking gut

microbial alterations to chronic diseases such as type 2 diabetes, inflammatory bowel disease, and cardiovascular disorders [10,11].

Lifestyle behaviors, such as the consumption of fast food, high-sugar/low-fiber diets, and irregular sleep, can significantly perturb gut microbial communities, yet public awareness of these connections remains limited [12]. Under normal conditions, a balanced microbiota can outcompete pathogens via nutrient competition and immune modulation [13]. However, an imbalance (dysbiosis) often triggers chronic inflammatory states, enhances gut permeability ("leaky gut"), and increases the risk of a host of systemic diseases [14]. Recent findings indicate that dysbiosis can induce metabolic disturbances by influencing insulin resistance and oxidative stress [15]. Nevertheless, the precise interplay between gut microbial composition, immunomodulation, and metabolic pathways is still being unraveled, reinforcing the need for in-depth research in this area.

Given these critical gaps and the mounting evidence of the microbiota's far-reaching effects, this review aims to examine the mechanisms by which the gut microbiota sustains immunological balance and metabolic health, with a focus on preventing chronic diseases. By synthesizing key insights from recent and foundational studies, we seek to bridge the current knowledge gaps and shed light on targeted strategies, including dietary interventions, probiotic use, and lifestyle modifications, to uphold gut microbial homeostasis and bolster long-term well-being.

Materials and Methods

Research Objectives

This study aims to analyze the role of gut microbiota and its metabolites in metabolism, immune responses within the gut, and its relationship to overall metabolic health.

Research Methods

Studies were included if they focused on the gut microbiota's role in metabolic health and immune responses, used original research designs or relevant reviews/meta-analyses, involved human or animal models with clear metabolic or immune outcomes, and were published in English within the last 50 years, contributing to a total of 100 journal articles. We excluded studies that did not directly address gut microbiota in metabolism or immunity, lacked rigorous methods (e.g., no clear microbiota analysis), were duplicates, or had no full text available.

In this review, no meta-analysis was conducted due to the heterogeneity of study designs, populations, and outcome measures. Instead, we employed a narrative synthesis to integrate both quantitative and qualitative findings. Additionally, we did not follow a formal systematic review protocol (e.g., PRISMA or PROSPERO); however, our methodology was structured, applying predefined inclusion and exclusion criteria, and adhering to recognized standards for literature reviews.

Result and Discussion

Result

Table 1. Organs and Identifiable Human Microbiota [16]

Organ	Microbiota	References
Intrauterine (in the womb)	Microbiota from placental tissue, amniotic fluid, and meconium (baby's first stool): ➢ *Lactobacillus crispatus* ➢ *Bifidobacterium* ➢ *Enterococcus* ➢ Staphylococcus ➢ Streptococcus	[17–21]
Infant gut (at birth)	Microbiota from mother's feces and vaginal microbiota (through normal delivery) ➢ *Bacteroides* ➢ *Prevotella* ➢ *Ruminococcus gnavus* ➢ *Clostridium difficile* ➢ *Eubacterium* ➢ *Bifidobacterium* ➢ *Lactobacillus* ➢ *Enterococcus faecium* ➢ *Escherichia coli*	[22–31]
Infant gut (via cesarean birth	Microbiota from skin and other environments ➢ Staphylococcus (e.g., *S. epidermidis*, *Staphylococcus aureus*) ➢ Corynebacterium ➢ Propionibacterium (now often classified as *Cutibacterium*) ➢ Other hospital-acquired or environmental microbes (e.g., certain strains of *Enterococcus* or *Enterobacter*).	[32–38]
Adult gut	Thousands of bacterial species, mainly from 8 of 55 primary bacterial phyla	[39–52]

	➢ Firmicutes (e.g., *Ruminococcus*, *Clostridium*, *Lactobacillus*) ➢ Bacteroidetes (e.g., *Bacteroides*, *Prevotella*) ➢ Actinobacteria (e.g., *Bifidobacterium*) ➢ Proteobacteria (e.g., *Escherichia*, *Salmonella*) ➢ Verrucomicrobia (e.g., *Akkermansia muciniphila*) ➢ Fusobacteria (e.g., *Fusobacterium*) ➢ Tenericutes (although often in low abundance) ➢ Euryarchaeota (archaea, like *Methanobrevibacter smithii*, sometimes considered separately from bacteria)	
Gut epithelium	Bacteria promoting antimicrobial peptides (e.g.: α and β defensin, Reg3γ, Ang4): ➢ Bacteroidetes (e.g., *Bacteroides fragilis*). ➢ Firmicutes (e.g., *Lactobacillus, Clostridium species*). ➢ *Akkermansia muciniphila* (Verrucomicrobia) ➢ *Enterococcus faecalis* (Firmicutes) ➢ *Faecalibacterium prausnitzii* (Firmicutes – Clostridiales). ➢ *Escherichia coli* (Proteobacteria – Komensal Strains). ➢ Ruminococcus (Firmicutes – Lachnospiraceae family).	[51,53–60]

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112 **Table 2. Gut Microbiota**

Organ/Area	Microbiota	Main Function	Reference
Gut (Intestinal Tract)	*Faecalibacterium prausnitzii*, *Roseburia intestinalis*, *Anaerostipes butyraticus*, *Bifidobacteria.*	Metabolize complex carbohydrates to produce SCFAs (butyrate, acetate, propionate); regulate immune response, maintain gut barrier integrity.	[57,61–63]
Gut Epithelium	Various gut microbiota as follows: ➢ Bacteroidetes (e.g., *Bacteroides fragilis*) ➢ Firmicutes (e.g., *Lactobacillus, Clostridium species*)	Modulate immune response; SCFAs regulate immune cell function, epithelial cell turnover, anti-inflammatory pathways.	[51,53–60]

	➢ *Akkermansia muciniphila* (Verrucomicrobia) ➢ *Enterococcus faecalis* (Firmicutes) ➢ *Faecalibacterium prausnitzii* (Firmicutes – Clostridiales) ➢ *Escherichia coli* (Proteobacteria – *Komensal Strains*) ➢ *Ruminococcus* (Firmicutes – Lachnospiraceae family).		
Mucosal Layer (Gut Barier)	Firmicutes, *Bifidobacteriaceae*.	Regulate immune tolerance, support epithelial integrity, reduce permeability to pathogens.	[64,65]
Immune cell (Makrofag, DC)	SCFA from gut (*Butirat, Asetat*), the microbiota as follows: ➢ Butirat: *Faecalibacterium prausnitzii, Roseburia spp., Eubacterium rectale, Anaerostipes spp., Clostridium butyricum.* ➢ Asetat: *Bifidobacterium spp., Escherichia coli (non-patogen), Lactobacillus spp., Akkermansia muciniphila.* ➢ Propionat: *Bacteroides spp., Veillonella spp., Prevotella spp.*	SCFAs activate GPR -G Protein-coupled receptor (GPR41, GPR43, GPR109a), modulate proinflammatory cytokines, promote T-reg cell differentiation.	[57,61, 66–72]
Colonocytes (Large	Butyrate-producing bacteria:	➢ Absorb SCFAs to support epithelial cell	[73–79]

Intestinal Cells)	➢ *Faecalibacterium prausnitzii* (Firmicutes – Clostridiales family) ➢ *Roseburia* spp. (Firmicutes – Lachnospiraceae family). ➢ *Eubacterium rectale* (Firmicutes – Lachnospiraceae family). ➢ *Anaerostipes* spp. (Firmicutes – Lachnospiraceae family). ➢ *Butyrivibrio* spp. (Firmicutes – Lachnospiraceae family). ➢ *Clostridium butyricum* (Firmicutes – Clostridiaceae family) ➢ *Ruminococcus* spp. (Firmicutes – Ruminococcaceae family)	health and barrier function; SCFAs enhance anti-inflammatory responses, regulate cell turnover. ➢ anti-inflammatory properties by producing butyrate and modulating immune responses. ➢ Produces butyrate through the fermentation of dietary fiber. ➢ Ferments dietary fiber to produce butyrate and propionate. ➢ Plays a role in gut. ➢ Associated with improved gut barrier function and reduced inflammation. ➢ Often used in probiotic formulations for its beneficial effects on gut health and immune modulation	
Small Intestine	Commensal anaerobic bacteria ➢ *Lactobacillus* spp. (Firmicutes – Lactobacillaceae family) ➢ *Bifidobacterium* spp. (Actinobacteria – Bifidobacteriaceae family) ➢ *Clostridium* spp. (Firmicutes – Clostridiaceae family)	➢ Maintain low pH, support healthy epithelial function, prevent pathogen colonization through SCFA production. ➢ Help in lactose digestion, production of lactic acid, and inhibition of pathogen colonization.	[52,56,68,71, 72,80–83]

	➢ *Prevotella* spp. (Bacteroidetes – Prevotellaceae family) ➢ *Veillonella* spp. (Firmicutes – Veillonellaceae family) ➢ *Peptostreptococcus* spp. (Firmicutes – Peptostreptococcaceae family) ➢ *Akkermansia muciniphila* (Verrucomicrobia family) ➢ *Fusobacterium* spp. (Fusobacteriaceae family) ➢ *Enterococcus* spp. (Firmicutes – Enterococcaceae family)	➢ Ferment complex carbohydrates and produce beneficial short-chain fatty acids (SCFAs) such as acetate. ➢ essential energy source for intestinal epithelial cells. ➢ associated with individuals consuming high-fiber diets. ➢ maintaining gut pH balance and reducing the accumulation of harmful metabolites. ➢ Play a role in protein metabolism and maintaining gut homeostasis. ➢ Specializes in mucin degradation, which supports intestinal barrier integrity. ➢ Can act as opportunistic pathogens under dysbiotic conditions but are part of the normal commensal flora. ➢ Play a role in bile salt metabolism and resistance to bile acids.	
Large Intestine (Colon)	➢ Bifidobacterium (Phylum: Actinobacteria): *Bifidobacterium longum, Bifidobacterium breve,*	➢ Major site of SCFA production, influencing immune response, inflammation regulation, and gut health.	[57,84–90]

	Bifidobacterium bifidum, *Bifidobacterium adolescentis*. ➢ Firmicutes (Phylum: Firmicutes): Faecalibacterium prausnitzii (Order: *Clostridiales*), Roseburia spp. (Order: *Lachnospiraceae*), Clostridium spp. (Order: *Clostridiaceae*), Eubacterium rectale (Order: *Lachnospiraceae*), Blautia spp. (Order: *Lachnospiraceae*) ➢ Ruminococcus spp. (Order: *Ruminococcaceae*): ➢ Lactobacillus spp. (Order: *Lactobacillales*)	➢ produce acetate and lactate, which help maintain gut health. ➢ involved in fiber digestion, SCFAs production (especially butyrate), and metabolic regulation. ➢ degrading resistant starch and plant fibers, contributing to overall gut health ➢ Production of butyrate: Supports colonocyte health and reduces inflammation. ➢ -Fermentation of dietary fibers: Helps in breaking down plant-based fibers into SCFAs. ➢ -Regulation of metabolism: Firmicutes have been linked to energy extraction from food, which can influence body weight. ➢ - Immune modulation: Interact with intestinal immune cells to maintain homeostasis and prevent excessive inflammation.

Discussion

The gut microbiota is essential in the development of human immunity (Table 1). Bacteria within the microbiota contribute to immune system maturation by modulating both innate and adaptive immune responses from an early age. Fermentation of fiber by

the microbiota produces SCFAs, which act as anti-inflammatory agents, support gut epithelial health, and protect against pathogen invasion. Additionally, the microbiota regulates immune responses by stimulating antibody and immune cell production, functioning as a "shield" against pathogens [4].

Gut microbiota also impacts metabolism, producing SCFAs from fiber and complex carbohydrate fermentation, supporting neurotransmitter function, and enhancing nutrient absorption (Table 2). Short-chain fatty acids (SCFAs) are formed through the anaerobic fermentation of complex carbohydrates by gut microbes. Acetate is produced from acetyl-CoA via acetyl phosphate. Propionate is generated through the succinate pathway or the acrylate pathway. Butyrate is produced through a series of reactions from acetyl-CoA to butyryl-CoA, which is then converted into butyrate. The main factors influencing SCFA biosynthesis include substrate availability, the composition of the microbial community, and gut environmental conditions such as pH. By understanding the pathways involved in SCFA formation, we can determine how nutritional interventions (for example, increasing fiber intake) or microbiota management can enhance the proportion of specific SCFAs that benefit health. Microbiota imbalance, or dysbiosis, can increase HPA axis activity during stress, affecting blood pressure, blood sugar levels, and other metabolic disturbances [5]. Types of Microorganisms in Gut Microbiota shows in Table 3 [6] :

Table 3. Types of Microorganisms in Gut Microbiota

No	Organ	Microbiota	Roles
1.	Gut Bacteria	*Lactobacillus* *Clostridium*	Involved in fiber fermentation producing short-chain fatty acids (SCFAs)
		Bacteroides	Break down complex polysaccharides, playing a key role in fiber metabolism
		Actinobacteria	Participates in carbohydrate metabolism
		Proteobacteria; *Escherichia coli*	Plays a metabolic role; includes aids in digestion and vitamin K synthesis
		Verrucomicrobi; *Akkermansia muciniphila*	Involved in mucus metabolism
2	Gut Fungi	*Saccharomyces*	acts as a probiotic,

		cerevisiae	
		Candida spp	pathogenic in dysbiosis
3	Gut Archaea	*Methanobrevibacter smithii*	involved in methane gas production from hydrogen fermentation
4	Gut Protozoa	*Entamoeba histolytica*	cause intestinal infections on human
		Giardia lamblia	cause intestinal infections on human
5	Gut Viruses		help control bacterial populations in the gut, influencing microbial interactions and potentially preventing pathogenic infections.
		Bacteriophages	

The Importance of Gut Microbiota Balance for Metabolic Health:

An imbalance in gut microbiota can lead to inflammation and metabolic diseases such as diabetes, dyslipidemia, and hyperuricemia. Thus, maintaining a balanced microbiota is crucial for metabolic health. Various lifestyle habits can disrupt this balance, known as dysbiosis, and negatively impact gut health:

1. Processed Foods High in Sugar, Saturated Fats, and Low in Fiber

Processed foods that are high in sugar, high in saturated fats, and low in fiber are types of food that typically undergo extensive processing and the addition of various additives. This processing can include preservation, the addition of chemical substances, refining, and even modifications to texture or flavor. These foods promote the growth of pathogenic bacteria while suppressing beneficial microbes. Excessive sugar, for example, can increase pathogenic bacteria like *Clostridium* and *Escherichia coli*, potentially causing intestinal inflammation [7].

2. Antibiotics

While antibiotics kill infection-causing bacteria, they also eliminate beneficial gut microbiota, reducing bacterial diversity. Long-term or repeated antibiotic use can increase dysbiosis risk, recurrent infections, and disrupt long-term microbial [8].

3. Low Fiber Intake

Fiber is a key food source for gut microbiota, fermenting into SCFAs that are crucial for gut mucosal health and immune response. Lack of fiber can reduce SCFA-producing bacteria like *Faecalibacterium prausnitzii* and *Bifidobacteria*, which act as natural anti-inflammatories. A diet low in fiber (and often high in simple sugars and

fats) frequently leads to a decrease in gut microbiota diversity. The reduction of fiber-degrading bacteria can create space for bacteria more tolerant of a high-fat/sugar diet, such as certain species within the Firmicutes phylum [9,10].

4. Stress

Stress elevates cortisol production, which negatively impacts gut microbiota. Cortisol increases gut permeability, allowing harmful microbes to enter the bloodstream and trigger inflammatory responses. Chronic stress may also reduce beneficial bacteria and heighten dysbiosis risk. Stress can lower the population of beneficial short-chain fatty acid (SCFA)-producing bacteria, such as Lactobacillus, Bifidobacterium, and *Faecalibacterium prausnitzii*. A reduction in these bacteria can weaken the intestinal mucosal barrier and decrease the production of crucial metabolites for gut health [5,11].

5. Smoking and Excessive Alcohol Consumption

Smoking alters gut microbiota composition by reducing beneficial bacteria and fostering pathogenic microbes. Excessive alcohol similarly increases gut permeability, inflammation, and microbial imbalance. The chemicals in cigarette smoke (including nicotine, tar, and free radicals) can compromise the tight junctions between intestinal epithelial cells, making them "leaky." This weakened barrier allows endotoxins and pathogenic microbes to cross into the bloodstream, increasing systemic inflammation. Alcohol and its metabolites (like acetaldehyde) also damage the gut lining. They disrupt tight junction proteins, impairing the gut's barrier function and enabling harmful substances to leak through the intestinal wall [12,13].

6. Insufficient Sleep or Irregular Sleep Patterns

Poor sleep disrupts the body's circadian rhythm, which also regulates gut microbiota activity. Inadequate sleep can reduce microbial diversity, increase pathogenic bacteria, and impair food metabolism [15]. The gut microbiota follows daily (circadian) cycles in growth and activity. Poor or irregular sleep disrupts these cycles, reducing beneficial microbial populations and promoting the growth of potentially pathogenic species. Chronic sleep loss increases cortisol (a stress hormone) and disrupts melatonin and appetite-regulating hormones (like ghrelin and leptin). These imbalances alter the gut environment, potentially increasing gut permeability and inflammation, and reducing the overall resilience of beneficial microbes.

7. Lack of Physical Activity

Physical activity boosts beneficial bacteria like *Akkermansia muciniphila*, which is important for gut mucosal health and metabolic function [91,92]. Physical exercise can promote blood flow and oxygenation within the gastrointestinal tract. This enhanced circulation supports a healthier gut environment, making it more hospitable to beneficial microbes such as *Akkermansia muciniphila*. In contrast, a sedentary lifestyle may diminish these supportive factors, leading to decreased populations of beneficial bacteria.

Avoiding these behaviors can help maintain a healthy gut microbiota balance, which is vital for immune and metabolic health. A balanced gut microbiota aids in digestion, immune regulation, metabolism, and pathogen protection. Imbalance, or dysbiosis, increases the risk of multiple diseases. Below is an analysis of the importance of gut microbiota balance and preventive efforts to reduce disease risks related to dysbiosis.

1. Disease Risk Analysis from Gut Dysbiosis

Significant changes in the gut microbiota composition (not merely the appearance or disappearance of certain species) — whether an increase or decrease in the proportion of specific bacterial groups — can trigger or indicate dysbiosis. Essentially, many microbes are present in both healthy and dysbiotic states; however, their relative proportions shift drastically enough to affect physiological functions and increase disease risk. An increase in Proteobacteria, opportunistic Firmicutes species, and inflammation-inducing bacteria, coupled with a decrease in *Bifidobacterium, Lactobacillus, Faecalibacterium, Roseburia, Eubacterium,* and *Akkermansia*, generally represents a dysbiotic pattern associated with various disease risks.

a. Metabolic Disorders: Dysbiosis affects body metabolism, contributing to obesity, type 2 diabetes, and metabolic syndrome. Imbalance disrupts carbohydrate and fat metabolism, increases insulin resistance and reduces cellular insulin sensitivity, heightening the risk of diabetes and obesity [93,94].

b. Autoimmune and Inflammatory Diseases: Dysbiosis impacts immune response, raising the risk of autoimmune and inflammatory diseases like inflammatory bowel disease (IBD), rheumatoid arthritis, and lupus. It can lead to increased gut permeability ("leaky gut"), allowing pathogens and toxins to enter the bloodstream and trigger excessive immune responses [94,95].

c. Mental Health Disorders: Gut microbiota communicates with the brain through the gut-brain axis. Dysbiosis may cause systemic inflammation and affect neurotransmitter production, contributing to mental health issues such as depression, anxiety, and chronic stress. [96,97].

d. Cardiovascular Disease: Certain gut microbiota metabolites can contribute to cardiovascular diseases. For instance, trimethylamine N-oxide (TMAO) from specific foods can increase atherosclerosis, hypertension, and heart disease risk. [94].

2. Prevention and Maintenance of Gut Microbiota Balance

Maintaining a balanced gut microbiota is key to preventing a range of diseases. Some main reasons for this include:

a. Reduced risk of chronic diseases, especially those linked to inflammation and metabolic disorders. A balanced microbiota provides natural protection against pathogens, reducing chronic infection and inflammation risks.

b. A healthy microbiota enables the immune system to effectively recognize and eliminate pathogens while maintaining tolerance toward commensal organisms. This reduces the risk of autoimmune diseases.

c. Gut-brain axis health is supported by a balanced microbiota, helping regulate neurotransmitters like serotonin and dopamine, potentially preventing mental disorders triggered by dysbiosis.

3. Dysbiosis Prevention Strategies

Preventing gut dysbiosis involves lifestyle and dietary changes to support gut health. Recommended strategies include:

a. Fiber: Sources from vegetables, fruits, and whole grains feed beneficial bacteria like *Bifidobacteria* and *Faecalibacterium prausnitzii*, producing SCFAs that benefit gut health and immunity.

b. Antibiotic Use: Antibiotics should be used judiciously and only as prescribed by a doctor for necessary infections.

c. Reducing Processed Foods: Foods high in sugar and saturated fat disrupt microbiota balance by encouraging pathogen growth while reducing such foods helps maintain beneficial bacteria.

d. Physical Activity and Adequate Sleep: Both contribute to microbiota diversity and help reduce stress, positively impacting gut health.

e. Probiotics and Prebiotics: Probiotics found in yogurt or supplements increase beneficial bacteria, while prebiotics in foods like garlic, onions, and asparagus feed these microbes, supporting their growth.

Certain foods can disrupt the balance of gut microbiota and reduce the number of beneficial bacteria. The widespread consumption of fast food across various groups in society highlights a low awareness of its harmful impact on gut microbiota. This consumption leads to a reduction in beneficial gut bacteria and increases the risk of dysbiosis. Antibiotic misuse has become fairly common, with people often taking antibiotics at the slightest discomfort without medical guidance.

Most people do not consume enough fruits, vegetables, or whole grains, which are essential for supporting the growth of beneficial gut bacteria. This trend reflects a general lack of knowledge about the role of fiber in maintaining a healthy gut microbiota. Many are also unaware that stress and poor sleep can negatively impact gut health, potentially causing digestive issues and inflammation.

Moreover, many view products that support gut health as mere trends, without understanding their role in preserving gut microbiota balance. However, few realize that certain habits affect not only organs like the lungs and liver but also disrupt the balance of bacteria in the gut. These diets are often followed without considering their impact on gut microbiota [96].

Beside that, age, residence, gender, and comorbidities are also factors that Influence the gut microbiota composition and function. Several factors, including age, residence, gender, and comorbidities, can influence the composition and function of gut microbiota. Table 4 shows types of microbiota in human gut in relation to age, residence, gender, and comorbidities, also the explanation of it.

Table 4. Types of Microorganisms in Human Gut

No	Category	Condition	Microbiota	Reference
1	Age	Gut microbiota in infants	*Bifidobacteria*	[98]
		Gut microbiota in adults	*Firmicutes*	[85]
			Bacteriodetes	[85]
		Gut microbiota in the elderly	*Clostridium difficile*	[99]
			Bifidobacteria	[100]

			Firmicutes	[85]
2	Residence	Urban residence	-Not mention-	[101]
		Rural residence	*Prevotella*	[102]
3	Gender	Effect of sex hormones	*Bifidobacteria*	[103]
			Lactobacillus	[104]
4	Comorbidities	Obesity	*Firmicutes*	[105]
			Bacteroidetes	[105]
		Type 2 diabetes	*Faecalibacterium prausnitzii*	[106]
		Autoimmune diseases	*Escherichia coli*	[107]

Here is the explanation:

1. *Age. The gut microbiota undergoes significant changes throughout human life, from infancy to old age*

Factors such as birth mode, diet, and environmental exposure shape microbiota composition from an early age.

- Gut microbiota in infants: At birth, the infant's gut is nearly sterile. Initial colonization is influenced by the mode of delivery (vaginal or cesarean section). Vaginally delivered infants acquire a richer microbiota dominated by *Bifidobacteria* from the mother's vaginal flora, whereas cesarean-delivered infants tend to develop a microbiota resembling skin flora. Breastfeeding promotes the growth of *Bifidobacteria*, which plays a crucial role in immune system development and metabolism.
- Gut microbiota in adults: More diverse compared to infants, with dominant phyla such as *Firmicutes* and *Bacteroidetes*. Diet, lifestyle, and environmental exposure play a significant role in maintaining microbiota balance.
- Gut microbiota in the elderly: Decreased microbial diversity, with an increase in opportunistic pathogens such as *Clostridium difficile*. Reduction in *Bifidobacteria* and *Firmicutes* may contribute to chronic inflammation and metabolic disorders.

2. *Residence (Environment and Geography)*

The living environment, such as urban or rural areas, also affects gut microbiota through diet, hygiene, and exposure to environmental microorganisms.

- Urban residence: Tends to have lower microbial diversity due to a diet high in processed foods and low in fiber. Higher antibiotic exposure and sanitation levels can reduce contact with beneficial microorganisms.
- Rural residence: Greater microbial diversity, with bacteria such as *Prevotella*, which are associated with high-fiber and complex carbohydrate diets. Greater exposure to soil and animals enhances microbial diversity.

3. Gender

Although gender differences in gut microbiota are not always prominent, research indicates that sex hormones can influence microbiota composition.

- Effect of sex hormones: Hormones such as estrogen and testosterone can impact microbial diversity and composition. Women tend to have higher levels of *Bifidobacteria* and *Lactobacillus*, which are associated with gut and reproductive health.
- Differences in disease risk related to microbiota: Women are more prone to digestive disorders such as irritable bowel syndrome (IBS), whereas men are more susceptible to metabolic disorders like microbiota-related obesity.

4. Comorbidities (Underlying Health Conditions)

Certain health conditions, such as obesity, diabetes, and autoimmune diseases, significantly affect gut microbiota composition.

- Obesity: An increase in bacteria from the *Firmicutes* phylum compared to *Bacteroidetes*, contributing to enhanced energy absorption from food. Production of inflammatory metabolites such as lipopolysaccharides (LPS), which trigger chronic inflammation.
- Type 2 diabetes: Gut microbiota associated with insulin resistance tends to have an increase in pathogenic bacteria and a decrease in SCFA (short-chain fatty acid)-producing bacteria such as *Faecalibacterium prausnitzii*.
- Autoimmune diseases (e.g., Crohn's disease and ulcerative colitis): Dysbiosis characterized by reduced microbial diversity and an increase in pro-inflammatory bacteria such as pathogenic *Escherichia coli*.

Maintaining gut microbiota balance is essential for overall health, including metabolic, immune, and mental well-being. Adopting a healthy lifestyle and avoiding risk factors for dysbiosis can significantly reduce the risk of chronic diseases associated with microbiota imbalance. Preventing dysbiosis may also extend lifespan and improve quality of life.

Conclusion

Based on the literature, gut microbiota plays a crucial role in regulating immune responses and metabolic health. Types of microorganisms in gut microbiota—such as bacteria (*Lactobacillus*, *Bacteroides*, *Bifidobacterium*), fungi (*Saccharomyces cerevisiae*), archaea (*Methanobrevibacter smithii*), protozoa (*Entamoeba histolytica*), and viruses (bacteriophages) interact to influence immunity and overall health. Balanced gut microbiota is a primary factor for human health, while imbalances can trigger metabolic and immunological diseases. Although many studies demonstrate a consistent role of the microbiota in metabolism and immune function, some findings remain contradictory or inconclusive. These discrepancies generally reflect the complex interactions between microbes and their host, various external factors (diet, lifestyle, environment), and variations in research methodologies. This situation highlights the need for more comprehensive and controlled studies, as well as a personalized approach to assessing microbiota health and implementing suitable interventions.

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