International Journal of Basic and Applied Sciences, 15 (2) (2026) 46-54

SPC

International Journal of Basic and Applied Sciences

Website: www.sciencepubco.com/index.php/IJBAS
https://doi.org/10.14419/cshmpj82
Research paper

Gut Microbiota Profiles in Common Non-Communicable Diseases- A Narrative Review

Thiagarajan Sowmiya[1], Emmanuel Bhaskar[2], Veeraraghavan Gayathri[3],
Parthasarathy Mohanalakshmi[4], Santhi Silambanan[5]*

[1] Department of Biochemistry, Sri Ramachandra Institute of Higher Education and Research, Porur, Chennai, India 600116.
[2] Department of General Medicine, Sri Ramachandra Institute of Higher Education and Research, Porur, Chennai, India 600116.
[3] CEFTE, Sri Ramachandra Institute of Higher Education and Research, Porur, Chennai, India 600116
[4] Department of Biochemistry, Sri Muthukumaran Medical College Hospital and Research Institute, Kunrathur Road, Chikkarayapuram, Chennai, India 600069.
[5] Department of Biochemistry, Sri Ramachandra Institute of Higher Education and Research, Porur, Chennai, India 600116.
**Corresponding author E-mail: santhisilambanan@sriramachandra.edu.in*

Received: January 14, 2026, Accepted: February 28, 2026, Published: March 2, 2026

Abstract

Purpose: The gastrointestinal tract in humans is composed of various microorganisms including bacteria, viruses, archaea, protozoa, and fungi. The interactions between gut and the microorganisms are crucial for the processes such as digestion, nutrient absorption, immune function, and metabolic regulation. Altered gut microbiota called dysbiosis, has been linked to the development and progression of various non-communicable diseases. This review has been aimed to address the alterations of gut microbiota in various non-communicable diseases.

Methods: A thorough literature search was conducted in PubMed, Embase, Web of Science, Scopus, and Google Scholar between January 2015 and December 2024 to identify studies examining the impact of gut microbiota in metabolic diseases. The search utilized keywords such as gut microbiota, gut dysbiosis, and altered microbiota in non-communicable diseases. The studies included were full research articles and studies published in English. In this comprehensive review, we explored the complex interplay between the gut microbiota and human health, focusing on its role in common metabolic disorders such as obesity, metabolic dysfunction-associated fatty liver disease, type 2 diabetes mellitus, cardiovascular diseases, autoimmune diseases, mental disorders, and cancer. Studies involving animal models, and cell lines and reviews were excluded.

Conclusions: The altered gut microbiota is involved in the initiation and progression of various non-communicable diseases. The dysbiosis not only aggravates the disease, but also causes refractoriness to the management of the disease. Hence, it is better to identify the causes of dysbiosis to control the harmful bacteria and to bring relief to the individuals with common con-communicable diseases.

Keywords: *Non-Communicable Diseases; Gut Microbiota; Gut Dysbiosis; Type 2 Diabetes Mellitus; Obesity.*

1. Introduction

The gastrointestinal tract (GIT) comprises a diverse array of microorganisms, serving as a crucial interface for the acquisition of energy. The organisms of the GIT possess the ability to transform dietary nutrients into different bioactive compounds, establishing a connection between the host and gut microbiota. The host factors that influence the composition of the microbiota include diet, genetic makeup, and immune response to environmental stimuli.

In India, non-communicable diseases (NCDs) account for 60% of deaths from various causes. Common NCD include cardiovascular disease (CVD), cerebrovascular disease, systemic hypertension, type 2 diabetes mellitus (T2DM), obesity, chronic respiratory disease, and cancers [1].

Knowledge gap: The metabolic and immune capabilities of the gut microbiota are pivotal in determining its impact on host health and susceptibility to diseases. Therefore, directing attention towards the gut microbiome and the associated metabolic pathways appears as a promising approach for developing effective therapeutic interventions for a range of diseases.

This review article aimed to analyze the involvement of the gut microbiome and its metabolites in the development of common NCDs such as obesity, T2DM, hypertension, CVD, metabolic dysfunction-associated steatotic liver disease (MASLD), autoimmune disorders, and cancers.

2. Methods

A thorough literature search was conducted in PubMed, Embase, Web of Science, Scopus, and Google Scholar between January 2015 and December 2024 to identify studies examining the impact of gut microbiota in metabolic diseases. The search utilized keywords such as gut microbiota, gut dysbiosis, and altered microbiota in non-communicable diseases. The studies included were full research articles and studies published in English.

In this comprehensive review, we explored the complex interplay between the gut microbiota and human health, focusing on its role in common metabolic disorders such as obesity, metabolic dysfunction-associated fatty liver disease, type 2 diabetes mellitus, cardiovascular diseases, autoimmune diseases, mental disorders, and cancer. Also, included studies with potential therapeutic avenues targeting the microbiome for disease management and prevention. Studies involving animal models, and cell lines and reviews were excluded. Additionally, we examined the factors influencing the gut microbiota composition, including diet, antibiotics, physical activity, stress, aging, and gastrointestinal surgeries.

Since this was a review article, ethics approval and informed consent were not required and hence, not obtained.

3. Discussion

The gut microbiota exhibits significant variations in composition and diversity across different regions of GIT, reflecting the distinct physiological and anatomical characteristics of each segment. From the oral cavity to the colon, the microbial populations within each region play specialized roles in host physiology, influencing processes such as digestion, metabolism, and immune responses [2].

The diverse groups of microorganisms within GIT, known as the human gut microbiota, are essential for preserving host health in the body. Gaining insights into the composition of the gut microbiota of GIT is essential for elucidating its impact on human health and the development of diseases, through mechanisms involving inflammation, immune dysregulation, metabolic alterations, and microbial metabolite production [2].

3.1. Gut Microbiota composition

The human GIT hosts a diverse and abundant microbial community, surpassing 100 trillion microorganisms, which is tenfold as many as the number of human cells. The microbiome comprises over three million genes, giving rise to thousands of metabolites and constituting over 100 times the genomic content found in the human genome, which includes approximately 23,000 genes [3].

The gut flora consists of various microorganisms, ranging from bacteria, viruses, fungi, protozoa, to archaea, engaging in intricate interactions with the host. These microorganisms play crucial roles in the digestion and absorption of nutrients and also exhibit an immune response [4].

Bacteria are the predominant microorganisms within the human gut microbiota, constituting an estimated 99% of the total microbiota [5]. The four primary phyla of bacteria in the human gut are Firmicutes, Bacteroidetes, Actinobacteria, and Proteobacteria. Firmicutes, classified as gram-positive bacteria, make up about 64% of the total colonic microbiota. This phylum encompasses over 200 distinct genera, including Lactobacillus, Clostridium, Bacillus, Ruminicoccus, and Enterococcus [6]. Bacteroidetes, characterized as gram-negative bacteria, contribute to approximately 23% of the total microbiota. Proteobacteria and Actinobacteria account for roughly 8% and 3% of the total microbiota, respectively [7]

3.2. Strain specificity

Bacterial strain specificity is important because different strains within the same species have different effects on health and disease. These functional differences influence the effectiveness of microbiome-based therapies. A well-known example is Escherichia coli. Some strains, such as enteropathogenic E. coli and enterohemorrhagic E. coli, are harmful and cause severe gastrointestinal disease, while other strains are harmless and form part of the normal gut microbiota [8].

3.3. GIT regional composition of gut microbiota

The gut microbiota composition undergoes variations throughout GIT, displaying marked distinctions in microbial populations within different regions [9] (Figure 1). GIT constitutes a sophisticated system, with each segment fulfilling specialized functions. They possess distinct epithelial structures and cell types that empower them to execute specific roles in the digestion, metabolism, immune responses, and endocrinological functions [10].

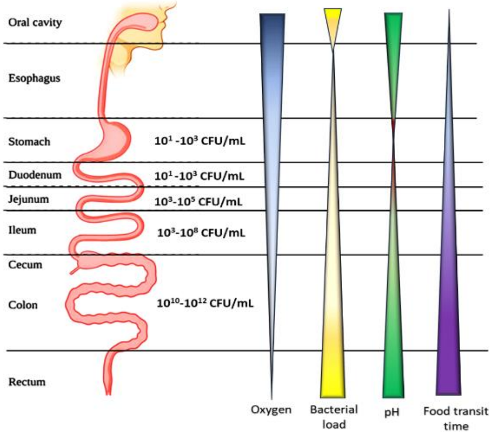

Fig. 1: Composition of Microbiota in Various Regions of Gastrointestinal Tract.

There is a complex relationship between the oral and gut microbiomes, with mutual colonization and pathogenic effects potentially leading to systemic diseases. The oral cavity hosts a diverse microbiome, encompassing more than 700 bacterial species. The balance within the oral microbiome ecosystem is crucial for sustaining oral health [11].

The composition of the oesophageal microbiota closely resembles that of the oral microbiota, featuring key phyla such as Firmicutes, Bacteroidetes, Proteobacteria, Actinobacteria, Saccharibacteria, and Fusobacteria, along with genera like Streptococcus, Prevotella, and Veillonella [12].

The stomach is a crucial organ in the GIT, with key roles in digestion and defence. The low bacterial load in the stomach is influenced by a variety of factors, including the gastric microbicidal barrier, low pH of 1.5 - 2.5, and other innate defence mechanisms against ingested pathogens, significantly limiting bacterial colonization. The stomach generally maintains a relatively low overall bacterial count of (10^1-10^3 CFU/mL) with diverse phyla, including Prevotella, Streptococcus, Rothia, and Veillonella [13].

The unique environment of small intestine, characterized by rapid motility, presence of antimicrobial peptides, and proximity to ingested nutrients, creates a rich ecosystem for microbe-microbe and host-microbe interactions [14]. The duodenum has a low bacterial load, ranging from 10^1-10^3 CFU/mL. The predominant bacterial phyla in the duodenum are Firmicutes, Proteobacteria, and Actinobacteria [15]. The jejunum exhibits several functional resemblances to the duodenum. In healthy individuals, the jejunum bacterial count is approximately 10^3-10^5 CFU/mL [9], [16]. The ileum possesses distinct characteristics in comparison to the jejunum and duodenum. When the digest reaches the ileum, it has exhausted almost all its nutrient contents. The main absorption functions of the ileum include the absorption of vitamin B12 and bile acids. This reduced nutrient absorption in the ileum leads to a relaxation of immune mechanisms that typically limit microbial diversity in the proximal small intestine. As a result, there is a notable increase in microbial quantity in the ileum compared to the jejunum, with estimates ranging from 10^3 to10^8 CFU/mL [9], [17].

The colon has the most diverse array of microbes within the human body, surpassing the combined microbial count of all other bodily regions and boasting a bacterial density ranging 10^{10} - 10^{12} CFU/mL [13]. Its highly anaerobic environment, slow motility with transit times of up to 30 hours, and the presence of indigestible complex polysaccharides and dietary fibre, along with traces of nutrients and residual bile acids that are not absorbed in the ileum, contribute to the flourishing microbial population [18]. A symbiotic companion has evolved over in the colon with adaptive strategies for immune evasion and modulation. This enhances the survival of the microbes in the GIT despite the defence mechanisms in the host.

A vital mutualistic role of the gut microbiome revolves around the microbial production of short-chain fatty acids (SCFAs) through the anaerobic fermentation of pectin, cellulose, high-amylose starch, inulin, or mucosal glycans by Firmicutes and Bacteroides phyla. The released SCFAs include acetate, propionate, and butyrate [19]. SCFAs are essential for maintaining host well-being, providing approximately 5 - 15% of the total caloric needs for humans. Additionally, they are involved in the production of vitamins such as vitamins K and B12 [19]. SCFAs participate in diverse metabolic pathways in both the host and microbial systems, with butyrate as the primary energy source for colonocytes. Colonocytes metabolize butyrate into CO_2, reducing oxygen levels in the colon and promoting an anaerobic state. This anaerobic setting is vital for maintaining immune balance, resisting pathogens, and facilitating the proliferation of anaerobic microbial populations that produce butyrate, thus establishing a positive feedback loop [19].

4. Factors Affecting Gut Microbiota Composition

Gut dysbiosis is characterized by disruption in the balance of the composition and hence the physiological functions of the gut microbes. This leads to alterations in the microbial diversity, increased abundance of pathogenic species, and changes in the metabolic functions of the microbiota, which can cause harmful effects on the host [20].

During gut dysbiosis, the intestinal epithelial barrier gets compromised, leading to increased permeability of the intestinal wall, also known as leaky gut. The integrity of the intestine can be compromised due to various mechanisms. The reduction of beneficial gram-positive bacteria, such as Firmicutes, leads to the inappropriate production of SCFAs [20]. SCFAs maintain the integrity of the intestine by increasing the production of mucus as well as increasing the expression of tight junction proteins [21].

Dysbiosis can also lead to the production of microbial metabolites, such as phenols and amines, which may be toxic to the intestinal epithelium and disrupt the integrity of the intestinal barrier [21]. Factors contributing to gut dysbiosis include antibiotic use, a high-fat diet, a low-fibre diet, stress, aging, physical activity, and gastrointestinal surgeries [21]. (Figure 2) The disruption of the gut barrier, increased production of endotoxins, and metabolic alterations are key mechanisms through which dysbiosis may contribute to the development of many non-communicable diseases like T2DM, obesity, cancer, autoimmune disease, major depressive disorder (MDD), CVD, and MAFLD.

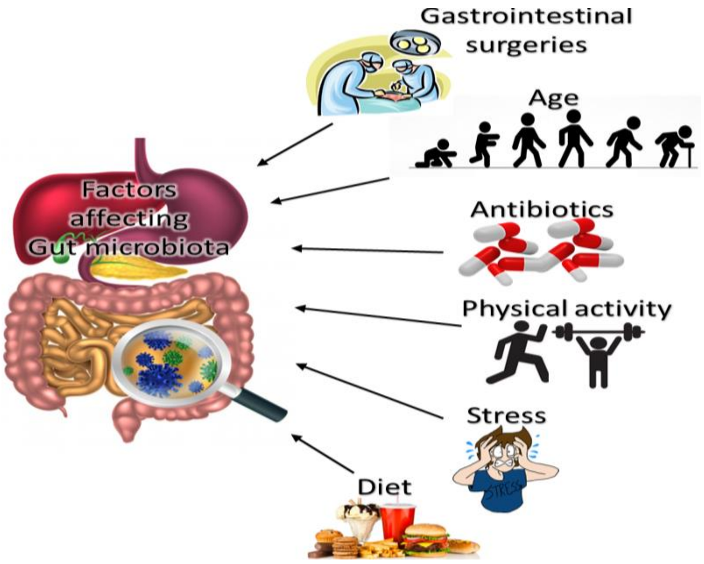

Fig. 2: Factors Affecting the Composition of Gut Microbiota.

4.1. Diet

Diet has a significant role in shaping the composition of the gut microbiome, and various types of diets can lead to gut dysbiosis. A high-calorie western-style diet is associated with the induction of gut dysbiosis, leaky gut, and inflammation, which can result in the translocation of gut-derived bacterial content causing hepatic inflammation [22]. Additionally, high-fructose diets can downregulate tight junction proteins, promote gut barrier deterioration and increase the leakage of gut-derived metabolites and bacterial products into the portal circulation [22].

Furthermore, high-fat diets can reduce the beneficial microbes such as Akkermansia muciniphila and Lactobacillus spp, which are associated with healthy metabolic states. Similarly, high-protein diets have been linked to reduced levels of butyrate-producing bacteria. Moreover, gluten-free diets have been associated with microbiota dysbiosis, potentially due to the reduced consumption of whole grains [23]. Increased adherence to the Mediterranean Diet is positively associated with the abundance of SCFAs producers in the gut microbiome [24].

4.2. Use of antibiotics

Exposure to high-level broad-spectrum antibiotics can lead to a reduction in microbial diversity, changes in the abundance of specific taxa, and alterations in gene expression, protein activity, and metabolome [25]. Antibiotics can also damage the three intestinal barriers: the epithelial cell layer, the mucus layer, and the immunological barrier, which can weaken the gut's ability to maintain a healthy microbial balance. Antibiotics can disrupt the natural defence mechanisms provided by the gut microbiota against opportunistic pathogens. This can lead to secondary infections and further dysbiosis [25].

4.3. Physical activity

Intense physical activity alters gut blood flow and oxygen levels, promoting the growth of oxygen-sensitive bacteria. This increases intestinal permeability, leading to a leaky gut. High-intensity exercise modulates the immune system, predisposing to inflammation. Athletes in intense training, may have specific dietary habits, affecting nutrient availability for different bacteria and potentially influencing the gut microbiota [26].

4.4. Stress

Stress through the hypothalamic-pituitary-adrenal axis stimulates the release of cortisol. Cortisol directly influences the gut environment and alters the microbial composition through its immunomodulatory and anti-inflammatory effects [27]. Stress can also activate the sympathetic and parasympathetic branches of the autonomic nervous system, changing gut motility, blood flow, and secretion, which impacts the local environment for gut microbes [28]. Stress hormones can influence microbial metabolism and nutrient utilization, selectively favoring the growth of certain bacteria and contributing to shifts in the gut microbiota composition. It can also induce epigenetic modifications that impact the gene expression involved in immune function and microbial-host interactions [29].

4.5. Age

The human gut microbiota undergoes significant developmental changes from infancy to old age, influenced by factors such as mode of delivery of the child, genetics, environment, diet, lifestyle, and use of antibiotics [30]. Specifically, the Firmicutes/Bacteroidetes ratio is altered through different ages of life from birth to adulthood [31]. By the age of three, the gut microbiota of a child closely resembles that of adults, dominated by Firmicutes, Bacteroidetes, and Actinobacteria. In individuals over the age of 70 years, changes in gut microbes are influenced by stress, digestion and absorption of nutrients, and weakened immune activity [2]. In the elderly, higher levels of Firmicutes, Proteobacteria, and Actinobacteria have been linked with inflammation and metabolic disorders, and intestinal inflammation and permeability [32].

4.6. Gastrointestinal surgeries

Gastric bypass surgery and colectomy lead to an increase in Bacteroidetes and a decrease in Firmicutes [33]. Altered ratios of clostridial clusters have been observed in colon cancer patients, potentially contributing to gut dysbiosis [34]. Ileostomy and colostomy procedures are associated with an abundance of Proteobacteria [35]. The removal of the vermiform appendix through appendectomy is linked to dysbiosis-induced diseases [36].

5. Gut Microbiota and Metabolic Diseases

Recently, research focus has increasingly shifted to the human microbiome as a major factor influencing health and disease. Early studies showed significant variation in gut microbiota between individuals and identified links with many conditions, including obesity, T2DM, CVD, inflammatory bowel disease, allergies, neurodegenerative disorders, and cancer. However, much of the research has been based on associations rather than clear causal relationships.

Although animal studies have provided useful mechanistic insights, their translation to human health has been limited. Similarly, microbiome-based interventions such as faecal microbiota transplantation (FMT), probiotics, prebiotics, and postbiotics have shown variable or modest benefits in clinical trials. These challenges highlight the need for more precise and functional analysis of the microbiome using multi-omics and artificial intelligence. This approach supports a shift toward systems biology and personalized medicine, enabling better understanding and targeted treatment of diseases [37].

5.1. Gut microbiota and type 2 diabetes mellitus (T2DM)

In T2DM, gut microbiota is characterized by a decrease in gram-positive bacteria such as Firmicutes, whereas there is an increase in gram-negative bacteria such as Bacteroidetes and Proteobacteria. This leads to increased intestinal inflammation and permeability, thereby contributing to insulin resistance (IR) and the further complications of T2DM [38]. Lipopolysaccharide (LPS) is an endotoxin shed from Gram-negative bacteria in the gut and excreted in the faeces. However, in gut dysbiosis, there is an overgrowth of gram-negative bacteria causing increased shedding of LPS [38].

LPS induces inflammation in the intestinal mucosa upon binding to LPS-binding protein and the cluster of differentiation 14 present on monocytes and macrophages. This further activates the Toll-like receptor 4 and nuclear factor-kappa B, leading to the release of pro-inflammatory cytokines [39]. LPS can also directly damage the intestine by downregulating the expression of claudin, occludin, and other tight junction proteins [40]. This leads to increased intestinal permeability of LPS into the bloodstream, leading to endotoxemia. In the bloodstream, LPS triggers an immune response, which can lead to IR and the development of T2DM [39]. Also, there is increased production of free radicals; this, along with activation of the c-Jun N-terminal kinase pathway, impairs insulin signalling [41].

Fructoselysine is an Amadori product formed from lysine and glucose that is one of the primary dietary advanced glycated end products (AGEs). Studies have shown that E. coli has the capacity to breakdown fructoselysine. Also, Intestinimonas spp can convert to butyrate. N-ε-carboxymethyllysine, another AGE formed in T2DM is completely utilized by Cloacibacillus and Oscillibacter spp [42].

5.2. Gut microbiota and obesity

Obesity affects the proportion of the gut microbiome. The Firmicutes/Bacteroidetes ratio can predict whether the individual is likely to develop obesity. Obese microbiota has a significantly higher ratio and the proportion of Firmicutes decreases as weight is lost [43]. Lactobacillus acidophilus regulates body weight, adiposity, inflammation, and IR in animal models by modulating the AMPK-SREBP-1c/PPARα pathway and activating brown adipose tissue and enhancing energy, glucose, and lipid metabolism [44].

5.3. Gut microbiota and cardiovascular disease (CVD)

The effects of dietary fat on gut microbial populations depend not only on the quantity of fat consumed but also on its composition, particularly the degree of saturation and the chain length of fatty acids. Saturated long-chain fatty acids have been reported to enhance the production of trimethylamine (TMA), which is subsequently converted into trimethylamine N-oxide (TMAO), a gut microbiota-derived biomarker linked to increased cardiovascular risk. This process is mediated through mechanisms such as elevated colonic oxygen levels and increased taurine-conjugated bile acids, which create a favorable environment for TMA-producing bacteria [45].

SCFAs and omega-3 polyunsaturated fatty acids exert protective effects by reducing intestinal pH and promoting the growth of beneficial SCFA-producing bacteria, while simultaneously suppressing TMA-producing microbial populations. Therefore, dietary strategies that focus on optimizing fatty acid composition may help modulate gut microbiota and their metabolites, offering a promising approach for the prevention and management of CVD [45]. Gut dysbiosis influences host blood pressure, potentially contributing to the development and progression of hypertension [46]. In hypertensive individuals, increased levels of TMAO predict adverse cardiovascular outcomes.

Reduced dietary intake of fibre leads to a decreased formation of butyrate. Lower levels of butyrate may promote local inflammation, worsen dysbiosis, and contribute to impaired gut barrier function, leading to the leakage of LPS, which can further fuel local and systemic inflammation. Decreased butyrate-producing microbes have been linked to CVD and heart failure [45]. Hence, dietary fibre enrichment, prebiotics, probiotics, and TMAO-lowering agents can act as adjunctive strategies in hypertension management [47].

5.4. Gut microbiota and lung diseases

Lung diseases such as idiopathic pulmonary fibrosis, chronic obstructive pulmonary disease, bronchial asthma, lung cancer, pulmonary arterial hypertension, and cystic fibrosis show altered gut microbiota. SCFAs and deaminotyrosine enter the lung from the gut and influence lung immune function. Also, immune cells and immune factors that enter the respiratory system from the intestine aggravate the inflammatory process [48].

5.5. Gut microbiota and metabolic dysfunction-associated steatotic liver disease (MASLD)

The gut-liver axis involves communication between the gut and liver through the intestinal barrier, gut microbiota, portal circulation, bile acids, and immune system [49]. Disruptions in this axis due to poor diet, alcohol intake, or metabolic disorders can cause intestinal dysbiosis and increased gut permeability. This allows harmful microbial products to reach the liver through the portal circulation, leading to liver inflammation and injury. Such changes are seen in MASLD, alcoholic liver disease, autoimmune liver diseases, and cirrhosis [50]. These disturbances also contribute to disease progression, complications, and poor treatment outcomes [49].

Restoring gut microbial balance, strengthening the intestinal barrier, and improving host–microbe interactions may help prevent or slow the progression of liver disease [49]. Lipid peroxidation, pro-inflammatory factors, cytotoxic effects of secondary bile acids, and IR, along with dysbiosis of intestinal microbiota are considered as the pathogenic mechanisms of MASLD [51]. Gut microbiota can metabolize and produce trimethylamine (TMA), which is further oxidized to form trimethylamine-N-oxide (TMAO) in the liver. High levels of TMAO have been linked with non-alcoholic steatohepatitis, contributing to MASLD [52].

5.6. Gut microbiota and cancer

The microbiota promotes the development of cancer through their influence on inflammation and also by causing genomic stability of host cells through the dysregulation of various signalling pathways. Helicobacter pylori (H. pylori) have been designated as a carcinogenic substance, emphasizing its role in gastric cancer. In H. pylori infected patients, there is altered gastric microbiota [53]. Microbial pathogens are recognized as drivers of approximately 20% of tumorigenesis, and a significant number of malignancies are associated with dysbiosis. During dysbiosis, there is release of toxins that induce DNA breaks, causing genomic instability, and progression to cancer in predisposed cells [54].

5.7. Gut microbiota and autoimmune diseases

Gut dysbiosis is associated with several autoimmune diseases, including rheumatoid arthritis, systemic lupus erythematosus, and Sjögren's syndrome. The intricate interplay between genetic predisposition, environmental factors, and microbial influences underscores the complexity of the balance between gut microorganisms and autoimmune diseases [55]. Environmental and genetic factors influence the immune system, leading to the abnormal generation of B cells producing autoantibodies, T cells with self-reactivity, and the abnormal production of proinflammatory cytokines [55].

On a molecular level, the gut microbiota mediates autoimmunity through processes involving the posttranslational modification and cross-reactivity of autoantigens. Due to impaired gut integrity, bacteria enter the liver, initiating direct interactions with immune cells and tissue cells. This process ultimately initiates systemic autoimmunity [55]. The gut microbiota influences the differentiation and function of Treg cells, with certain microbial components contributing to the induction and activation of Treg cells. This promotes an anti-inflammatory environment, suppressing excessive immune responses that could otherwise lead to autoimmunity [55].

5.8. Gut microbiota and mental disorders

The gut microbiota plays a significant role in influencing mental disorders, including anxiety, depression, and neurodevelopmental disorders, through the bidirectional communication established by the gut-brain axis. This link between the brain and the gut involves immunological, neuronal, and neuroendocrine pathways [56]. The microbiota generates various metabolites, which have the potential to influence the central nervous system by exerting effects on the nervous system of the intestine. Disruptions in any of these mechanisms, whether direct or indirect, can have implications for mental health [57].

Certain bacteria can produce neuroactive compounds, including neurotransmitters such as serotonin, gamma-aminobutyric acid, and dopamine. These compounds play a crucial role in regulating mood and emotional well-being. Dysbiosis is associated with inflammatory processes that can impact neural signalling, contributing to the onset of mental health disorders [56]. In a comprehensive meta-analysis on the gut microbiome in adults with major psychiatric disorders, it is found that there is a decrease in bacterial genera that produce SCFAs. Simultaneously, an increase in the bacterial genera, which is associated with pro-inflammatory responses in individuals diagnosed with conditions such as psychosis, schizophrenia, MDD, bipolar disorder, and anxiety [57].

6. Gut Microbiota Targeted Therapies

6.1. Probiotics

Probiotics and prebiotics have gained substantial attention in therapeutic interventions, showcasing their potential to impact various aspects of health positively [58]. The term probiotic, derived from the Greek meaning "for life," refers to beneficial microorganisms that support health. The modern definition, given by the Food and Agriculture Organization and World Health Organization in 2001, describes probiotics as live microorganisms that, when administered in adequate amounts, provide health benefits to the host [59].

Common probiotics include Lactobacillus, Bifidobacterium, Enterococcus, Lactococcus, and Streptococcus. To be useful and safe, probiotics should be genetically stable, able to survive stomach acid and bile, attach to the intestinal lining, and be non-pathogenic. They should also produce lactic acid, tolerate processing conditions, and multiply quickly. Probiotics strengthen the intestinal barrier, help beneficial bacteria attach to the gut, and prevent harmful microbes from attaching. They also support the immune system and produce antimicrobial substances that inhibit pathogens. These antimicrobial substances are called bacteriocins, which are protein-based compounds. Most Lactobacillus and Bifidobacterium strains produce bacteriocins, helping protect gut health [60].

6.2 Prebiotics

Prebiotics are nondigestible dietary components that promote health by selectively stimulating the growth and activity of beneficial gut microorganisms, as defined by the International Scientific Association for Probiotics and Prebiotics (ISAPP). Most prebiotics are carbohydrates, such as inulin, fructo-oligosaccharides, galacto-oligosaccharides, and lactulose. They are naturally found in foods like whole grains, onions, garlic, and bananas. Since they are not digested by human enzymes, prebiotics reach the colon where they are fermented by beneficial bacteria, producing SCFAs. These metabolites help maintain gut microbiota balance, strengthen the intestinal barrier, regulate immune function, and support overall gut health [59].

6.3 Postbiotics

Postbiotics, as defined by ISAPP in 2021, are preparations of inactivated microorganisms and/or their components that provide health benefits to the host. They include inactivated microbial cells, cell components, proteins, peptides, SCFAs, vitamins, bacteriocins, and other bioactive metabolites. Their composition depends on the probiotic strain, growth conditions, and fermentation substrates. Postbiotics exert beneficial effects by modulating gut microbiota, inhibiting harmful bacteria, strengthening the intestinal barrier, and providing anti-inflammatory and antioxidant effects. They also influence immune responses and host metabolism through signaling mechanisms. Since postbiotics are non-living and do not replicate, they are considered safer than probiotics, especially for immunocompromised or critically ill individuals [59].

Prebiotics help regulate gut microbiota and maintain intestinal integrity. Probiotics have shown benefits in managing gastrointestinal disorders and also demonstrate potential in reducing hypercholesterolemia and hypertriglyceridemia, and supporting the management of T2DM. Synbiotics, a combination of prebiotics and probiotics, exert synergistic effects that enhance gut health and immune function. Postbiotics act as bioactive compounds with antibacterial and anti-inflammatory properties [61].

6.4 Faecal microbiota transplantation (FMT)

FMT can help restore disrupted gut microbiota balance. While these interventions have been widely studied in gastrointestinal disorders, future research should also explore their role in metabolic conditions such as CVD and hyperuricemia [61]. According to He et al., the safety and potential efficacy of selected donor FMT to enhance first-line immune checkpoint inhibitor-based treatment in metastatic renal cell carcinoma deserves further investigations [51].

7. Advances in Microbiome Research

Multi-omics technologies, including genomics, transcriptomics, proteomics, and metabolomics, have advanced microbiome research by providing a comprehensive understanding of microbial functions and host–microbiome interactions. These approaches support personalized medicine by enabling more accurate diagnosis and targeted therapies [62].

Machine learning (ML) and deep learning (DL) can analyze complex microbiome and metabolome data to predict disease risk and identify microbial biomarkers in conditions such as T2DM and inflammatory bowel disease. These artificial intelligence (AI)-based tools help uncover links between gut microbiota, microbial metabolites, and disease mechanisms. Overall, multi-omics and AI have shifted microbiome research from simply identifying microbes to understanding their functional roles, creating new opportunities for precision diagnostics and microbiome-based therapies [62].

8. Limitations

It is often difficult to determine whether changes in the gut microbiome cause disease or are simply a result of the disease. Factors such as diet, medications, and inflammation can alter the microbiome, making interpretation challenging. There is no single healthy microbiome, and since each person's microbiome is unique, universal probiotic or dietary treatments may not be effective. The microbiome can also change quickly due to short-term influences like infections or diet, which may affect study results.

Most research uses faecal samples, which do not fully represent microbes in other parts of the gut. In addition, many gut bacteria cannot be cultured, and technical factors like contamination may affect accuracy. Differences between individuals, limitations of animal studies, and the influence of medications further complicate research. The long-term safety and effects of microbiome-based therapies are still unclear. Therefore, more standardized, long-term, and multi-omics studies are needed to better understand the microbiome and develop reliable, personalized treatments.

9. Conclusion

In conclusion, gut dysbiosis is found to promote the development and progression of various metabolic diseases. The altered gut microbiome influences metabolic processes, immune responses, and inflammation, thereby contributing significantly to disease pathogenesis. The intricate mechanisms involved in the interactions between the gut microbiota and humans require additional exploration. Furthermore, it is essential to employ new technologies to examine and control the microbiota with precision for targeted interventions.

10. Future Directions

Gut microbiome research is shifting from simply identifying microbes to developing personalized treatments that improve health. Key areas include using multi-omics and artificial intelligence to better understand microbial functions and predict disease risk. This will help create precision therapies such as targeted probiotics, prebiotics, postbiotics, and bacteriophage treatments.

Research is also focusing on tailoring interventions based on an individual's microbiome to manage chronic diseases. Scientists are studying not only bacteria but also other microbes such as fungi, viruses, and archaea to understand their roles in health and disease.

New technologies, including metabolomics, machine learning, and gut-on-a-chip models, are helping to reveal how the microbiome interacts with the brain, immune system, and metabolism. These advances aim to move microbiome research toward precise, personalized therapies for improving overall health and treating complex diseases.

11. Declarations

Ethics Approval

Since this is a review article, ethics approval was not obtained. The study did not require written informed consent.

Consent for Publication

NA

Acknowledgements

NA

Availability of Data and Materials

The manuscript incorporates all datasets produced or examined throughout this research study. The information related to the manuscript can be obtained from the corresponding author on reasonable request within a period of five years from the time of publication of this manuscript.

Conflict of Interest

The authors declare that there were no conflicts of interest.

Funding Statement

The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

Authors' Contributions

All the authors have contributed equally to writing this manuscript.

References

[1] Burden of NCDs and their risk factors in India. Available at-http://www.searo.who.int/india/topics/noncommunicable_diseases/ncd_situation_global_report_ncds_2014.pdf.

[2] Rinninella, E., Raoul, P., Cintoni, M., Franceschi, F., Miggiano, G. A. D., Gasbarrini, A., & Mele, M. C. (2019). What is the healthy gut microbiota composition? A changing ecosystem across age, environment, diet, and diseases. Microorganisms, 7(1), 14. https://doi.org/10.3390/microorganisms7010014.

[3] Ma, Z. F., & Lee, Y. Y. (2025). The Role of the Gut Microbiota in Health, Diet, and Disease with a Focus on Obesity. Foods, 14(3), 492. https://doi.org/10.3390/foods14030492.

[4] Zhao, M. A., Chu, J., Feng, S., Guo, C., Xue, B., He, K., & Li, L. (2023). Immunological mechanisms of inflammatory diseases caused by gut microbiota dysbiosis: A review. Biomedicine & Pharmacotherapy, 164, 114985. https://doi.org/10.1016/j.biopha.2023.114985.

[5] Hu, C., & Shen, H. (2024). Microbes in Health and Disease: Human Gut Microbiota. Applied Sciences, 14(23), 11354. https://doi.org/10.3390/app142311354.

[6] Plamada, D., & Vodnar, D. C. (2021). Polyphenols—Gut microbiota interrelationship: A transition to a new generation of prebiotics. Nutrients, 14(1), 137. https://doi.org/10.3390/nu14010137.

[7] Yanni, A. E., Mitropoulou, G., Prapa, I., Agrogiannis, G., Kostomitsopoulos, N., Bezirtzoglou, E., et al. (2020). Functional modulation of gut microbiota in diabetic rats following dietary intervention with pistachio nuts (Pistacia vera L.). Metabolism Open, 7, 100040. https://doi.org/10.1016/j.metop.2020.100040.

[8] Van Hul, M., Cani, P. D., Petitfils, C., De Vos, W. M., Tilg, H., & El-Omar, E. M. (2024). What defines a healthy gut microbiome? Gut, 73(11), 1893-1908. https://doi.org/10.1136/gutjnl-2024-333378.

[9] Rehner, J., Molano, L. A. G., Christodoulou, C., Holländer, S., Förster, M. O., Keller, V., et al. (2025). Examining spatial microbiome variations across gastrointestinal tract regions in obesity. Scientific Reports, 15(1), 25423. https://doi.org/10.1038/s41598-025-10931-0.

[10] Ogobuiro, I., Gonzales, J., Shumway, K. R., Tuma, F. Physiology, Gastrointestinal. 2023 Apr 8. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 30725788.

[11] Rajasekaran, J. J., Krishnamurthy, H. K., Bosco, J., Jayaraman, V., Krishna, K., Wang, T., & Bei, K. (2024). Oral microbiome: a review of its impact on oral and systemic health. Microorganisms, 12(9), 1797. https://doi.org/10.3390/microorganisms12091797.

[12] Barchi, A., Massimino, L., Mandarino, F. V., Vespa, E., Sinagra, E., Almolla, O., et al. (2024). Microbiota profiling in esophageal diseases: Novel insights into molecular staining and clinical outcomes. Computational and structural biotechnology journal, 23, 626-637. https://doi.org/10.1016/j.csbj.2023.12.026.

[13] Kennedy, M. S., & Chang, E. B. (2020). The microbiome: Composition and locations. Progress in Molecular Biology and Translational Science, 176, 1–42. https://doi.org/10.1016/bs.pmbts.2020.08.013.

[14] Kastl, A. J., Jr., Terry, N. A., Wu, G. D., & Albenberg, L. G. (2020). The structure and function of the human small intestinal microbiota: Current understanding and future directions. Cellular and Molecular Gastroenterology and Hepatology, 9(1), 33–45. https://doi.org/10.1016/j.jcmgh.2019.07.006.

[15] Chavoya-Guardado, M. A., Vasquez-Garibay, E. M., Ruiz-Quezada, S. L., Ramírez-Cordero, M. I., Larrosa-Haro, A., Castro-Albarran, J. (2022). Firmicutes, Bacteroidetes and Actinobacteria in Human Milk and Maternal Adiposity. Nutrients, 14(14), 2887. https://doi.org/10.3390/nu14142887.

[16] Jensen, B. A. H., Heyndrickx, M., Jonkers, D., Mackie, A., Millet, S., Naghibi, M., et al. (2023). Small intestine vs. colon ecology and physiology: Why it matters in probiotic administration. Cell Reports Medicine, 4(9). https://doi.org/10.1016/j.xcrm.2023.101190.

[17] Sasso, J. M., Ammar, R. M., Tenchov, R., Lemmel, S., Kelber, O., Grieswelle, M., & Zhou, Q. A. (2023). Gut microbiome–brain alliance: a landscape view into mental and gastrointestinal health and disorders. ACS Chemical Neuroscience, 14(10), 1717-1763. https://doi.org/10.1021/acschemneuro.3c00127.

[18] Shalon, D., Culver, R. N., Grembi, J. A., Folz, J., Treit, P. V., Shi, H., et al. (2023). Profiling the human intestinal environment under physiological conditions. Nature, 617(7961), 581–591. https://doi.org/10.1038/s41586-023-05989-7.

[19] Fusco, W., Lorenzo, M. B., Cintoni, M., Porcari, S., Rinninella, E., Kaitsas, F. et al. (2023). Short-chain fatty-acid-producing bacteria: key components of the human gut microbiota. Nutrients, 15(9), 2211. https://doi.org/10.3390/nu15092211.

[20] Shen, Y., Fan, N., Ma, S. X., Cheng, X., Yang, X., & Wang, G. (2025). Gut microbiota dysbiosis: pathogenesis, diseases, prevention, and therapy. MedComm, 6(5), e70168. https://doi.org/10.1002/mco2.70168.

[21] Pérez-Reytor, D., Puebla, C., Karahanian, E., & García, K. (2021). Use of short-chain fatty acids for the recovery of the intestinal epithelial barrier affected by bacterial toxins. Frontiers in physiology, 12, 650313. https://doi.org/10.3389/fphys.2021.650313.

[22] Kang, G. G., Trevaskis, N. L., Murphy, A. J., & Febbraio, M. A. (2022). Diet-induced gut dysbiosis and inflammation: Key drivers of obesity-driven NASH. iScience, 26(1), 105905. https://doi.org/10.1016/j.isci.2022.105905.

[23] Du, W., Zou, Z. P., Ye, B. C., & Zhou, Y. (2025). Gut microbiota and associated metabolites: key players in high-fat diet-induced chronic diseases. Gut Microbes, 17(1), 2494703. https://doi.org/10.1080/19490976.2025.2494703.

[24] Khavandegar, A., Heidarzadeh, A., Angoorani, P., Hasani-Ranjbar, S., Ejtahed, H. S., Larijani, B., & Qorbani, M. (2024). Adherence to the Mediterranean diet can beneficially affect the gut microbiota composition: a systematic review. BMC Medical Genomics, 17(1), 91. https://doi.org/10.1186/s12920-024-01861-3.

[25] Kesavelu, D., & Jog, P. (2023). Current understanding of antibiotic-associated dysbiosis and approaches for its management. Therapeutic advances in infectious disease, 10, 20499361231154443. https://doi.org/10.1177/20499361231154443.

[26] Hoseini, R., Rahim, H. A., Saifalddin, D. L., Kareem, D. A., & Fatah, A. M. (2025). Exercise intensity-mediated regulation of gut epithelial cells and immune function in gut microbiota dysbiosis. Journal of Translational Medicine, 24, 10. https://doi.org/10.1186/s12967-025-07492-1.

[27] Tofani, G. S., Leigh, S. J., Gheorghe, C. E., Bastiaanssen, T. F., Wilmes, L., Sen, P., et al. (2025). Gut microbiota regulates stress responsivity via the circadian system. Cell metabolism, 37(1), 138-153. https://doi.org/10.1016/j.cmet.2024.10.003.

[28] Rusch, J. A., Layden, B. T., & Dugas, L. R. (2023). Signalling cognition: the gut microbiota and hypothalamic-pituitary-adrenal axis. Frontiers in endocrinology, 14, 1130689. https://doi.org/10.3389/fendo.2023.1130689.

[29] Zhang, Q., Liu, Y., Li, Y., Bai, G., Pang, J., Wu, M., et al. (2025). Implications of gut microbiota-mediated epigenetic modifications in intestinal diseases. Gut Microbes, 17(1), 2508426. https://doi.org/10.1080/19490976.2025.2508426.

[30] Schoultz, I., Claesson, M. J., Dominguez-Bello, M. G., Fåk Hållenius, F., Konturek, P., Korpela, K., et al. (2025). Gut microbiota development across the lifespan: Disease links and health-promoting interventions. Journal of internal medicine, 297(6), 560-583. https://doi.org/10.1111/joim.20089.

[31] Mariat, D., Firmesse, O., Levenez, F., Guimarães, V., Sokol, H., Doré, J., et al. (2009). The Firmicutes/Bacteroidetes ratio of the human microbiota changes with age. BMC Microbiology, 9, 123. https://doi.org/10.1186/1471-2180-9-123.

[32] Gyriki, D., Nikolaidis, C. G., Bezirtzoglou, E., Voidarou, C., Stavropoulou, E., & Tsigalou, C. (2025). The gut microbiota and aging: interactions, implications, and interventions. Frontiers in aging, 6, 1452917. https://doi.org/10.3389/fragi.2025.1452917.

[33] Zheng, Z., Hu, Y., Tang, J., Xu, W., Zhu, W., & Zhang, W. (2023). The implication of gut microbiota in recovery from gastrointestinal surgery. Frontiers in cellular and infection microbiology, 13, 1110787. https://doi.org/10.3389/fcimb.2023.1110787.

[34] Suga, D., Mizutani, H., Fukui, S., Kobayashi, M., Shimada, Y., Nakazawa, Y., et al. (2022). The gut microbiota composition in patients with right- and left-sided colorectal cancer and after curative colectomy, as analyzed by 16S rRNA gene amplicon sequencing. BMC Gastroenterology, 22(1), 313. https://doi.org/10.1186/s12876-022-02382-y.

[35] Segal, J. P., Oke, S., Hold, G. L., Clark, S. K., Faiz, O. D., & Hart, A. L. (2018). Systematic review: Ileoanal pouch microbiota in health and disease. Alimentary Pharmacology & Therapeutics, 47(4), 466–477. https://doi.org/10.1111/apt.14454.

[36] Shi, F., Liu, G., Lin, Y., Guo, C. L., Han, J., Chu, E. S. H., et al. (2023). Altered gut microbiome composition by appendectomy contributes to colorectal cancer. Oncogene, 42(7), 530–540. https://doi.org/10.1038/s41388-022-02569-3.

[37] Van Hul, M., & Cani, P. D. (2026). From microbiome to metabolism: Bridging a two-decade translational gap. Cell metabolism, 38(1), 14-32. https://doi.org/10.1016/j.cmet.2025.10.011.

[38] Cunningham, A. L., Stephens, J. W., & Harris, D. A. (2021). Gut microbiota influence in type 2 diabetes mellitus (T2DM). Gut pathogens, 13(1), 50. https://doi.org/10.1186/s13099-021-00446-0.

[39] Wang, C., Ge, F., Ge, F., Xu, Z., & Jiang, J. (2025). Harnessing stem cell therapeutics in LPS-induced animal models: mechanisms, efficacies, and future directions. Stem Cell Research & Therapy, 16(1), 176. https://doi.org/10.1186/s13287-025-04290-w.

[40] Skinner, A. G., Malik, A., Siddiqui, M. R., Singh, V., & Akhtar, S. (2025). Inulin Protects Caco-2 Cells against Lipopolysaccharide-induced Epithelial Barrier Dysfunction. Food Science & Nutrition, 13(4), e70046. https://doi.org/10.1002/fsn3.70046.

[41] Yu, X., Li, M., & Li, H. (2025). The role of gut dysbiosis and mitochondrial dysfunction in type 2 diabetes: Insights on pathogenesis, intervention and future perspective. Biomedicine & Pharmacotherapy, 193, 118846. https://doi.org/10.1016/j.biopha.2025.118846.

[42] De Vos, W. M., Tilg, H., Van Hul, M., & Cani, P. D. (2022). Gut microbiome and health: mechanistic insights. Gut, 71(5), 1020-1032. https://doi.org/10.1136/gutjnl-2021-326789.

[43] Noor, J., Chaudhry, A., Batool, S., Noor, R., & Fatima, G. (2023). Exploring the impact of the gut microbiome on obesity and weight loss: a review article. Cureus, 15(6), e40948. https://doi.org/10.7759/cureus.40948.

[44] Zhang, K., Zhang, Q., Qiu, H., Ma, Y., Hou, N., Zhang, J., et al. (2024). The complex link between the gut microbiome and obesity-associated metabolic disorders: Mechanisms and therapeutic opportunities. Heliyon, 10(17). https://doi.org/10.1016/j.heliyon.2024.e37609.

[45] Kilic, E., & Basaran, P. (2025). Dietary Fatty Acids Modulate Gut Microbiota-Derived Trimethylamine-N-Oxide: Potential Mechanisms and Future Perspective. Nutrients, 17(23), 3787. https://doi.org/10.3390/nu17233787.

[46] Trøseid, M., Andersen, G. Ø., Broch, K., & Hov, J. R. (2020). The gut microbiome in coronary artery disease and heart failure: Current knowledge and future directions. EBioMedicine, 52, 102649. https://doi.org/10.1016/j.ebiom.2020.102649.

[47] Avram, A. C., Craciun, M. L., Pah, A. M., Buleu, F., Cotet, I. G., Mateescu, D. M., et al. (2025). Association Between Gut Microbiome Alterations and Hypertension-Related Cardiovascular Outcomes: A Systematic Review and Meta-Analysis. Microbiology Research, 16(11), 244. https://doi.org/10.3390/microbiolres16110244.

[48] Sun, M., Lu, F., Yu, D., Wang, Y., Chen, P., & Liu, S. (2014). Respiratory diseases and gut microbiota: relevance, pathogenesis, and treatment. Front Microbiol, 15, 1358597. https://doi.org/10.3389/fmicb.2024.1358597.

[49] Ramírez-Mejía, M. M., Ponciano-Rodríguez, G., & Méndez-Sánchez, N. (2025). Implications of the liver-gut axis in liver disease: from mechanisms to therapeutic targets. Archives of Medical Research, 56(8), 103335. https://doi.org/10.1016/j.arcmed.2025.103335.

[50] He, X., Hu, M., Xu, Y., Xia, F., Tan, Y., Wang, Y, et al. (2025). The gut–brain axis underlying hepatic encephalopathy in liver cirrhosis. Nature medicine, 31(2), 627-638. https://doi.org/10.1038/s41591-024-03405-9.

[51] Ren, G., Bai, C., Yi, S., Cong, Q., & Zhu, Y. (2024). Mechanisms and therapeutic strategies for MAFLD targeting TLR4 signaling pathways. Journal of Innate Immunity, 16(1), 45-55. https://doi.org/10.1159/000535524.

[52] Shen, S., Liu, Y., Wang, N., Huang, Z., & Deng, G. (2025). The role of microbiota in nonalcoholic fatty liver disease: mechanism of action and treatment strategy. Frontiers in Microbiology, 16, 1621583. https://doi.org/10.3389/fmicb.2025.1621583.

[53] Lopez, L. R., Bleich, R. M., & Arthur, J. C. (2021). Microbiota effects on carcinogenesis: initiation, promotion, and progression. Annual review of medicine, 72(1), 243-261. https://doi.org/10.1146/annurev-med-080719-091604.

[54] Yao, Y., Zhu, Y., Chen, K., Chen, J., Li, Y., Li, D., & Wei, P. (2026). Microbiota in cancer: current understandings and future perspectives. Signal Transduction and Targeted Therapy, 11(1), 39. https://doi.org/10.1038/s41392-025-02335-3.

[55] Chang, S. H., & Choi, Y. (2023). Gut dysbiosis in autoimmune diseases: Association with mortality. Frontiers in cellular and infection microbiology, 13, 1157918. https://doi.org/10.3389/fcimb.2023.1157918.

[56] Kumar, A., Pramanik, J., Goyal, N., Chauhan, D., Sivamaruthi, B. S., Prajapati, B. G., & Chaiyasut, C. (2023). Gut microbiota in anxiety and depression: unveiling the relationships and management options. Pharmaceuticals, 16(4), 565. https://doi.org/10.3390/ph16040565.

[57] Ataei, P., Kalantari, H., Bodnar, T. S., & Turner, R. J. (2025). The gut–brain connection: microbes' influence on mental health and psychological disorders. Frontiers in Microbiomes, 4, 1701608. https://doi.org/10.3389/frmbi.2025.1701608.

[58] Singh, S., & Rastogi, M. (2024). Prebiotics and probiotics: An overview on mechanism of action and therapeutic applications. SALT Journal of Scientific Research in Healthcare, 10–16. https://doi.org/10.56735/saltjsrh.ms2404011016.

[59] Ji, J., Jin, W., Liu, S. J., Jiao, Z., & Li, X. (2023). Probiotics, prebiotics, and postbiotics in health and disease. MedComm, 4(6), e420. https://doi.org/10.1002/mco2.420.

[60] de Sequeira C. L. M., Hengstberger C., Enck P., Mack I. (2022). Effect of probiotics on psychiatric symptoms and central nervous system functions in human health and disease: a systematic review and meta-analysis. Nutrients, 14, 621. https://doi.org/10.3390/nu14030621.

[61] Al-Habsi, N., Al-Khalili, M., Haque, S. A., Elias, M., Olqi, N. A., & Al Uraimi, T. (2024). Health benefits of prebiotics, probiotics, synbiotics, and postbiotics. Nutrients, 16(22), 3955. https://doi.org/10.3390/nu16223955.

[62] Yang, S. Y., Han, S. M., Lee, J. Y., Kim, K. S., Lee, J. E., & Lee, D. W. (2025). Advancing gut microbiome research: the shift from metagenomics to multi-omics and future perspectives. Journal of microbiology and biotechnology, 35, e2412001. https://doi.org/10.4014/jmb.2412.12001.