Gut Microbiomes Alterations in Obesity: A Review of Pathophysiological Mechanism and Therapeutic Interventions

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DOI: https://doi.org/10.51584/IJRIAS.2025.101100097

Received: 05 December 2025; Accepted: 12 December 2025; Published: 22 December 2025

ABSTRACT

The bacterial cells in the human gastrointestinal tract (GIT) outweigh the host cells by a factor of ten, and the genes encoded by the bacteria in the GIT outnumber their host genes by more than a hundredfold. The gut microbiome refers to the bacteria associated with the human digestive tract. The human gut microbiome and its role in health and disease have been extensively studied, with findings indicating participation in human metabolism, nutrition, physiology, and immunological function. Imbalances in the normal gut microbiota have been related to gastrointestinal disorders such as inflammatory bowel disease, type 2 diabetes, and atopy. In the first part of this review, we evaluate our evolving knowledge of the composition, pathophysiological mechanism, and Regulation of Appetite, and the ways in which the microbial community is perturbed in dysbiotic disease states; the second part of this review covers the role of interventions that have been shown to modulate and stabilize the gut microbiota, as well as restore it to its healthy composition from the dysbiotic states seen in IBS, IBD, obesity, type 2 diabetes.

Keywords: Gut Microbiomes, Inflammatory bowel disease (IBD), irritable bowel syndrome (IBS), Immunological function

INTRODUCTION

Obesity is a worldwide public health problem that continues to rise and accounts for over 60% of deaths related to high body mass index (BMI) [1]. Obesity is considered a complex and multifactorial condition. The association and causative role played by gut bacteria in obesity represent one of the most important findings in the field. The gut microbiota (GM) is intertwined with host physiology and pathophysiology. GM has recently been recognized as an important factor in the development of metabolic diseases. Changes in the composition of GM may result in a change in the relationship between the bacteria and the host, which can lead to an inflammatory process and metabolic disorders seen in obesity [2]. The human gut contains approximately 10^14 microorganisms, predominantly composed of Firmicutes and Bacteroidetes species. Different bacterial species occupy distinct sections of the intestine; for instance, Firmicutes often predominate at the top of the gut crypto-villous unit while Proteobacteria predominates at the bottom. The functional consistency of each bacterial genus is quite high and is not affected by the host's age, sex, BMI, or nationality [3]. Our understanding of the inter-relationships between GM and the development of obesity remains descriptive, with significant gaps between clinical and experimental expertise. This review provides a brief introduction to GM and its mode of action in connection to obesity, influential factors on microbiota, including dysbiosis, and therapies suggested for obesity with respect to GM [1].

Table 1. Major microbes associated with obesity and their role.

S No.	Microorganism	Role in Obesity	Effect on host	Reference
1.	*Escherichia coli*	Escherichia coli is a very normal bacteria found in the human gut but some studies show that the increased in level of some E.	Its effects are increased fat storage, decreased insulin	[35]

		coli strains are the reason behind the obesity and metabolic diseases. It is a Gram-negative bacterium whose outer membrane contains lipopolysaccharide (LPS). E. coli contributes to obesity by increasing gut permeability, allowing LPS to enter bloodstream known as metabolic endotoxemia. Circulating LPS binds to TLR4 receptors on immune cells. This activates NF-Kb-mediated inflammatory pathway, also causes chronic low-grade inflammation. Higher levels of LPS disrupt normal glucose utilization, which promotes adipogenesis (fat cell formation).	sensitivity, promotes weight gain, higher systemic inflammation, gut barrier disruption.	
2.	*Firmicutes spp.*	Firmicutes inhibit specialised (carbohydrate-degrading) enzymes that break down indigestible polysaccharides into short chain fatty acids (SCFAs) such as Acetate, Butyrate, Propionate. They also induce fat synthesis (lipogenesis) as these SCFAs excite liver enzymes like Acetyl-CoA carboxylase (ACC) and Fatty acid synthase (FAS) to participate in the fat formation.	It enhances fat storage in adipose tissue and causes insulin resistance and low-grade chronic inflammation.	[36]
3.	*Bacteroidetes spp.*	Bacteroidetes are normally associated with healthy or lean body weight. It supports maintaining balanced energy extraction. In obesity levels of Bacteroidetes usually reduced, and firmicutes are increased. Less Bacteroidetes means less efficient carbohydrates breakdown, altered SCFA production. More energy extraction from food means more calorie absorption.	Reduced metabolic flexibility and increased fat accumulation	[36]
4.	*Lachnoclostridium spp.*	Promotes inflammatory cytokines such as IL-6, and endotoxemia in obesity	Systemic low-grade inflammation and insulin resistance	[37]
5.	*Akkermansia muciniphila*	Regulates glucose metabolism; depletion increases adiposity	Gut barrier function and insulin resistance	[38]

Gut Microbiomes Composition In Health And Obesity

The human GM consists of approximately 100 trillion cells, which is 10 times the number of human cells. The density and composition of the GM increase from the upper to the lower intestines, with the highest diversity found in the colon. The composition of these microbial communities varies across different body sites and is influenced by various factors including host genetics, age, sex, weight, diet, immune system, oxygen levels, pH, bile acids, gastrointestinal transit time, mucus production, disease states, medications, probiotics, certain surgeries like gastric bypass, and environmental factors .

The colon, with its high transit time, favourable pH, low cell turnover, and redox potential, is particularly conducive to bacterial proliferation. The healthy human GM comprises over 1000 bacterial species, predominantly from 6 phyla: Firmicutes, Bacteroidetes, Actinobacteria, Proteobacteria, Fusobacteria, and Verrucomicrobia.

Colonization begins shortly after birth and progresses into adulthood. The GM composition varies in early life, influenced by delivery mode, feeding method, and diet with differences noted between vaginally born and caesarean-born infants. The maternal microbiome during pregnancy and whether the infant is breastfed or formula-fed also play crucial roles. By the age of 3, a more adult-like GM pattern is established. A diverse GM is rich in number and variety. The GM undergoes significant changes and transitions during childhood before reaching a relatively stable adult-like state [4].

Case Study

A Japanese study investigated age-related changes in GM composition in 367 healthy individuals aged 0 to 104 years by analysing the faecal samples. Significant GM transformations occurred during childhood (<20 years) as it matured and again after 70 years of age, shifting to an "elderly type." Actinobacteria abundance markedly decreased after weaning and continued to decline with age. Firmicutes became dominant postweaning but were less abundant in children under 4 years compared to older subjects. Bacteroidetes and Proteobacteria increased after age 70 years, opposite to the Firmicutes trend. Distinct co abundance groups (CAGs) dominated at different life stages: Bacteroides, Eubacterium, and Clostridiaceae CAGs (elderly associated); Enterobacteriaceae CAGs (infant and elderly-associated); Bifidobacterium CAGs (infant/child-associated); Lachnospiraceae CAGs (adult-associated); and Megamonas and Peptoniphilus CAGs (relatively enhanced in the elderly). Sequential changes occurred in Bacteroides, Lachnospiraceae, and Bifidobacterium CAGs in the GM during childhood and adolescence [5]. These changes likely reflect the interplay between the developing GM and host physiological factors across the lifespan.

Pathophysiological Mechanism Linking Gut Microbiota In Obesity

The term pathophysiological mechanism refers to the biological processes and changes that occur in the body during the development of a disease or abnormal condition. Pathophysiological mechanisms linking gut microbiota and obesity refer to the biological processes and pathways through which alterations in the gut microbiota (the community of microorganisms in the digestive tract) contribute to the development, progression, and maintenance of obesity. These mechanisms explain how gut microbiota influence host metabolism, fat storage, energy balance, and inflammation, ultimately affecting body weight and health.

Enhanced Energy Harvesting and Modulation of Lipid Metabolism

Certain gut microbiota possesses the ability to extract additional calories from indigestible dietary polysaccharides, converting them into absorbable short-chain fatty acids (SCFAs). This increased energy extraction can contribute to a positive energy balance and fat accumulation, promoting obesity. Gut microbiota influence lipid metabolism by producing metabolites that affect fat storage and lipid profiles. Alterations in microbiota composition can lead to increased fat deposition and changes in lipid metabolism, contributing to obesity [6].

Influence on Insulin Resistance and Impact on Bile Acid Metabolism

Insulin is a hormone which is composed by 51 amino acids and plays a crucial role in glucose homeostasis, cell growth and metabolism [33]. Insulin was believed to be produced by β cells of the pancreas; but recent evidence has shown that low concentration is also found in neurons of the central nervous system (CNS) [34]. Altered gut microbiota can affect glucose metabolism and insulin sensitivity, contributing to the development of insulin resistance, a common feature in obesity. Gut microbiotas play a role in the metabolism of bile acids, which are involved in dietary fat digestion and regulation of lipid and glucose metabolism. Alterations in bile acid metabolism by gut microbiota can influence energy balance and contribute to obesity.[6].

Regulation of Appetite and Satiety

The gut microbiota interacts with the host's enteroendocrine system, influencing the secretion of hormones that regulate appetite and satiety. Ghrelin is the hunger hormone which is released from stomach and makes you feel hungry and Leptin is the satiety hormone which is released from adipose tissue and functions to signal brain Wheater you are full or not. Disruptions in this interaction can lead to increased food intake and weight gain [7]. The gut microbiota affects food intake by influencing brain function through various mechanisms- for example, by helping produce neuromodulators like serotonin, which is essential for controlling gastrointestinal activity [30]. Additionally, lactate generated by Lactobacillus and Bifidobacterium serves as a fuel or neurons and can extend the feeling of fullness after eating [31].

Induction of low-grade inflammation

People with obesity often have a constant, mild form of inflammation in their bodies, which causes changes in how certain inflammatory chemicals are made and released [32]. Dysbiosis, or an imbalance in gut microbiota, can increase intestinal permeability, allowing bacterial endotoxins like lipopolysaccharides (LPS) to enter the bloodstream. This triggers systemic low-grade inflammation, which is associated with the development of obesity and metabolic disorders [31].

Factors Influencing Gut Microbiota In Obesity

Understanding the factors that influence gut microbiota in obesity is crucial for developing effective interventions. Key factors include:

Diet and lifestyle factors play a critical role in shaping gut microbiota and influencing obesity. Consumption of high-fat diets has been shown to alter gut microbiota composition by increasing Firmicutes and decreasing Bacteroidetes, a microbial shift commonly associated with obesity. Similarly, diets low in dietary fiber reduces microbial diversity, which negatively affects gut health and contributes to obesity. On the other hand, regular physical activity supports a diverse gut microbiota, which is linked to a reduced risk of obesity. Additionally, medications such as antibiotics can disrupt the balance of gut microbiota, potentially leading to weight gain and increased obesity risk [8].

Host genetics play a crucial role in shaping the composition and diversity of gut microbiota, which can directly influence an individual's risk of developing obesity. Genetic predisposition affects the abundance of specific microbial taxa, potentially altering metabolic functions such as energy extraction, fat storage, and inflammation. These genetic influences can create a gut environment more prone to supporting obesity-related microbial profiles. Understanding the interaction between host genes and gut microbes is essential for identifying individuals at greater risk for obesity and developing personalized interventions based on genetic makeup and microbiome composition [9].

These factors interact in complex ways to influence gut microbiota composition and function, thereby affecting obesity risk. Understanding these interactions is essential for developing targeted strategies to modulate the gut microbiome in the prevention and treatment of obesity.

Diseases Caused By Alteration In Gut Microbiomes

Alterations in gut microbiota, commonly referred to as gut dysbiosis, have been extensively linked to various gastrointestinal and metabolic diseases. Below is an overview of these associations, supported by research finding.

Gastrointestinal Diseases

Inflammatory Bowel Disease (IBD) and Irritable Bowel Syndrome (IBS)

Inflammatory Bowel Disease (IBD), encompassing Crohn’s disease and ulcerative colitis, is characterized by chronic inflammation of the gastrointestinal tract. IBD includes formation of patchy inflammation or ulcer mainly at the colon region of the gastrointestinal tract. Dysbiosis in IBD patients often includes reduced microbial diversity and a decrease in beneficial bacteria such as Faecalibacterium prausnitzii, leading to impaired gut barrier function and heightened immune responses. IBS is a functional gastrointestinal disorder marked by abdominal pain and altered bowel habits. Altered gut microbiota composition can disrupt fermentation processes, leading to gas production and bloating.[10].

Colorectal Cancer

Colorectal cancer is a malignant tumour that develops in the colon or rectum. It usually begins as a non-cancerous polyp on the inner lining of the intestine, which slowly grows and can turn into cancer. It can also be influenced by genetic mutation like: Adenomatous polyposis coli (APC) gene mutation, KRAS gene mutation and later on P53 gene mutation. Dysbiosis may promote colorectal cancer through chronic inflammation and the production of carcinogenic metabolites by certain gut microbes.[11]. Colorectal cancer (CRC) is one of the major causes of deaths from cancer in humans, accounting for 9.4% of deaths worldwide in 2020 [18]. Gut dusbiosis refers to the compositional and functional changes which are caused by imbalance between symbiotic and opportunistic microbes [19].Dysbiosis is categorized into three types: (i) loss of beneficial microbes, (ii) expansion of pathogenic microbes, and (iii) loss of microbial diversity [20].An imbalance in the gut microbiota plays a role in various diseases, including diabetes [21][22],obesity [23][24] , neurogenerative diseases[25] and cancer [26].

Metabolic Diseases

There are majorly four metabolic diseases influenced by dysbiosis in gut microbiomes. These four diseases are explained below:

Obesity

Obesity is the excessive accumulation of body fat due to imbalance between energy intake and expenditure. In this condition the firmicute's colony is increased and Bacteroidetes are decreased. The microbe which is responsible for energy harvesting is Firmicutes and its increased number extract even the last bit of energy from food. This extraction of extra energy from food causes fat deposition in body [12].

Insulin Resistance

Insulin resistance means body's cells do not respond properly to insulin, so glucose stays in the blood. This causes increase in gut permeability and allows bacterial toxins (LPS) to came into circulation, which causes chronic low-grade inflammation. Inflammatory cytokines (IL-6) blocks insulin signalling in cells. As a result, cells stop absorbing glucose properly [13].

Type 2 diabetes mellitus

It is a metabolic disorder in which blood glucose level remains high which is caused by insulin resistance, decrease in insulin production or reduction in the production of SCFA. SCFA helps in insulin sensitivity by allowing the cells to use the circulating glucose properly [13].

Non-Alcoholic Fatty Liver Disease (NAFLD)

It is the accumulation of fat in the liver which is not influenced by alcohol. This disease is caused by increase in gut permeability, due to this increase in gut permeability some toxins leak out and reaches liver and activates liver's immune cells which causes inflammation. This dysbiosis alters bile acid metabolism and increases fat deposition in the liver [10].

Therapeutic Interventions Targeting The Gut Microbes

Microbial manipulation may be employed to prevent or treat weight gain and associated comorbidities. Approaches to this include use of probiotics, prebiotics, synbiotics, fecal microbiota transplant (FMT), and other interventions. The success of these therapies largely depends on factors such as the nature of resident microbiota composition and structure and understanding of the dynamic alterations that occur over time.

Prebiotics and Probiotics and Fecal Microbiota Transplantation

Prebiotics have been studied widely for their use in treating obesity. Numerous clinical studies have shown the benefits of using prebiotics in obesity by improving appetite control and reduction of body fat. Prebiotics are a class of nutritional compounds categorized together, not necessarily by structural affinity, but by the potential to promote the growth and/or activity of specific beneficial bacteria (probiotics) in GM [2].

FMT refers to altering the host's gut microbiome to provide a therapeutic effect. It involves the introduction of microbiota from a healthy donor's feces to the morbid individual's GIT (Gastrointestinal Tract) and has been used in metabolic syndrome (MS) and diabetes [2].

Probiotics are living microbes that, when consumed in sufficient quantities, offer several health benefits.[27]. Probiotics help reshape the gut microbiota by reducing harmful microbes and promoting the growth of beneficial ones [28]. Probiotics offer a wide range of health benefits, such as boosting immune regulation, lowering colitis and blood cholesterol, suppressing harmful bacteria, and helping prevent colorectal cancer (CRC) [29].

Bacterial Consortium Therapy

An alternative to FMT would be a well-defined microbiota that is rebalanced, comprising gut bacteria, or bacterial consortium therapy (BCT) [15]. BCT involves the use of defined drug compositions produced from clonally isolated bacteria that can trigger targeted immune responses. Specific intestinal ecosystem modulation could be performed with BCT. A recent study showed complete recovery and effects comparable to those of FMT with BCT as a substitute [16]. Bacterial consortiums are defined accurately and can be prepared based on different levels or types of dysbiosis. Patient safety in this regard is improved as the bacterial combination can be controlled for pathogenic microbes. In this context, BCT could be a safer alternative to FMT to modulate intestinal dysbiosis [17].

Phage Therapy

Bacteria-specific viruses (phages) have a great influence on the bacterial population of microbes. They have good therapeutic potential and can be used as an alternative to antibiotics or to modulate the composition of the gut flora. Given the presence of our microbial ecosystem, the risks of phage therapy do not appear to be high. Phage suspensions can be prepared for both local (introduced directly in the gut) and systemic therapy, keeping in mind the amplification of phages after administration. The kinetics of amplification usually depends on the concentration of susceptible bacteria, the immune responses of the host, etc. Due to these variables, the dosing and timing of administration of phage therapy have been problematic.[2].

DISCUSSION

The gut microbiome plays a pivotal role in the development and progression of obesity through various pathophysiological mechanisms. Obese people often have changes in their microbial composition, such as a higher proportion of Firmicutes to Bacteroidetes, which are associated with improved energy harvest, fat storage, and low-grade chronic inflammation. The integrity of the intestinal barrier is compromised by these alterations, which raise endotoxin levels and fuel metabolic dysfunction. Additionally, sedentary lifestyles, antibiotic usage, genetic predisposition, high-fat, low-fiber diets, and other factors aggravate microbiome imbalance. Promising methods for managing obesity include therapeutic therapies that target the gut flora. Strategies like dietary changes (such eating more fiber), regular exercise, probiotics, prebiotics, and even fecal microbiota transplantation (FMT) have demonstrated promise in re-establishing microbial balance and enhancing metabolic health. Developing efficient, individualized treatments for obesity requires an understanding of the intricate interactions between host physiology and gut bacteria.

CONCLUSION

In conclusion, the microorganisms in our digestive system are intimately linked to our body's regulation of weight and reaction to food. When this microbial ecosystem is disrupted, it can lead to weight increase and various health issues. However, with advancing research, we are discovering that lifestyle modifications and innovative therapies focusing on enhancing gut health may serve as essential strategies in combating obesity. Customizing these methods to fit everyone's distinct biology might make obesity treatment more efficient and enduring in the future.

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