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Unraveling Microbiota Dysbiosis in Gastrointestinal Disorders

Virender Kaur[1,*], Suresh Babu Kondaveeti[2], H.S. Batra[2], Preeti Biswas[3], Arushi[4], Sadhana Rai[5]

[1]College of Pharmacy, Graphic Era Hill University, Bhimtal campus, (Uttarakhand)- 263136, India; verumalwa@gmail.com
[2]Department of Biochemistry, Symbiosis Medical College For Women, Symbiosis International (Deemed University), Pune, India; ksuresh.babu@smcw.siu.edu.in; hod.biochemistry@smcw.siu.edu.in
[3]Faculty of Pharmacy, Mohammad Ali Jauhar University, Rampur, Uttar Pradesh, 244901, India; preetibiswas15@gmail.com
[4]Department of Pharmaceutics, RIMS Split Campus, Motherhood University, Roorkee, Uttarakhand-247670, India; arushi12334@gmail.com
[5]Department of Botany, NIMS University Jaipur, Rajasthan; sadhanaraigzp4040@gmail.com

***Corresponding Author:** Virender Kaur, College of Pharmacy, Graphic Era Hill University, Bhimtal campus, (Uttarakhand)- 263136, India; **Email ID:** verumalwa@gmail.com

ABSTRACT

The recent development of advanced sequencing methods has revealed the complexity and diversity of roles played by the gut bacteria. Furthermore, a number of gastrointestinal illnesses are associated with dysbiosis, which is a shift in the composition or balance of the intestinal microbiota. Dysbiosis of the gut microbiota is increasingly implicated in the pathogenesis of intestinal and extra-intestinal diseases. Celiac disease, inflammatory bowel disease, and irritable bowel syndrome (IBS) are intestinal disorders, whereas allergies, asthma, metabolic syndrome, cardiovascular disease, and obesity are extra-intestinal ailments. In many of these conditions, the pathways leading to the development of sickness are significantly influenced by the vital mutualistic interaction between the host immune system, the intestinal microbiota, and their metabolic products. Maintaining intestinal homeostasis appears to depend on the development of a "healthy" link early in life. In particular, the kinds of bacteria that inhabit the mucous layer of the colon may have an effect on the induction of inflammatory processes or the preservation of host cellular homeostasis. Direct contact with host cells or indirect communication via bacterial metabolites can also result in this. This review explores the intricate relationship between microbiota dysbiosis and gastrointestinal (GIT) disorders to explain how alterations in gut microbial communities contribute to disease etiology.

Keywords: Microbiota dysbiosis, Gastrointestinal disorders, Inflammatory bowel disease, Gut microbiome.

INTRODUCTION

The majority of the microorganisms that make up the human gut microbiota are non-pathogenic bacteria and viruses, which number in the trillions. To stop pathogen colonization and invasion, the microbiota cooperates with the host's defenses and immune system. By acting as a source of vitamins and other minerals and assisting in the extraction of energy and nutrients from meals, such as short-chain fatty acids (SCFA) and amino acids, it also performs an essential metabolic function. Ultimately, the gut microbiota of the host is required for numerous vital functions that could enhance health. However, determining the precise function of gut microbiota in human disease and its impact on human health is difficult (1-5). The microbiota may alter as a result of exposure to a variety of environmental factors, including as food, chemicals, medications, and illnesses. Enteric pathogens have the greatest ability to induce microbial dysbiosis in experimental animal models, as demonstrated by type 1 diabetes and the T-cell-mediated death of insulin-producing pancreatic β-cells. By altering the microbiota's composition and barrier function, foodborne viral infections can induce both local and systemic inflammation, which can lead to the development of autoimmunity (6-10). Documenting dysbiosis has always relied on the ability to produce pure isolates for identification and classification—a skill that is permanently limited to "culturable" microbes and traditional microbiological techniques. High-throughput DNA-based pyrosequencing technology has made it feasible to quickly and thoroughly profile complex communities of microorganisms by classifying bacteria and archaea based on individual 16S rRNA sequences from human samples (mostly fecal in origin) without the need for culturing. Since the inception of this approach, it has been demonstrated that the composition of the gut microbiota varies significantly among individuals. With favorable connections between the host's genetic relatedness under observation and similarity in the dominant fecal microbial communities, this could be partially explained by genetic variances among hosts. Although the phylums Bacteroidetes and Firmicutes are prevalent, other phylums include Proteobacteria, Actinobacteria, Fusobacteria, Spriochaetes, Verrucomicrobia, and Lentisphaerae. Using metagenomic analysis to investigate the functional capability of the intestinal microbiota genome (microbiome), it has been shown that approximately 40% of the microbial genes found in each individual are shared with at least half of the general population. The existence of a functional core, or core microbiome, is supported by this research (11-14). The main technique for studying alterations in the composition of the intestinal microbiota in relation to disease has been the phylogenetic characterisation of the microbiota of sick individuals in comparison to those who appear to be healthy. However, because the makeup of the gut microbiota varies significantly within and across individuals, as well as with age, it is difficult to pinpoint the precise correlations between the presence and relative abundance of specific microbial communities and human health (15-18). Future studies could make it possible to use specific changes in compositional diversity even functional diversity as biomarkers for specific diseases or health conditions. However, it is important to keep in mind that it is questionable whether differences in phylogenetic composition cause or contribute to a specific disease (19-25). Perhaps the strongest evidence of the direct role of the gut microbiota or its necessity in disease pathogenesis comes from studies conducted on germ-free animal models of human autoimmune diseases. With the use of these models, scientists may ascertain how exposure to and colonization by environmental microbes affect the development and course of the illness. Most disease models, though not all of them, show that the severity and incidence of illness are lower in germ-free surroundings. This is consistent with the microbiota serving as a "trigger" to advance the disease. However, thus far, there has been no success in identifying the "pathogenic" microbiota members (pathobionts) that can replicate the overall influence of the microbiota. It should not be surprising that intestinal dysbiosis is most commonly associated with GI-related disorders, where alterations in the host's (immune system's) response to antigens and lumen-derived stimuli result in and sustain uncontrollably high levels of inflammation in the intestinal mucosa and, in some cases, beyond (26-30).

2 Metabolomic Effect of the Relationship between Diet and the Microbiome on Human Health

In the small intestine, food particles are not completely broken down; instead, endogenous materials such as mucus, lost epithelial cells, and digestive enzymes enter the colon and are fermented by the colonic microbiota. In the vicinity of their host's cells, bacteria transform these materials into a variety of metabolites. The metabolic phenotype of the host and the likelihood of disease can be affected by these metabolites (31-34). The majority of the substrates that the microbiota can use are proteins and undigested carbohydrates. As these substrates ferment, a variety of metabolites are created, including branched-chain fatty acids (SCFA), ammonia, amines, phenolic compounds, and gasses like hydrogen, methane, and hydrogen sulfide. Additionally, vitamin production, the transformation of prodrugs into their bioactive forms, the modification of bile acids and xenobiotics, and the activation or inactivation of bioactive dietary components such as isoflavonoids and plant lignans are all influenced by the gut microbiota (see **Figure 1**) (35-40) The metabolomic interplay between diet and the microbiome significantly influences human health by modulating metabolic pathways, immune responses, and disease susceptibility. Dietary components alter gut microbiota composition, leading to the production of bioactive metabolites such as short-chain fatty acids, which impact inflammation, energy balance, and overall physiological homeostasis.The metabolomic interplay between diet and the microbiome significantly influences human health by modulating metabolic pathways, immune responses, and disease susceptibility.

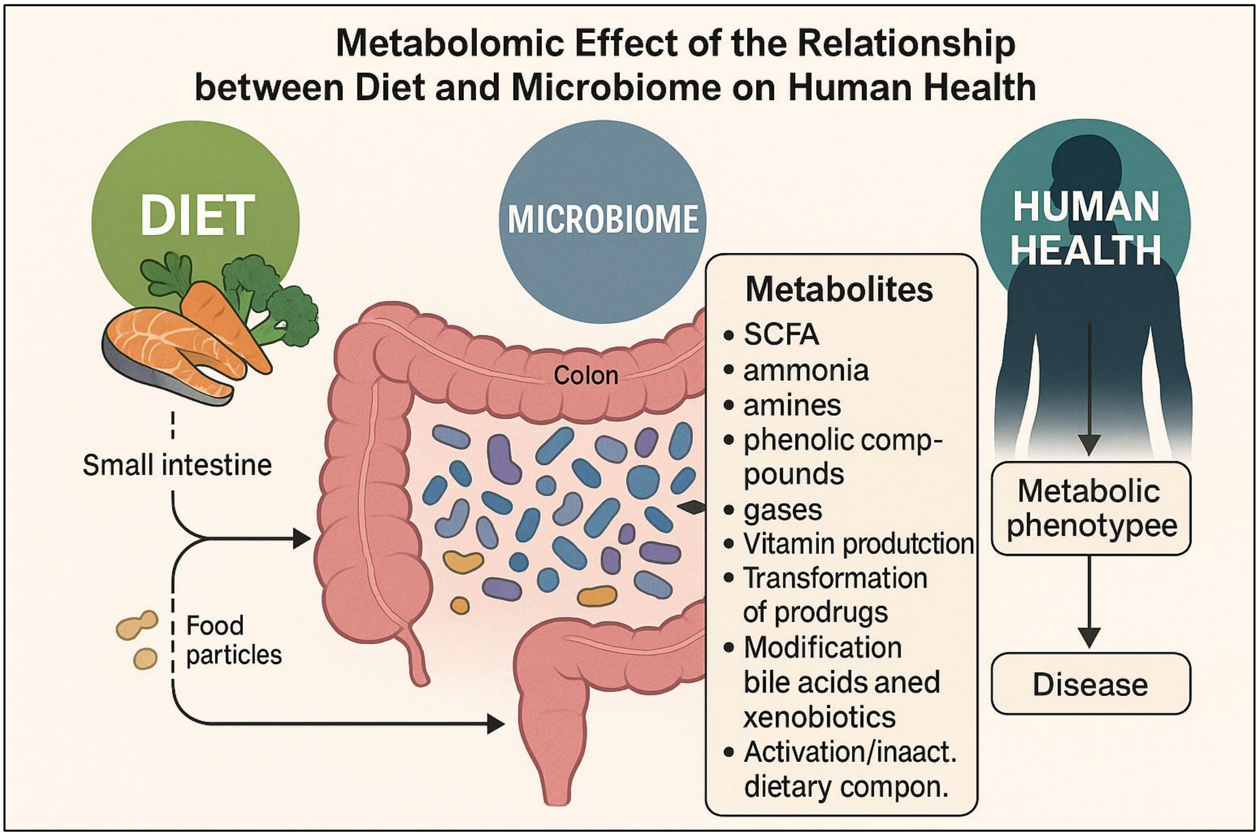

Figure 1. Metabolomic effect of the relationship between diet and the microbiome on human health

3 MAIN FUNCTIONS OF THE MICROBIOTA

Instead of existing as passive observers, our gut microbes have developed a close symbiotic relationship with humans. Average growth, development, and physiological equilibrium depend on microbial interactions with the surrounding mucosa as well as indirect cross-talk with the host through their metabolites (like short chain fatty acids) and host signaling pathways (like toll-like receptors). Studies on germ-free (GF) and antibiotic-treated mice demonstrated the important function of the gut microbiota (41-45). The resident microbiota offers a crucial initial line of defense against pathogen colonization because of their fight for resources in their biological niche and attachment sites on the gut epithelium's surface. They can also produce antimicrobial peptides like bacteriocins and regulate the intestinal mucin synthesis of goblet cells. The latter might prevent the germs from adhering to the cells of the epithelium. Gavaged Salmonella in GF mice infiltrate the intestinal mucosa more quickly than in their traditionally produced, normal gut microbiota counterparts, reducing this "colonization resistance" in both people and GF mice following antibiotic therapy (46-48). The microbiome also supports intestinal epithelial barrier integrity by maintaining tight junctions, promoting epithelial repair, and regulating enterocyte turnover. The GF mice's abnormal villi and crypts demonstrate the critical function of gut microbes in fostering the signals and gene expression necessary for barrier integrity and homeostasis (49-53). The digestive enzymes and metabolic activities needed to break down complex, "non-digestible" substances like proteins and carbohydrates are provided by the gut flora. Because they lack this more efficient metabolism, GF mice must consume significantly more food to obtain the same number of calories as their counterparts with normal microbiota. Microbiota metabolism aids in the biotransformation of bile acid, calcium, magnesium, and iron intake as well as the synthesis of vitamin K (54-56). The fermentation cycle culminates in the creation of short-chain fatty acids (SCFA), which provide energy to colonocytes and stimulate intestinal epithelial cell proliferation and differentiation in vivo, as well as mucin secretion, trefoil factor, and antimicrobial peptide synthesis (57-62). The immune system's main point of contact with the outside world and its antigens is the gut mucosa; the development of the immune system depends on the host-microbiota relationship. GF animals have a compromised immune system without this exposure to microbial antigens, as seen by smaller Peyers patches, fewer lymph nodes, decreased B and T cell activity, aberrant antibody production, and decreased oral tolerance. Recent studies have shown that SCFA generated by microbial fermentation is necessary for the production of regulatory T cells (63-69).

DYSBIOSIS IN DISEASE

4.1 Dysbiosis and GI-tract-related disorders

4.1.1 Inflammatory bowel disease

Crohn's disease (CD) and ulcerative colitis (UC) are the two most prevalent forms of inflammatory bowel disease (IBD), which harm the intestinal mucosa. Chronic, recurring inflammation is a hallmark of both illnesses. There is growing evidence that gut microbial dysbiosis contributes to the pathophysiology of IBD, despite the fact that the exact origin of both disorders is unknown (70-75). Overall, patients exhibit a decrease in the microbial population, stability, and functional diversity of their gut microbiota, along with an increase in facultative anaerobes such Enterobacteriaceae and a decrease in some Firmicutes. Additionally, there are discernible differences in the microbiota between patients with UC and CD. The primary dysbiosis in CD has been connected to five bacterial species; variations in the quantity of Faecalibacterium prausnitzii have been linked to the prolongation of disease remission, and this bacterium has been demonstrated to have therapeutic effects in animal models of colitis (76-82). However, although a causal relationship has not yet been established, adherent-invasive E. coli and Mycobacterium paratuberculosis have been connected to CD pathogenesis. It is still unclear if intestinal microbial dysbiosis is the source of the inflammation linked to inflammatory bowel disease (IBD) or if it is merely the result of an unbalanced environment in the GI tract. In one study, early-diagnosed CD patients' microbiome conditions were investigated (83-87). This studylooked at the microbiota of a sizable group of pediatric CD patients and found that the bacterial populations of CD patients differed significantly from those of healthy control subjects. Enterobacteriaceae, Pasteurellaceae, Veillonellaceae, and Fusobacteriaceae were more abundant in CD patients than in healthy control individuals, but Erysipelotrichales, Bacteroidales, and Clostridiales were less abundant. It is interesting to note that these differences were only seen in mucosal samples, not fecal ones, indicating that the bacteria that reside in the mucosal layer might be more important in the etiology of disease (see **Figure 2**) (88-95). Inflammatory bowel disease (IBD) is a chronic, relapsing-remitting inflammatory condition of the gastrointestinal (GI) tract, primarily encompassing two major disorders: Crohn’s disease (CD) and ulcerative colitis (UC). Both conditions significantly impair patients' quality of life, with symptoms ranging from abdominal pain, diarrhea, weight loss, rectal bleeding, and fatigue to more systemic complications such as arthritis, skin manifestations, and ocular inflammation. Though IBD is more prevalent in Western countries, its incidence and prevalence are rising globally, including in newly industrialized regions, suggesting an interplay between environmental, genetic, immunological, and microbiota-related factors in its pathogenesis. Ulcerative colitis is limited to the colon and rectum and primarily affects the mucosal and submucosal layers, beginning in the rectum and extending proximally in a continuous fashion. In contrast, Crohn’s disease can involve any part of the GI tract from mouth to anus and is characterized by transmural inflammation with skip lesions and granuloma formation. The heterogeneity in disease location and severity contributes to the variability in clinical presentation and management strategies. The exact etiology of IBD remains unclear, but it is widely accepted that a complex interaction among host genetics, gut microbiota, environmental triggers, and immune dysregulation contributes to disease development. Genetic studies have identified multiple susceptibility loci, such as NOD2, ATG16L1, and IL23R, associated with IBD risk. These genes are involved in microbial sensing, autophagy, and regulation of inflammatory responses. Dysbiosis—or an imbalance in the gut microbiome—is a hallmark of IBD, characterized by reduced diversity and an overrepresentation of pro-inflammatory bacteria such as Escherichia coli and a depletion of beneficial commensals like Faecalibacterium prausnitzii. Environmental factors such as diet, antibiotic use, smoking (a risk factor for Crohn’s but protective in UC), and urbanization also influence disease risk and course.

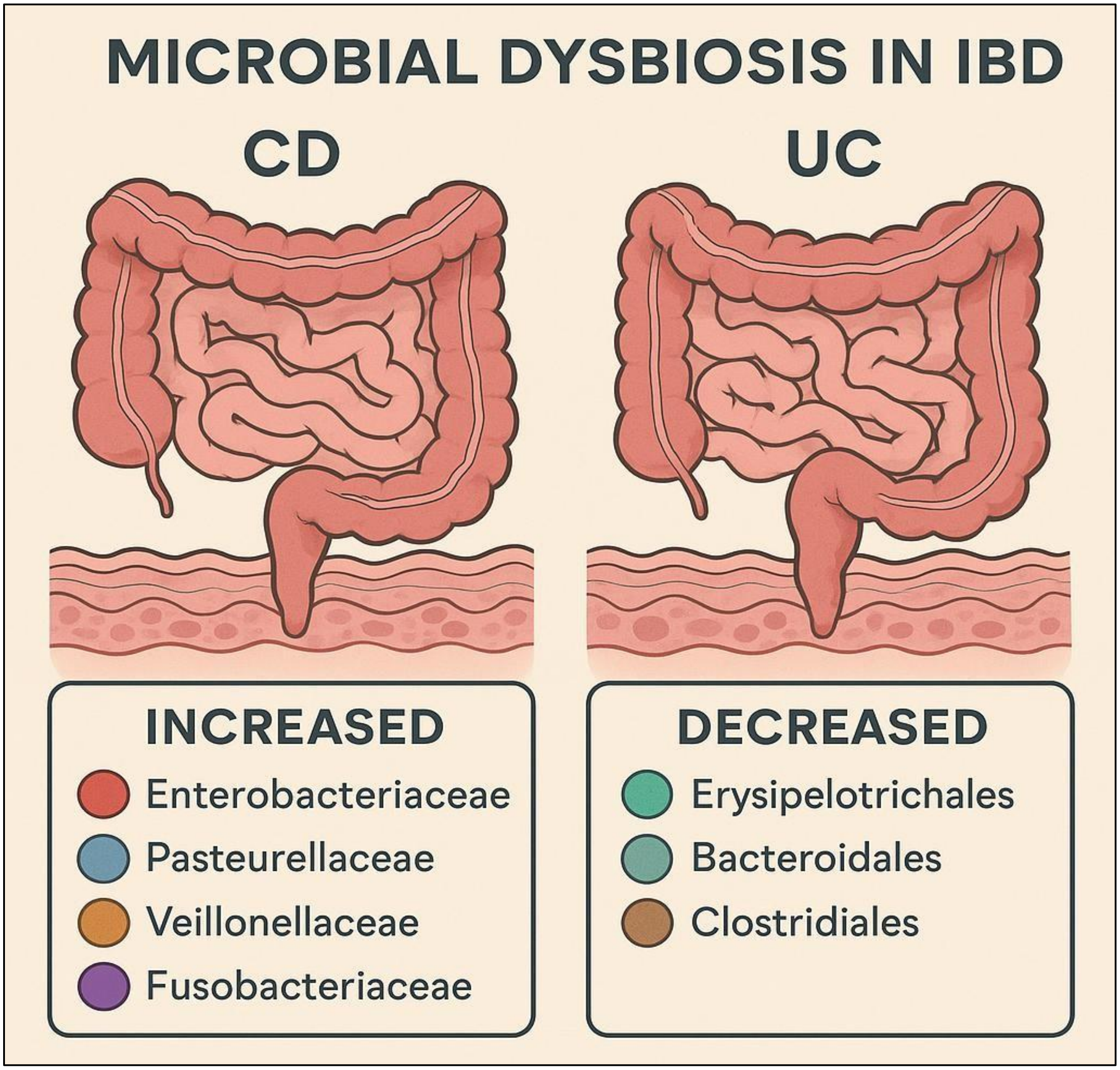

Figure 2. Microbial shifts in IBD: CD and UC gut bacteria compared

4.1.2 Dysbiosis and Associated Illnesses of the GI Tract

In addition to IBD, metabolic disorders, obesity, and type 2 diabetes (T2D), the intestinal microbiota has also been associated with a number of other (chronic) GI-related diseases and conditions, such as colorectal cancer (CRC), celiac disease, and irritable bowel syndrome (IBS). Variations in the microbiota composition of the,different IBS subtypes have been described in comparison to healthy individuals, although the changes are not consistent (96-102). Celiac disease and colorectal cancer (CRC) have also been associated with changes in the composition of the microbiota, with more diversity and richness observed in comparison to control subjects (103-110). To yet, however, no distinct pattern of microbiome changes has been identified in any of these conditions. However, recent studies on celiac disease have shed light on the connection between the illness's development and the host's genetic composition and microbiome. The expression of the leukocyte antigen DQ2 is closely linked to the onset of celiac disease. Children with this haplotype have a distinct microbiota composition than non-HLA DQ2 individuals before a disease shows clinical symptoms. CD4 T- cell responsiveness to dietary gliadin causes celiac disease because some bacterial species can digest gliadin and may reduce its immunological-pathogenicity when eaten (111-117).

4.1.3 Dysbiosis and Non-alcoholic Fatty Liver Disease

Fat buildup in the liver without the presence of alcohol or other secondary causes is the hallmark of Non-Alcoholic Fatty Liver Disease (NAFLD), the hepatic manifestation of the metabolic syndrome (118-125). About one-third of Americans suffer from hepatic steatosis; 10–15% of those who have steatosis also have non-alcoholic steatohepatitis (NASH), and some of these persons develop cirrhosis. The precise etiology of non-alcoholic fatty liver disease is still unclear, but it involves hepatic injury, intestinal dysbiosis-induced inflammation, and intrahepatic lipid accumulation (126-135). Obesity and insulin resistance, which frequently precede non-alcoholic fatty liver disease (NAFLD) and have an impact on metabolism and function, are known to be influenced by the microbiome. The host's food can now provide the "obese metagenome" with greater energy. Gut bacteria further inhibit the generation and release of Fiaf in the small intestine, which raises LPL activity and lipid accumulation in the liver. Additional changes in bacterial metabolism associated with obesity and non-alcoholic fatty liver disease (NAFLD) result in elevated blood ethanol levels (136-145). Ethanol, which is carried by the portal circulation, causes hepatocytes to accumulate triglycerides and can cause inflammation and reactive oxygen species, which can function as a "second hit" to an already obese liver. Choline is another bacterial metabolite linked to NAFLD. It is a phospholipid component of cell membranes and an acetylcholine precursor (146-152). Choline deficiency in the diet has long been associated with liver disease. However, it was only recently demonstrated that a high-fat diet produces a microbiota that reduces phosphatidylcholine and transforms dietary choline into toxic methylamine, simulating a choline-deficient state. Phosphatidylcholine is necessary for VLDL formation and secretion, thus when it is lacking, triglycerides build up in hepatocytes. Additionally, there is a correlation between increased trimethylamine-N-oxide and cardiovascular disease (153-159). In another interesting study, mice were fed a high-fat diet (HFD), and groups of mice were referred to as "responders" based on the development of steatosis, high levels of proinflammatory cytokines, and severe hyperglycemia (160-166). These "responders" or "non-responders" with reduced metabolic responses had their gut microbiota colonized in GF mice. Mice fed a high-fat diet (HFD) and colonized by "responder" gut microbiota developed fatty livers with increased expression of the lipogenesis gene and liver triglycerides (167-175) (see **Figure 3**) (176-185).

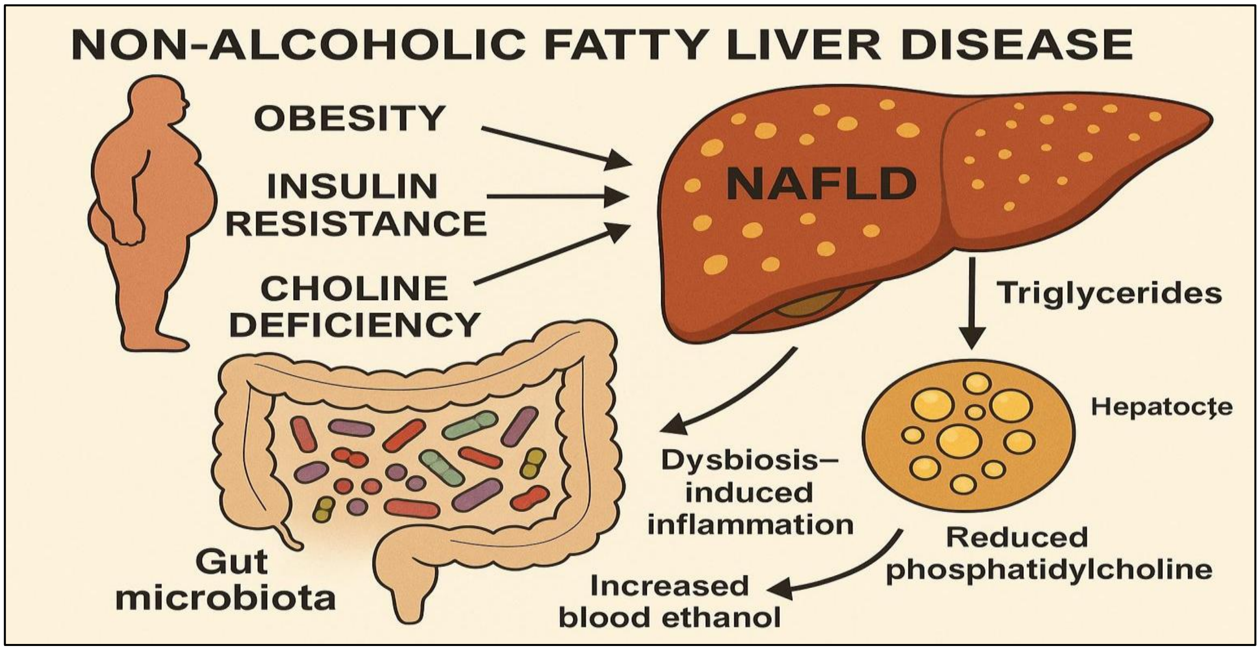

Figure 3. Dysbiosis and non-alcoholic fatty liver disease

FUTURE METHODS: BACTERIOTHERAPY-BASED RESTORATION OF THE INTESTINAL MICROBIOTA

Manipulation can greatly improve, preserve, or restore the microbiota in ill or vulnerable individuals (186-190). Understanding what constitutes a "healthy" microbiome over the course of a person's life is an essential precondition for bacteriotherapy, or treatment based on bacteria. Depending on the individual and the community, this definition may change (191-197). The diversity of microbial genes (microbiome), species, and strains in the GI tract and their function in human development from birth to death need more research thousands of years, probiotic-based treatment methods and the more drastic and unsanitary approach of replacing the entire microbiota through fecal transplantation have been used with differing degrees of success (198-200). This supplement's independent review, "Manipulating the gut microbiota to maintain health and treat disease," delves more into the practicality of these methods. Understanding the molecular mechanisms of action of these and other more advanced approaches using chemically defined bacterial products in the clinic, as well as the unique characteristics of each host that require personalization of approach, will be necessary for bacterial/probiotic therapies to realize their full potential in the treatment and management of human health (200-204).

CONCLUSION

The intricate connection between gut microbiota and gastrointestinal disorders emphasizes the part that microbial dysbiosis plays in the genesis of disease. We have examined the intricate processes by which alterations in the composition and activity of bacteria contribute to the emergence of various gastrointestinal illnesses in this review. Irritable bowel syndrome and inflammatory bowel illnesses are among the conditions for which targeting microbial dysbiosis may be a promising therapeutic strategy. However, despite these amazing developments, many uncertainties remain about the precise mechanisms behind gastrointestinal. disorders brought on by microbial dysbiosis. Future studies should focus on understanding the complex interactions between host genetics, environmental factors, and the gut microbiota in order to provide more effective diagnostic methods and customized treatments. The review concludes by emphasizing the importance of gut microbiota dysbiosis in gastrointestinal illnesses and the need for additional study to fully understand the therapeutic potential of altering the gut microbiota for improved patient outcomes and quality of life.

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