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# INTERACTION BETWEEN OBESITY AND GUT MICROBIOTA: NEW APPROACHES FOR DIAGNOSIS AND THERAPY

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**Abstract**

Obesity is a serious health problem and is associated with many physiological and mental disorders, including diabetes, stroke, and depression. Gut microbiota is considered important in the development of metabolic diseases and obesity, as well as in maintaining energy homeostasis and host immunity. In recent years, scientific research has increasingly provided evidence linking obesity to the gut microbiota. Regulation of the gut microbiota is being considered as a new method of obesity treatment. However, the complex interactions among genetics, environment, gut microbiota, and obesity have not been fully elucidated

**Keywords**

Metabolism, obesity, prebiotic, probiotic, gut microbiota, immune system, treatment, homeostasis, diagnosis, microbiota.

Introduction

The World Health Organization (WHO) defines obesity as having a body mass index (BMI) greater than 30. According to recent studies, approximately one-third of the world's population are overweight, and about 10% are classified as obese. Projections suggest that by 2030, the number of obese individuals worldwide may reach 1.12 billion. Recent research indicates that an imbalance in the gut microbiota may be one of the contributing factors to the development of obesity.

The gut microbiota is located in the human intestinal tract and consists of a large number of microorganisms, including bacteria, fungi, viruses, archaea, protozoa, and others. The total weight of the gut microbiota is 1–2 kg, and the number of genes it contains is more than 100 times greater than that of the human genome. In a healthy state, the gut microbiota coexists harmoniously with the human body and participates in the regulation of many physiological functions. These mainly include the digestion and absorption of nutrients, protection against harmful microbes, and maintenance of immune homeostasis. Conversely, an imbalance in the gut microbiota may lead to negative consequences. Altered gut

microbiota may participate in the pathogenesis of obesity by disrupting energy homeostasis, altering lipid synthesis and storage, regulating central appetite and feeding behavior, and promoting chronic low-grade inflammation.

Currently, there are several approaches to obesity treatment, including the adoption of a healthy lifestyle, weight-reducing drugs, and bariatric surgery. However, due to the difficulty of maintaining long-term dietary control and regular physical activity, as well as the undesirable effects of drugs and surgery, it is necessary to explore new therapeutic strategies.

Bacteria-specific viruses affect 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. Considering the presence of our microbial ecosystem, the risks of phage therapy are not high. Phage suspensions can be prepared for both local and systemic therapy, and it is necessary to determine the replication of phages after administration.The kinetics of replication depend on the concentration of susceptible bacteria, the immune response, and other factors. Due to these variables, the dosage and timing of phage therapy are considered problematic.

The metabolic activity of the gut microbiota is aimed at extracting calories from ingested food, storing these calories in adipose tissue for later use, and providing energy and nutrients for the growth and proliferation of microbes. Individual differences in energy recovery may represent a physiological explanation for why some obese patients appear not to overeat but still gain weight. In this context, it has been determined that the gut microbiota of a person has a certain metabolic efficiency, and that some characteristics of its composition may predispose to obesity.

Prebiotics are chemical substances, nondigestible food components, that stimulate the growth or activity of gut bacteria.By reducing lipopolysaccharide production in the gut microbiota, prebiotics are capable of managing obesity. In a double-blind, placebo-controlled clinical study, administration of 8 g/day oligofructose-enriched inulin for 16 weeks to overweight children significantly increased the amount of Bifidobacterium and slowed body weight gain compared with the placebo group.

Probiotics are non-pathogenic living microorganisms that exert a direct or indirect effect on the gut microbiota. They can also manage obesity by influencing the gut microbiota and reducing lipopolysaccharide production. In a double-blind, placebo-controlled clinical study, overweight adults who were given fermented

milk containing Lactobacillus gasseri for 12 weeks showed a significant reduction in body weight.

Bariatric surgery affects body weight by altering the gut microbiota. In this process, after Roux-en-Y gastric bypass surgery in obese patients, the Firmicutes/Bacteroidetes phyla ratio decreases, and an increase in the Proteobacteria phylum is observed.

Through metabolomic profiling, obesity can be characterized by specific changes in the gut microbiota and circulating metabolites, including alterations in amino acid and lipid levels.

In obesity, dysbiosis biomarkers are characterized by a reduction in bacterial diversity, an increased Firmicutes/Bacteroidetes ratio, and a decreased abundance of beneficial bacteria such as Akkermansia muciniphila and Faecalibacterium prausnitzii. These changes are associated with chronic inflammation, increased intestinal permeability, and metabolic disturbances, accompanied by elevated levels of inflammatory markers such as C-reactive protein, interleukin-6, and lipopolysaccharides.

Recent studies have shown that GM may be useful in preventing obesity by enhancing UCP-1–dependent thermogenesis. In addition, GM also mediates the effect of curcumin in preventing and treating obesity through the UCP-1 pathway. New scientific research suggests that changes in the composition of the intestinal microbiota (IM) may occur in obesity, T2DM, dyslipidemia, and non-alcoholic fatty liver disease.

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