JOURNAL OF PHARMA INSIGHTS AND RESEARCH

REVIEW ARTICLE

The Role of Gut Microbiomes in Different Diseases

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Publication history**:** Received on 12th February; Revised on 24th February; Accepted on 27th February

Article DOI: 10.5281/zenodo.10845566

Abstract: The human gastrointestinal tract harbors trillions of microbial organisms collectively known as the gut microbiota. Next-generation sequencing techniques have revealed that the gut microbiota is composed of hundreds of bacterial species belonging predominantly to the phyla Firmicutes and Bacteroidetes. Under homeostatic conditions, the gut microbiota performs vital functions such as educating the immune system, defending against pathogen colonization, metabolizing nutrients, and producing neurotransmitters and vitamins. However, disruptions to the delicate balance of the gut microbiota composition, known as dysbiosis, have been linked to several diseases. Recent studies have demonstrated associations between dysbiosis and various systemic illnesses including rheumatoid arthritis, metabolic syndrome, neurological disorders, cardiovascular diseases, respiratory illnesses, and liver conditions. Furthermore, certain gastrointestinal disorders themselves have been shown to alter the gut microbiota constituents. This comprehensive review aims to summarize the latest research investigating the potential role of gut microbial dysbiosis in the pathogenesis and progression of the aforementioned diseases. Understanding microbiota-disease relationships could lead to novel diagnostic, therapeutic and preventive strategies.

Keywords: Gut Microbiome; Microbiota Dysbiosis; Gut Bacteria; Microbial Homeostasis; Microbiome-Based Therapies

1. Introduction

A population of bacteria and other microorganisms known as the gut microbiome influences how nutrition affects human health, both directly and indirectly [1]. Numerous investigations have revealed that the gut microbiota is important in immune-related illnesses and in regulating disease susceptibility [2]. Due to nutritional, environmental, and genetic variables, The makeup of the gut microbiome, which starts to grow and multiply from birth, might change [3]. Interacting with the human host, bacteria, fungi, viruses, and archaea are just a few of the diverse, complex, and dynamic microorganisms that make up the ecosystem [4]. The four primary groups that make up the gut microbiome are Actinomycetes, Bacteroides, Firmicutes, and Proteus [4]. Immune responses, digestion, metabolism, and intestinal permeability can all be impacted by modifications in the makeup and activity within the gut microbiome [5]. The identification of microbiological populations have been created. possible by recent developments in metagenomics, bioinformatics tools, and next-generation sequencing technology [6]. Among other important characteristics, it has a considerable impact on inflammation, metabolism, and immunological response, among other illnesses [7, 8]. Numerous disorders, including mental health illnesses obesity, diabetes, metabolic disorders, neurodegenerative disorders, cardiovascular conditions, and inflammatory bowel disease, have been linked to imbalances in the microbiome [8]. Although the precise origin of rheumatoid arthritis (RA) is still unknown, It's believed to be a result of both environmental and inherited factors [9]. Although the precise origin of rheumatoid arthritis (RA) is still unknown, it is thought to be a result of both environmental and inherited factors [10]. Patients with the condition, which attacks self-antigens in the bone, cartilage, and synovium, usually experience joint injury and functional impairment [11]. According to autoantibodies, an autoreactive immune response occurs much before clinical signs of RA manifest [11]. Dysbiosis of the gut microbiota, a complex process with significant health effects that may lead to chronic illnesses, can be brought on by genetic modification (GM) [12]. Over the past few decades, a rise in sedentary behavior and high-calorie food consumption has created a positive energy balance, which has altered the key risk factors for obesity [13]. Research demonstrates that the gut microbiota is important in neurodegeneration and that there is a great deal of interaction between the two because of certain characteristics [14]. Because the microbiota may influence neurotransmitter production and brain inflammation via the gut-brain axis, new research points to a connection between gut health and conditions including Parkinson's and Alzheimer's [14, 15]. The microbiota in the gut is a distinct endocrine organ that regulates host cardiometabolic function, according to recent research, and the TMAO molecule is predictive of both the prevalence of cardiovascular illnesses and future cardiovascular events [16]. Numerous medical problems, including bronchiectasis, asthma, copd, and respiratory virus infections, have been studied in relation

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to lung microbiome [17]. Numerous illnesses, particularly liver ailments, are connected to dysbiosis, a breakdown of the host and microbiota's mutual interaction [18]. Dysbiosis, a breakdown of the mutual interaction between the microbiota and host bacteria, is linked to diseases that impair immunity, health, digestion, metabolism, and nutritional absorption. Liver issues are among the conditions that fall under this category [18]. Changes in the gut's microbes have the potential to produce gastrointestinal illnesses including IBS and IBD because they change the composition of the microbial population [19]. From early life to old age, the host-immune system is greatly influenced by the microbiome in terms of its growth, function, and regulation [20].

2. Factors affecting Gut microbiota

2.1. Age and mode of parturition

Delivery methods impact the early development of the microbiota that is present in the placenta and amniotic fluid during microbial gut colonization in pregnancy. The parturition pattern like vaginal birth, cesarean section can also impact how the microorganisms grow and the products that they produce are involved [21].

2.2. Diet

The microbiota of the infant's gut throughout infancy influences immunity. Western diets increase bacteria and decrease Firmicutes, but vegetarian diets preserve a balanced, diversified gut microbiota [21].

2.3. Medications, especially antibiotics

Antibiotic therapies disrupt the microbial populations in the gut, which impacts pathogenic and host-associated bacteria. Antibiotic-resistant bacteria may be fostered by them, which could result in immunological dysregulation and heightened susceptibility to illness [22]

2.4. Probiotics

In combination with Lactobacillus rhamnoses and Saccharomyces boulardii, probiotics have been shown to improve gut flora, stabilize the microbiome, and inhibit pathogenic microorganisms when administered in sufficient dosages [23]

2.5. Prebiotics

Prebiotics and a high-fiber diet can stimulate Bifidobacterium growth, enhance metabolic performance, and improve the composition of the microbiota, as gut microbes metabolize indigestible substances [24]

3. Role of microbiota in diseases

3.1. Rheumatoid arthritis

Rheumatoid arthritis (RA) is a chronic inflammatory condition with a multifaceted causation, arising from an interplay of environmental triggers and genetic predispositions [25,26]. The precise role of the gut microbiota in the pathogenesis of human RA remains elusive. However, several studies have demonstrated that individuals with RA exhibit distinct alterations in the composition of their gut microbial communities. Vaahtovuo et al. employed a multi-pronged approach, combining flow cytometry techniques, 16S rRNA hybridization, and DNA staining, to characterize the gut microbiota profiles in patients with early-stage RA or fibromyalgia [26]. The RA patients had higher prevalences of Eisenbergiella, Flavobacterium, Escherichia coli, and Klebsiella than the healthy controls had of Fusicatenibacter, Megamonas, and Enterococcus [28]. Researchers discovered that the species Bifidobacterium, Eubacterium rectale-Clostridium coccoides, and among RA patients, the Bacteroides fragilis subgroup was all less common [27]. A growing body of research has looked into the connections between the gut microbiota and those who have RA in recent years. For example, it was shown that Prevotella copri is common and that persons with newly formed untreated RA show chromosomal rearrangement; additionally, one of its 27-kDa proteins was reported to be able to boost the Th1 response in 42 percent of RA patients [26]. Effects of probiotic therapy on treating and preventing arthritis [29]

The effects of the gut microbiomes on rheumatoid arthritis (RA) are:

- Modification of the immune system: In individuals with rheumatoid arthritis, an imbalance of the gut microbiota balance can result in persistent inflammation.
- Microbial Metabolites: Chemicals and metabolites made by gut bacteria have the potential to affect the body as a whole. For example, studies show that the creation of short-chain fatty acids (SCFAs) by gut bacteria has anti-inflammatory

properties. Variations in the synthesis of these metabolites could impact the inflammatory mechanisms associated with rheumatoid arthritis.

- Intestinal Permeability: Damage to the intestinal lining that results in increased permeability may facilitate the entry of microorganisms or germs into the bloodstream. This may exacerbate similar as rheumatoid arthritis by inducing inflammatory and immunological reactions.
- Synovial Inflammation: Rheumatoid arthritis may develop or worsen as a result of systemic effects from gut-derived chemicals, such as inflammation. Synovial joint tissues may potentially
- Synovial Inflammation: Rheumatoid arthritis may develop or worsen as a result of systemic effects from gut-derived chemicals, such as inflammation. Synovial joint tissues may potentially be directly impacted by inflammation. [30]

3.2. Metabolic Disorders

Metabolic diseases that are closely linked to unhealthy lifestyles include weight gain, type 2 diabetes, nonalcoholic fatty liver disease, and cardiovascular disease. However, an increasing number of studies has also demonstrated the significance of the gut microbiota and its metabolites for the development and course of certain diseases. [31].

3.2.1. Type I Diabetes

This condition is long-term and results in minimal or no insulin production by the pancreas. Juvenile diabetes is another term for this condition. Symptoms may include thirst, hunger, tiredness, sweating, nausea, vomiting, blurred vision, a fast heartbeat, drowsiness, or weight loss. The gut immune system plays a major role in the development of autoimmune diabetes, and the same mechanisms that regulate the gut immune system also regulate beta-cell autoimmunity. [32]. A significant amount of variety is seen in these microorganisms because of things like host lifestyle, location, illnesses, food, sex, age, and genetic background [33]. medications that alter the microbiota of the intestines. Surprisingly, the majority of specialists think that type 1 diabetes is primarily caused by autoimmunity, other scientists believe that other factors—such as stress on the endoplasmic reticulum, which results in beta cell death—may have a greater bearing. Previously, hyperglycemia that occurred or existed before β-cell ablation and the presence of one or more autoantibodies were used to diagnose type 1 diabetes (T1D) [33]. The turning on of anti-inflammatory Treg cells have been connected to other beneficial bacteria, such as Lactobacillus, Clostridium species, and Bifidobacteria. Conversely, research has demonstrated that Bacteroides fragilis polysaccharide A (PSA) inhibits Th17 cell responses and increases IL-10 production. [33]. an autoimmune condition when the immune system mistakenly targets the insulin-producing pancreatic cells [33, 34]. Fortunately, probiotics have been shown to have potential as a treatment for type 1 diabetes [35]. This dysbiosis may impact the inflammatory environment that could trigger the onset of autoimmune reactions. Certain gut bacteria produce metabolites, like short-chain fatty acids (SCFAs), which have been linked to immunological regulation [35]

3.2.2. Type II Diabetes

Type 2 diabetes is a chronic metabolic illness characterized by insulin resistance, or the body's inability to use insulin as intended, and insufficient pancreatic insulin synthesis, which results in hyperglycemia, or increased blood glucose levels. It is a metabolic disease that is growing at the fastest rate in the world. Numerous clinical investigations have shown that individuals with type 2 diabetes (T2D) have chronic inflammatory states, altered gut flora, and increased permeability of the intestines [36]. Due to aberrant intestinal metabolites and intestinal barrier disruption brought by the gut microbiota's dysbiosis in type 2 diabetes, intestinal bacteria and their harmful byproducts are more easily able to enter the circulation [36]. The abnormal entrance will impair immunological homeostasis, glucose metabolism, and insulin sensitivity, which will affect multiple organs [36]. Numerous studies have shown that obesity is a major risk factor for type 2 diabetes [T2D], which is characterized by insulin resistance and hyperglycemia. Type 2 diabetes (T2D) is characterized by insulin resistance and hyperglycemia, and as the condition progresses, a chronic inflammatory state persists. Research from preclinical and clinical contexts provides credence to the notion that a variety of metabolic diseases might be prevented or treated by intermittent fasting. Patients diagnosed with diabetes mellitus exhibited increased levels of Escherichia coli, Shigella, Lachnospiraceae incertae_sedis, Subdoligranulum, Enterococcus, and Klebsiella at the genus level, with differing degrees of increase. Prevotella and Bacteroides exhibited the greatest decrease. Millions of individuals with diabetes mellitus experience severe consequences such retinopathy, renal disease, and neuropathy [37]

3.2.3. Obesity

It is characterized as an unhealthy, atypical, or excessive accumulation of fat. Animals with higher body weight and fat mass were created when the gut microbiome of genetically modified obese mice was transferred to germ-free mice (mice without any bacteria) [38]. The majority of the human mammalian gut microbiota is composed of four bacterial phyla: the Gram-negative Bacteroidetes and Proteobacteria, and the Gram-positive Actinobacteria and Firmicutes [39]. The role of the gut microbiota in regulating body weight was first shown by alterations in its composition and function in obesity models controlled by diet and genetics [40]. A varied, well-balanced microbiome has been linked to a healthier weight in studies, highlighting the significance of nutrition and lifestyle in preserving microbial equilibrium [41]. The variety of the microbiota, energy harvesting, and food absorption are some of the variables that help to explain how the microbiome plays a role in obesity [Alterations in the microbiome's composition could

improve the body's ability to absorb calories from diet, which could result in weight gain], metabolic functions (disorders in the microbiome may affect metabolic pathways, leading to increased fat storage), inflammation and insulin resistance, short-chain fatty acids (SCFAs), modulation of the endocannabinoid system, and personalized responses [41]. Probiotics have been recommended as a treatment approach to prevent certain intestinal diseases. Probiotics are known to have antibacterial, anti-inflammatory, and even anti-carcinogenic capabilities, which may help repair the unbalanced gut flora [42]

3.3. Neurodegenerative diseases

Non-communicable diseases (NDDs) are a major global health concern that affect millions of people worldwide. The three most frequent pathologies are amyotrophic lateral sclerosis (ALS), Parkinson's disease (PD), and Alzheimer's disease (AD) [43].

3.3.1. Parkinson's Disease

Parkinson's disease is a progressive neurodegenerative condition characterized by the loss of dopaminergic neurons in the substantia nigra [44]. Enteral dysfunctions associated with Parkinson's disease (PD) include malnourishment, H. pylori infection, and constipation. According to studies on the significance of GI tract anomalies in Parkinson's disease (PD), Patients had a higher incidence of present α-syn than controls; studies on animals have shown that α-syn cannot cross from the gut to the central nervous system (CNS) if the vagus nerve is severed; additionally, intestinal inflammation in animals can lead to neuroinflammation in the substantia nigra, which promotes the death of dopaminergic neurons; further research by Forsyth et al. has shown that intestinal inflammation and "leaky intestines" influence the rate of progression of Parkinson's disease. [45]. An increasing amount of evidence suggests that the GM makeup of the PD and healthy groups changes considerably, yet the results are marginally different. The gut microorganisms of individuals with Parkinson's disease (PD) contained significantly higher abundances of Bifidobacterium, Pasteurella, and Enterococcus than of Prevotella, Faecococcus, and Brautella, according to several investigations. Bifidobacterium is found in higher concentrations in people with Parkinson's disease (PD), which may act as a buffer against the progression of neurodegenerative diseases. It follows that a probiotic intervention, such as one containing Bifidobacterium, might stop Parkinson's disease (PD) from getting worse [46].

3.3.2. Alzheimer's Disease

The degenerative neurological disorder known as Alzheimer's disease (AD) is typified by changes in behavior, memory loss, and difficulties with daily functioning. The two most important neuropathologic indicators for the diagnosis of AD are the extracellular deposition of β-amyloid and the intracellular buildup of hyperphosphorylated tauThe gut microbiota's acceleration of neuroinflammation has an impact on the development of AD [47], which encourages the evolution of senile plaques even further. In a clinical investigation of AD patients, amyloid-positive patients had higher levels of Shigella and Escherichia coli in their stools and lower levels of Bacillus subtilis and Eubacterium rectale. Based on this research, cognitive impairment is associated with both amyloid and relevant bacterial accumulation [48]. Declines in the number of helpful bacteria have been linked to decreased quantities of SCFAs, which have been linked to intestinal barrier failure, neuroinflammation, and an increased susceptibility of AD neurons to injury. Additionally, increased levels of the bacteria that produce lipophilic acid (LPS) have been connected to the enhancement of neuroinflammation, a crucial clinical feature linked to a number of neurological disorders, including AD, as well as the translocation of microbiological substances into the brain. [49]

3.4. Cardiovascular disorders

The relationship between cardiovascular illnesses and gut microbiota has been demonstrated. The main pathogenic processes of hypertension, atherosclerosis, and cardiovascular disease (CVD) are inflammatory or infectious states, and these states can be improved by physiological changes brought on by a lack of diversity in the microbiota [50]. Variations in the microbiome could have a part in the development of cardiovascular diseases (CVD).

3.4.1. Hypertension

Throughout the world, high blood pressure is a common cause of CVD. The first evidence of the function of gut microbiota in the genesis of hypertension came from a test of rats' blood pressure in response to antibiotic treatment. Therefore, it was discovered that the fecal microbiota of animal models and hypertensive individuals had lower levels of microbial evenness, variety, and richness. The severity of hypertension was found to be connected with high abundances of opportunistic pathogenic taxa (e.g., Streptococcus spp. and Klebsiella spp.), fewer numbers of bacteria that produce butyrate and acetate, and a higher number of bacterial species related to hypertension [51]

3.4.2. Atherosclerosis

Atherosclerosis, a chronic inflammatory condition marked by the buildup of lipids and cells in the artery walls, is the main cause of cardiovascular disease (CVD). Leukocytes, endothelium, and foam cells in membranes and calcified regions are some of these cells. [52]. As per current research, gut dysbiosis may encourage atherosclerosis. When compared to healthy controls, study found that individuals with atherosclerosis had higher levels of Collinsella and reduced relative abundances of Roseburia and Eubacterium [53].

Moreover, the anti-atherosclerotic and gut barrier-enhancing properties of Akkermansia muciniphilia were discovered. The connection between the beginning of atherosclerosis, trimethylamine (TMA), and TMAO has been the focus of numerous analyses. The digestion of food-borne components such as carnitine, choline, and lecithin causes gut microbes, especially those in the Enterobacteriaceae and Clostridia families, to produce TMA. The hepatic enzyme flavin monooxygenase oxidizes TMA upon consumption, resulting in the formation of trimethylamine-N-oxide (TMAO). Research indicates that TMAO increases the synthesis of pro-inflammatory cytokines such as TNF-alpha and IL-1B while decreasing the synthesis of anti-inflammatory cytokines such as IL-10. Atherosclerotic thrombotic events have been linked to TMAO-induced platelet hyperreactivity, which may facilitate thrombosis. [54, 55]

3.5. Respiratory diseases

Digestive problems or symptoms are frequently linked to a number of chronic respiratory illnesses, such as respiratory virus infections, asthma, and chronic obstructive pulmonary disease (COPD). Pulmonary dysfunction and respiratory infections are more common in patients with digestive disorders such as inflammatory bowel disease (IBD) and gastroesophageal reflux disease [56]. Asthma is a chronic airway condition characterized by eosinophilia, increased Th2 cytokine production, increased mucus generation, and excessive smooth muscle contraction of the airways [57]. Certain microbiota components are necessary for the immune system's growth and maturation, and their absence may make a person more susceptible to allergic reactions and asthma attacks. In instance, a direct link has been shown between microbial diversity and asthma. For instance, the intestines of asthmatic patients contained greater amounts of Clostridium and Eggerthella lenta than those of healthy controls [58]. Furthermore, a higher risk of allergies and asthma in children was linked to a decrease in the bacterial abundances of Bifidobacterium, Akkermansia, and Faecali and an increase in the bacterial abundances of Rhodotorula and Candida [59].

3.5.1. Chronic Obstructive Pulmonary Disease (COPD)

It's a lung condition marked by limited airflow and enduring respiratory symptoms. These include chronic bronchitis, bronchiolitis, asthma, mucus hypersecretion, and small airway disease [60]. Research indicates that different COPD patients may have different amounts and kinds of specific bacterial species in their stomachs. Variations have been documented in specific bacteria, including Bacteroides, Prevotella, and Firmicutes [60]. Numerous organisms are also linked to decreased lung function, including Streptococcus vestibularis, sp000187445, and other Lachnospiraceae family members. [61]. Untargeted metabolomics reveals a COPD signature made up of 20% xenobiotic, 20% amino acid related, and 46% lipid related compounds [61]. Compositional changes may affect immunological responses and inflammation, which in turn may affect the course and severity of COPD [62]. Exacerbations of COPD are associated with a worsening of the patient's quality of life, health status, and the course of the illness, making patient follow-up and treatment difficult. This is because they quicken the rate at which lung function degrades. For COPD patients, exacerbations are the primary cause of death and the basis for recurrent medical visits, hospital stays, and drug modifications [62]. The main causes of risk for COPD include cigarette smoking, both active and passive [62]. Interestingly, Western-diet-following COPD patients have lower lung function and higher mortality rates. Conversely, a diet high in antioxidants improves lung function and reduces death from COPD [62]. Nutrition is one easily modifiable factor, and diets deficient in key nutrients, such as fiber, are common in people with COPD [63]. Short chain fatty acids (SCFAs) with anti-inflammatory properties are produced when gut microbiomes break down dietary fiber, and this may provide protection against lung inflammation [63]. Through fermentation, the gut microbiota creates several metabolites, the most well-known of which are short-chain fatty acids (SCFAs) [63]. Sturdy anti-inflammatory compounds known as short-chain fatty acids (SCFAs) reduce immune cell adhesion and chemotaxis, cause apoptosis, and produce more anti-inflammatory cytokines [63].

3.5.2. Allergies

It has been noted that eczema often appears as the initial symptom of an atopic diathesis and that newborns with eczema have altered gut microbiome diversity (64). According to research, the gut microbiome of people who have Alzheimer's disease (AD) is characterized by higher concentrations of harmful bacteria like Staphylococcus aureus and Streptococcus pyogenes and lower amounts of beneficial bacteria like Lactobacillus and Bifidobacterium. These changes in the gut microbiota may be the cause of the onset and development of AD, exacerbating inflammation and compromising the integrity of the intestinal barrier. Based on the skin microbiome investigation, new suggestions have been put forth about the mechanisms of microbial dysbiosis [65]. One such theory links the development of allergies to Staphylococcus aureus colonization, which is a hallmark characteristic of AD. It is complex and has a lot of features. Two potential causes are impaired barrier function and inflammation. Allergies may manifest themselves [65]. The first biologic for AD, dupilumab, an anti-IL-4 receptor α antibody, will be available in clinical practice in 2017. It successfully completed phase III clinical trials for the treatment of AD [65]

3.6. Liver disorders

The gut-liver axis represents a profound physiological link between the intestines and the liver. The liver is the initial organ exposed to blood originating from the gut, rendering it highly susceptible to the influence of the gut microbiota and its metabolic byproducts. Disruptions in intestinal permeability and dysbiosis of the gut microbial communities are recognized as primary drivers of aberrant

liver responses. Pathological conditions affecting the liver encompass viral hepatitis, cirrhosis, and non-alcoholic fatty liver disease [66].

3.6.1. Non Alcoholic fatty liver disease

Non-alcoholic fatty liver disease (NAFLD) refers to the pathological accumulation of fat in the liver, resulting from metabolic abnormalities. It stands as the primary causative factor underlying chronic liver disease [67]. Individuals with NAFLD exhibit a distinct gut microbial profile, characterized by a predominance of the Fusobacteria species, while Oscillospira and Ruminococcus genera are relatively less abundant. Notably, pediatric NAFLD patients display a higher prevalence of Ruminococcus and Oscillospira compared to healthy children. Obesity represents a significant risk factor for NAFLD, and the accompanying dysbiosis exacerbates the condition's progression. Non-obese adults with NAFLD exhibit gut dysbiosis, manifested by a phylum-level shift in the gut microbiota composition, with a decrease in Firmicutes and a concomitant increase in Bacteroidetes [68]. Furthermore, children with NAFLD exhibit higher levels of Ruminococcus and Oscillospira compared to their healthy counterparts. Obesity is a well-established risk factor for NAFLD, and the dysbiotic state associated with obesity exacerbates the condition's severity. Non-obese adults with NAFLD also demonstrate gut dysbiosis, characterized by an altered phylum-level composition of the gut microbiota, with a reduction in Firmicutes and a corresponding increase in Bacteroidetes populations [68]. Despite the lack of a clear symptom of dysbiosis, every pediatric study has found decreased α-diversity, notable β-diversity alterations, or variable abundances of bacteria at the phylum or genus levels. Diminished α-diversity, commonly called richness and evenness, is arguably the most precise indicator of the dysbiosis connected to pediatric non-alcoholic fatty liver disease. Usually, the Shannon and Chao 1 diversity indices are used to calculate it [69]

3.6.2. Hepatitis

Persistent infections with hepatitis B virus (HBV) and hepatitis C virus (HCV), collectively termed as hepatitis viruses, are implicated in the development of chronic liver diseases. The gut microbiome exerts a profound influence on the progression of liver injury induced by these viral pathogens. Alterations in the gut microbial composition exhibit a strong correlation with the clinical course of liver pathologies [70]. The gut microbiota plays a pivotal role in the eventual manifestation of liver failure and is recognized as a crucial determinant in the development of chronic hepatitis B infection. Research studies have suggested that specific bacterial genera, including Faecalibacterium, Pseudobutyrivibrio, Lachnoclostridium, Ruminoclostridium, Prevotella, Alloprevotella, and Phascolarctobacterium, may possess anti-inflammatory properties mediated by an increased abundance of short-chain fatty acids (SCFAs), such as butyrate, relative to healthy individuals. Investigations have revealed notable variations in the copy numbers of F. prausnitzii, E. faecalis, Enterobacteriaceae, Bifidobacteria, and lactic acid bacteria in the intestinal tract of patients with HBV-related cirrhosis. Furthermore, an imbalance in the oral microbiome has been associated with HBV infection, with yellow tongue coating indicative of a decrease in Bacteroidetes and a concomitant increase in Proteobacteria [71]. Chronic hepatitis C infection represents another significant contributor to the development of cirrhosis, hepatocellular carcinoma (HCC), and, in some cases, liver failure and mortality. The majority of long-term HCV patients exhibit a reduced abundance of Firmicutes and a corresponding increase in Bacteroidetes and Enterobacteriaceae populations. Significantly elevated levels of LPS are a marker of HCV infection, and as the disease progresses, these levels may indicate microbial translocation and inflammation [72]

3.7. Gastrointestinal disorders

There is limited knowledge pertaining to the composition of the intestinal microbiota and their involvement in the gastrointestinal (GI) illnesses. Patients with inflammatory bowel diseases (IBD), diverticular disease (DD), and irritable bowel syndrome (IBS) had different gut microbiota compositions. Those with DD, IBS, and IBD were included in the study in addition to healthy individuals [73].

3.7.1. Inflammatory Bowel Disease (IBD)

Both ulcerative colitis and Crohn's disease are the two main inflammatory bowel diseases [IBD] that have a clinical diagnosis. These illnesses are recurrent idiopathic inflammatory aliments that can impact either the colon wall [UC] or only the outermost layers of the gastrointestinal tract

3.7.2. Crohn's Disease (CD)

Patients with IBD are less likely to be classified as firmicutes, which is the most notable alteration that has been seen. People with CD are known to have fewer Firmicutes—more especially, Faecalibacterium prausnitzii—than controls. Conversely, a pediatric cohort of CD patients exhibited higher levels of Faecalibacterium prausnitzii, indicating a more dynamic role for this bacterium and maybe a preventative effect at the outset of IBD. [75]

3.7.3. Ulcerative colitis (UC)

In contrast to healthy people, those who have IBD have an unstable gut microbiota that varies less over time. The phases of quiescence and activity of the gut microbiota are not the same. Furthermore, even in individuals with ulcerative colitis (UC) who

were in remission, the gut microbiota of these patients remained unstable, according to a year-long study. A reduction in the variety of the gut microbiota and common anaerobic bacteria, such as Ruminococcin, Bacteroides, Escherichia, Eubacterium, and Lactobacillus, occurs before UC recurrence [76]. Table 1 shows a list of diseases where the microbiota are linked.

Table 1 Summary showing the role of microbiota in several diseases

Disease	Associated Bacteria (Increased/Decreased)	Role of Microbiota	References
Rheumatoid Arthritis	Increased: Eisenbergiella, Flavobacterium, Escherichia coli, Klebsiella Decreased: Fusicatenibacter, Megamonas, Enterococcus, Bifidobacterium, Eubacterium rectale-Clostridium coccoides	Modulation of immune system, microbial metabolites impact inflammation, intestinal permeability influences disease	[26-30]
Type 1 Diabetes	Decreased: Bifidobacteria, Lactobacillus, Clostridium species Increased: Bacteroidetes, Proteobacteria	Dysbiosis impacts inflammatory environment triggering autoimmune reactions, certain bacteria produce metabolites regulating immunity	[32-35]
Type 2 Diabetes	Increased: Escherichia coli, Shigella, Lachnospiraceae incertae_sedis, Subdoligranulum, Enterococcus, Klebsiella Decreased: Prevotella, Bacteroides	Dysbiosis leads to intestinal metabolites and barrier disruption increasing circulatory bacteria and impairing metabolism/insulin sensitivity	[36-37]
Obesity	Increased: Actinobacteria, Proteobacteria Decreased: Bacteroidetes, Firmicutes	Variations in microbiome composition, energy harvesting and nutrient absorption impact body weight regulation	[38-42]
Parkinson's Disease	Increased: Bifidobacterium Decreased: Prevotella, Faecococcus, Brautella	Dysbiosis increases neuroinflammation via gut-brain axis exacerbating disease progression	[44-46]
Alzheimer's Disease	Increased: Shigella, Escherichia coli Decreased: Bacillus subtilis, Eubacterium rectale	Dysbiosis increases neuroinflammation and compromise intestinal barrier contributing to disease pathogenesis	[47-49]
Hypertension	Increased: Streptococcus spp., Klebsiella spp. Decreased: Butyrate- and acetate-producing bacteria	Dysbiosis impacts host inflammatory states influencing disease development	[51]
NAFLD	Increased: Fusobacteria Decreased: Oscillospira, Ruminococcus	Dysbiosis exacerbates disease progression via metabolic abnormalities and inflammation	[67-69]

4. Conclusion

The human gut microbiome plays a significant role in various diseases, both beneficial and detrimental. The composition and function of the gut microbiome can differ in individuals with diseases such as rheumatoid arthritis, metabolic disorders, neurodegenerative diseases, respiratory diseases, liver diseases, and gastrointestinal diseases. These changes in the gut microbial landscape, known as dysbiosis, can contribute to the pathogenesis and progression of these conditions. Conversely, restoring a healthy gut microbiome balance may alleviate disease symptoms and improve overall health outcomes. Further research is warranted to elucidate the intricate interplay between the gut microbiota and various pathologies, paving the way for novel microbiome-based therapeutic interventions.

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