

Nutritional Correlation between Gut Microbiota and Obesity

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Abstract:

Obesity is a multifactorial chronic metabolic disease that has become a global public health crisis, with more than 1.3 billion individuals affected worldwide. Emerging evidence highlights the gut microbiota as a critical factor linking nutrition and obesity. Alterations in microbial diversity and composition—particularly an increased Firmicutes/Bacteroidetes ratio—are closely associated with enhanced energy harvest, impaired intestinal barrier function, systemic low-grade inflammation, and metabolic disorders. These microbial imbalances form a vicious cycle that exacerbates obesity and related comorbidities such as type 2 diabetes, cardiovascular disease, and nonalcoholic fatty liver disease. Nutritional strategies targeting the gut microbiome have demonstrated promising therapeutic potential. Dietary interventions rich in fiber, polyphenols, and prebiotics can remodel microbial communities, increase short-chain fatty acid production, and improve metabolic regulation. Probiotic and synbiotic supplementation, particularly multi-strain formulations, have shown benefits in modulating microbial homeostasis and reducing visceral fat. Furthermore, fecal microbiota transplantation (FMT) has emerged as an innovative approach, improving insulin sensitivity and inflammatory markers, though its long-term efficacy and safety remain uncertain. Advances in metagenomics, metabolomics, and precision nutrition are expected to refine personalized microbiome-based therapies. Overall, understanding the bidirectional interaction between gut microbiota and obesity provides a scientific basis for developing effective nutritional interventions, which may contribute to comprehensive strategies for obesity prevention and treatment.

Keywords: gut microbiota, obesity, nutritional intervention, probiotics, prebiotics, short-chain fatty acids, fecal microbiota transplantation

1 Introduction

Obesity is a complex, chronic metabolic disease driven by multiple factors, primarily characterized by abnormal or excessive accumulation of adipose tissue. According to the World Health Organization (WHO), an adult with a body mass index (BMI) ≥ 30 kg/m² is considered obese, while a BMI between 25 and 29.9 kg/m² is defined as overweight. In recent years, with changes in lifestyle, a Westernized diet, and decreased physical activity, the global prevalence of obesity has increased significantly, becoming one of the most serious public health challenges of the 21st century. According to WHO statistics from 2024, the number of obese people worldwide has exceeded 1.3 billion, including a large number of children and adolescents, and the incidence of obesity is trending at younger ages and at a higher rate.

Obesity not only affects an individual's body shape and quality of life but is also a significant independent risk factor for a variety of chronic diseases, including type 2 diabetes, cardiovascular disease, nonalcoholic fatty liver disease, hypertension, metabolic syndrome, and certain malignancies (such as colon and breast cancer). Furthermore, obesity can lead to a range of metabolic disorders, including systemic chronic inflammation, insulin resistance, impaired intestinal barrier function, and endocrine abnormalities, profoundly impacting physical health. Therefore, in-depth exploration of the etiological mechanisms of obesity and the development of effective prevention and control strategies are of great clinical and public health significance.

In addition to the traditional factors of concern such as genetic susceptibility, excess energy intake, and lack of physical activity, the role of "gut flora" in the development of obesity has received increasing attention in recent years. There are more than 100 trillion microorganisms coexisting in the human intestine, mainly including bacteria, fungi, viruses, etc., and the vast majority are concentrated in the colon. These microbial communities not only participate in the host's nutrient absorption and energy metabolism, but also play a key role in immune regulation, intestinal barrier maintenance, hormone synthesis and inflammation control. More and more studies have found that the structure and function of the intestinal flora of obese individuals are significantly abnormal. The most common change is "an increase in the proportion of Firmicutes and Bacteroidetes", suggesting that the intestinal flora may be involved in the development of obesity by affecting energy acquisition efficiency, fat storage capacity and metabolic homeostasis.

Gut microbial metabolites, particularly short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate, not only provide energy for intestinal epithelial cells but also influence host lipid and glucose metabolism through

mechanisms such as activation of G protein-coupled receptors and modulation of inflammatory and metabolic signaling pathways. Therefore, the balance of the intestinal microbiome is directly related to energy metabolism and weight regulation. Diet, as the most direct and modifiable external factor influencing the gut microbiome, plays a crucial role in maintaining microbial homeostasis and preventing obesity. Studies have shown that a high-fat, high-sugar diet can easily lead to microbial imbalance, promoting fat deposition and inflammatory responses. However, a diet rich in dietary fiber, prebiotics, probiotics, and plant polyphenols, such as the Mediterranean diet or a plant-based diet, can effectively improve microbiome structure, enhance metabolic function, and reduce the risk of obesity.

With the deepening of research on the relationship between intestinal microecology and nutritional metabolism, "prevention and treatment strategies based on microecological intervention" have gradually shown their potential. New methods including probiotic/prebiotic supplementation, individualized dietary nutrition regulation and fecal microbiota transplantation (FMT) have shown certain results in preclinical and clinical studies.

2. Two aspects of the nutritional correlation between intestinal flora and obesity

2.1 The mechanism of obesity's impact on intestinal flora:

To explore how obesity changes the diversity and function of intestinal microecology through metabolic disorders, inflammatory response, host genetic background and other factors, thereby forming a vicious cycle of obesity-microbial imbalance.

2.2 Research progress and application prospects of intervening in obesity by regulating intestinal flora:

Evaluate the effects and mechanisms of regulating the composition and function of the intestinal flora through dietary intervention, probiotics/prebiotics, fecal microbiota transplantation, etc. on weight control and metabolic improvement, and provide a scientific basis for nutritional intervention strategies based on the intestinal flora.

3. The mechanism of obesity's impact on intestinal flora

In recent years, a wealth of research evidence has demonstrated that obesity is not only the result of an imbalance

between energy intake and expenditure but is also closely related to the composition and function of the intestinal microbiome. The intestinal microbiome of obese individuals often exhibits a specific imbalance, manifested by decreased microbial diversity, a reduced proportion of beneficial bacteria, an increase in harmful or opportunistic pathogens, and abnormal metabolite profiles. These changes are not only a concomitant of obesity but may also play a pathogenic role in the development and maintenance of obesity.

3.1 Changes in the structural characteristics of the intestinal flora in obese individuals

Firmicutes and Bacteroidetes are the two most dominant groups in the gut microbiome. Numerous studies in animals and humans have found that obese individuals often exhibit an increased relative abundance of Firmicutes and a decreased abundance of Bacteroidetes, known as an “increased Firmicutes/Bacteroidetes ratio.” This shift in microbial composition is closely associated with improved energy efficiency. A review by Gérard (2016) noted that certain genera within the Firmicutes phylum, such as *Clostridium* and *Eubacterium*, possess enhanced ability to ferment complex carbohydrates and produce short-chain fatty acids (SCFAs), thereby improving the host’s efficiency in utilizing dietary energy and providing a “microecological foundation” for obesity [Gérard, 2016].

In addition, obesity is closely associated with reduced gut microbial diversity. A low-diversity microecological environment often means a single metabolic function, a decreased ability of the host to interact with the microbiota, and an increased space for the expansion of susceptible pathogens, which leads to a weakening of the host’s intestinal barrier function, triggering systemic low-grade inflammation and providing a breeding ground for metabolic disorders.

3.2 The interaction mechanism between obesity-related metabolic disorders and microbiota

Obesity is often accompanied by pathological processes such as insulin resistance, impaired lipid metabolism, increased oxidative stress, and elevated inflammation. These factors can affect the homeostasis of the intestinal microbiome through various pathways.

First, excessive secretion of pro-inflammatory cytokines (such as $\text{TNF-}\alpha$ and IL-6) by adipose tissue can alter the local immune microenvironment of the intestine, affecting the interaction between intestinal epithelial cells and microbes, leading to a decrease in beneficial bacteria and an expansion of harmful bacteria. Second, metabolic disturbances can also alter the synthesis and excretion of bile acids. As selective regulators of the microbiome, changes in bile acid composition can significantly influence the

structure of the intestinal microbiome. For example, reduced production of secondary bile acids can weaken the inhibition of opportunistic pathogens, leading to an imbalance in the microbiome.

Furthermore, obesity may further affect microbial colonization and survival through altered host gene expression, decreased intestinal motility, and reduced mucus secretion, exacerbating microbial imbalance.

3.3 Damage to the intestinal barrier and increased endotoxin permeability

Obesity often impairs intestinal barrier function, leading to downregulation of tight junction proteins (such as occludin and claudin) and increased intestinal permeability. This allows bacterial metabolites, particularly endotoxins such as lipopolysaccharide (LPS), to more easily penetrate the intestinal wall and enter the circulatory system, inducing systemic chronic low-grade inflammation (metabolic endotoxemia). This process is believed to be a key mechanism underlying obesity-related chronic diseases such as insulin resistance, fatty liver disease, and atherosclerosis [Gérard, 2016].

Animal experiments have shown that mice induced by a high-fat diet exhibit significantly elevated plasma LPS levels, accompanied by weight gain, fat accumulation, and upregulation of inflammatory cytokines. However, antibiotics that partially eliminate intestinal microbiota or enhance intestinal barrier function can significantly alleviate these pathological manifestations. This finding further supports the causal link between intestinal dysbiosis and obesity.

4. Research progress and application prospects of intervening in obesity by regulating intestinal flora

With a deeper understanding of the role of the gut microbiome in the pathogenesis of obesity, a growing number of studies are focusing on preventing and intervening in obesity by regulating the gut microbiome. Nutritional intervention, as a safe, feasible, and sustainable approach, has shown great potential in modulating gut microbiome structure, restoring microbial homeostasis, and improving metabolic function. Current mainstream interventions include dietary optimization, probiotic and prebiotic supplementation, synbiotic strategies, and microecological therapies such as fecal microbiota transplantation (FMT).

4.1 Dietary intervention and intestinal flora remodeling

Diet is the most direct and important external factor influencing the structure of the gut microbiome. Research has

shown that different dietary compositions can rapidly alter the composition and functional characteristics of the microbiome in the short term, profoundly impacting energy metabolism and fat accumulation.

High-fat and high-sugar diets have been shown to cause gut microbial imbalance, reducing the proportion of beneficial bacteria (such as *Bifidobacteria* and *Lactobacilli*), increasing the abundance of opportunistic pathogens and pro-inflammatory bacteria, and reducing microbial diversity, thereby promoting fat accumulation, inflammation, and insulin resistance. Diets rich in nutrients such as dietary fiber, polyunsaturated fatty acids, and plant polyphenols (such as the Mediterranean diet, vegetarian diet, and Nordic diet) are believed to be beneficial for maintaining microbial balance, increasing the proportion of butyrate-producing Firmicutes, promoting short-chain fatty acid production, and regulating intestinal pH, immune response, and energy metabolism, ultimately achieving fat loss and weight control.

In addition, the intake of prebiotic ingredients (such as inulin, oligofructose, and resistant starch) can help selectively stimulate the growth of beneficial bacteria, improve intestinal barrier function, and reduce metabolic inflammation.

4.2 Intervention potential of probiotics, prebiotics and synbiotics

Probiotics are active microorganisms that, when consumed in sufficient quantities, benefit host health. Common strains include *Bifidobacterium* and *Lactobacillus*. They have positive effects on weight control and obesity-related metabolic disorders by modulating microbial composition, inhibiting the colonization of harmful bacteria, enhancing mucosal immunity, improving intestinal barrier function, and regulating metabolic pathways.

In their review, Sheykhsaran et al. (2023) noted that multiple animal studies and some clinical studies support the potential use of specific probiotic strains in weight management. For example, 8–12 weeks of *Lactobacillus gasseri* SBT2055 significantly reduced abdominal visceral fat area, BMI, and waist circumference [Sheykhsaran et al., 2023]. Furthermore, some “multi-strain combined intervention” programs have shown greater lipid-lowering effects than single-strain interventions, possibly due to their broader metabolic regulatory potential and synergistic effects with the microbiome.

Prebiotics are substrates that can be selectively utilized by probiotics, indirectly exerting regulatory effects by promoting the growth of beneficial bacteria. Synbiotics are combined preparations of probiotics and prebiotics, designed to enhance the survival and colonization of probiotics. Their effectiveness in weight management has also garnered widespread attention.

Although numerous experimental studies have yielded positive results, the efficacy of probiotics in actual clinical practice is influenced by factors such as strain specificity, dosage, duration of administration, and individual differences in the host. Further high-quality, multicenter, long-term randomized controlled trials (RCTs) are needed to further validate their efficacy and safety.

4.3 Fecal microbiota transplantation (FMT) as a cutting-edge intervention strategy

Fecal microbiota transplantation (FMT), which involves transplanting fecal microbiota from healthy donors into the recipient's intestine to reconstitute the recipient's intestinal microbiome, has been successful in treating refractory *Clostridium difficile* infection and is gradually expanding into metabolic diseases.

Studies of obesity-related FMT have shown that transplanting gut microbiota from lean donors into patients with obesity or metabolic syndrome can improve insulin sensitivity, metabolic parameters, and some inflammatory markers. However, its long-term efficacy in weight management remains unclear.

Sheykhsaran et al. noted in their publication that while FMT shows potential as a microbial therapy, its application still faces numerous challenges, including inconsistent donor selection criteria, poor colonization stability, long-term fluctuations in efficacy, and ethical and legal issues [Sheykhsaran et al., 2023]. Future development of a “precision microbiome transplantation” strategy, combining personalized microbiome testing with functional analysis to target functional microbiota, may be necessary to improve the efficiency and safety of FMT in obesity intervention.

5. Conclusion

In summary, the gut microbiome plays a crucial role in the development and progression of obesity. Its composition, structure, and metabolites are closely linked to numerous physiological processes, including host energy metabolism, inflammatory responses, and intestinal barrier function. Obesity not only leads to an imbalance in the gut microbiome but also exacerbates intestinal microecological disturbances through various mechanisms, creating a vicious cycle of metabolic abnormalities and dysbiosis.

Gut microbiome-based nutritional intervention strategies, such as dietary manipulation, probiotic/prebiotic supplementation, synbiotic combinations, and fecal microbiota transplantation, are becoming increasingly important in the prevention and treatment of obesity. While numerous experimental studies and preliminary clinical trials have demonstrated the positive effects of these strategies in modulating the microbiome and improving metabolism,

their long-term efficacy, personalized application, and safety still require further verification in larger, high-quality population studies.

In the future, the advancement of cutting-edge technologies such as metagenomics, metabolomics, and artificial intelligence will help to further elucidate the causal relationship and molecular mechanisms between the gut microbiome and obesity, thereby promoting personalized microbiome-based precision nutritional interventions and providing more effective scientific evidence and clinical tools for the comprehensive prevention and control of obesity.

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