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Correlation between Gut Microbiota and Obesity

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Abstract:

Obesity and its related metabolic disorder have turned into a significant global health issue. This review combines the present comprehension of the intricate ways by which the gut microbiome impacts the host's metabolism and obesity.Besides,it elucidates how the structure and functional makeup of the gut microbiome affect the energy equilibrium by controlling the energy derived from the diet, managing systemic lipid metabolism, and influencing endocrine functions. One vital mechanism is the function of the microbiome in disturbing the integrity of the intestinal barrier resulting in metabolic endotoxemia and chronic low - grade inflammation which then leads to insulin resistance and promotes fat accumulation and moreover this review underlines the part of microbial metabolites (such as short - chain fatty acids (SCFAs) and secondary bile acids) as signaling molecules which can influence the host's appetite regulation, insulin sensitivity, and thermogenesis in adipose tissue and evidence indicates that the traits of the gut microbiome causing obesity are typically an elevated proportion of Bacteroides and Fusobacterium which boost energy extraction and storage. While a healthy and diverse microbiome might prevent obesity induced by diet by increasing energy expenditure.

Keywords: Obesity; Gut Microbiota; SCFAs.

1. Introduction

Obesity is a chronic disease caused by excessive accumulation of fat especially visceral fat.BMI is the ratio between body mass and the square of height,BMI is the standard to evaluate obesity, W.H.O defined that BMI greater than or equal to 30(kg/m^2) as being of obese but different counties have different standards [1].Nonetheless it is inappropriate using only BMI as the criterion because when the muscle content in human body increases the BMI increases as well.Compares to BMI,body fat rate is more accurate,25 percent is the bound of obesity for male and 35 percent for female [2]. In recent years,with the rapid economic development,the quality of people's lives has been improved.Global obesity rates continue to rise [3].According to statistics from WHO,the global population suffering from obesity exceeded 1 billion. Since 1990, the number of obese adults worldwide has more than doubled, and the number of

obese children and teenagers (aged 5 to 19) has increased by three times.

Metabolic syndrome triggered by obesity have emerged as a global health concern which is a threat to health,such as cardiovascular diseases, diabetes, cancer, neurological diseases, chronic respiratory diseases, and digestive system diseases [4]. There are plenty reasons for obesity including unhealthy lifestyle habit,inheriting from parents,failure of immune and metabolic system,consuming excess food [5].This leads to energy intake override the energy expenditure.The role of gut microbiota in this context is gradually gaining increased attentiong.Existing research has revealed that the gut microbiota participates in the development of obesity through multiple mechanisms, such as energy metabolism,inflammatory response, and immune function .Therefore,regulating the Gut Microbiota has become a new strategy for preventing and treating obesity and its associated metabolic syndromes .But,Currently, there lacks a specific elaboration,systematic organization, and analysis regarding the association between gut microbiota and obesity.This research aims to provide theoretical support and practical guidance for related research by conducting a comprehensive review on the mechanisms,regulatory strategies,and potential therapeutic targets of the relationship between Gut Microbiota and obesity.

2. Gut Microbiota

Gut microbiota constitutes a complex ecosystem within the human body,which consists of archaea,viruses,fungi and bacteria.The maintenance of an organism's health depends on a stable intestinal ecological environment and this is achieved by synergistic effect among multiple microorganisms,which are interdependent and mutually restrictive [6].Gut microbiota is closely related to the energy balance and metabolism of the host.When the ecosystem of gut microbiota is unbalanced,there is possibility of causing abnormal metabolism.Gut microbiota is actually consist of countless microorganisms,the factors driving the types of initial organism includes different childbirth ways,feeding patterns,ages,geographical position and gender.So the gut microbiota began to form since the moment baby born.The microorganisms depends on what people eat. For instance, eating diet high in protein and fat for long term,there may be metabolic waste produced which can damage blood vessels.However,if people consume sufficient dietary fiber,production of short chain fatty acids can contribute to the inhibition of inflammation and modify blood glucose in appropriate leve [2].

3. Mechanisms by Which Gut Microbiota Affect Obesity

3.1 Intestinal Barrier and Inflammation

The human intestinal barrier consists of four parts: first, there is the mechanical barrier formed by closely packed intestinal epithelial cells and a mucus layer; second, the chemical barrier made up of substances like digestive juices secreted by the intestines, bile acids, and antimicrobial peptides; third, the immune barrier composed of Gut - associated lymphoid tissue (GALT) and various immune cells; fourth, the microbial barrier formed by symbiotic bacterial communities [7]. And all of them are able to separate the internal organs from inflammatory factors, some harmful microorganisms, etc.

A healthy intestine has selectivity in being permeable to nutrients, water, and bacteria, but once the barriers are impaired, harmful microorganisms can pass through the intestine, reach the host tissues, cause inflammation, and lead to diseases [2];one crucial mechanism connecting the gut microbiota to obesity is the induction of low - grade systemic inflammation, a key element in this process is the decreased expression of tight junction proteins like ZO - 1 and occludin, which makes the intestinal permeability increase [8]. This allow bacterial endotoxins, especially lipopolysaccharide (LPS) from Gram - negative bacteria, to seep into the portal circulation and then into the systemic circulation - a situation known as metabolic endotoxemia, and LPS is a strong activator of the innate immune system via binding to Toll - like receptor 4 (TLR4) on immune cells, triggering the release of pro - inflammatory cytokines (such as TNF - α, IL - 6, IL - 1β) from macrophages and other immune cells that have invaded adipose tissue and the liver, and this chronic low - grade inflammation is a major factor in insulin resistance as inflammatory pathways disrupt insulin signaling, also hindering fatty acid oxidation and promoting lipid accumulation, forming a vicious cycle that worsens obesity and its metabolic complications [9].

3.2 Energy Harvesting

One significant manner in which the gut microbiota gives rise to obesity is through augmenting energy extraction from the diet as humans are devoid of the enzymatic capability to completely break down numerous complex dietary polysaccharides and certain commensal bacteria, particularly those within the Firmicutes phylum which encode a substantial quantity of carbohydrate - active enzymes, efficiently assume this function by fermenting indigestible fibers and resistant starches to generate short

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- chain fatty acids (SCFAs) like acetate, propionate, and butyrate; although SCFAs serve as a crucial energy source for colonic cells and possess advantageous anti - inflammatory characteristics, their systemic absorption impacts the host‘s overall energy balance; in the dysbiotic microbial community state frequently observed in obesity, there are distinctive alterations in the proportion of intestinal bacteria with an upsurge in the abundance of Firmicutes in comparison to other bacteria and this modified community structure is linked to augmented energy reaping from the identical diet resulting in more calorie absorption, heightened de novo lipogenesis in the liver, and subsequent fat sequestration in adipose tissue thus promoting weight gain; research from Nature emphasizes the role of the Firmicutes phylum in aiding energy extraction as these bacteria were demonstrated to enhance the host‘s capacity to glean energy from dietary fibers consequently contributing to adipose tissue growth then leads to obesity [10,11].

3.3 Lipid Metabolism

Beyond just extracting energy, the gut microbiota has an essential part to play in regulating the body‘s lipid metabolism overall, for bacterial by - products, especially bile acids and SCFAs, serve as strong signaling agents that affect the host‘s lipid balance, and primary bile acids made in the liver are transformed by gut bacteria into secondary bile acids which aren‘t simply worthless substances but work like hormones activating nuclear receptors like the farnesoid X receptor (FXR) and the G protein - coupled bile acid receptor 1 (TGR5), and when these receptors get activated, they control a number of metabolic procedures, among them hepatic gluconeogenesis, fatty acid breakdown, energy usage, and insulin sensitivity, so if there are changes in the makeup of the gut microbiota, it can cause substantial alterations in the bile acid pool, which then upsets metabolic well - being and contributes to obesity, and likewise, SCFAs attach to particular G protein - coupled receptors (GPCRs), such as GPR41 and GPR43 (also named FFAR2 and FFAR3), present on enteroendocrine cells, fat cells, and immune cells, and this attachment governs processes like fat synthesis, fat breakdown, and energy expenditure, once more emphasizing the crucial role of the microbiota in lipid control [11].

3.4 Endocrine Hormones Secretion

The gut microbiota acts like a virtual endocrine organ, having a great influence on how the host regulates its appetite and metabolism through hormonal means; it is involved in controlling the secretion of important endocrine hormones from the L cells within the intestinal epithelium, such as glucagon - like peptide - 1 (GLP - 1) and peptide YY (PYY), with SCFAs, particularly propionate and butyrate, being strong stimuli for releasing these hormones; GLP - 1 and PYY play vital roles in making one feel full, delaying gastric emptying, and boosting insulin secretion after meals, so if there was an imbalance among microbes, the production or signaling of these hormones might be affected, resulting in enhanced appetite, decreased feeling of fullness, and poor blood sugar management, all of which could lead to overeating and weight gain.

New research has elucidated how the gut microbiota regulates the biology of adipose tissue and thermogenesis; besides white adipose tissue (WAT) that stores energy, mammals also have brown adipose tissue (BAT) and beige adipose tissue (which shows up in WAT), both of which are metabolically active and expel energy as heat via a process known as non - shivering thermogenesis, with this process being facilitated by uncoupling protein 1 (UCP1); some microbial metabolites, such as certain bile acids and SCFAs, were found to activate this thermogenic mechanism, for example, secondary bile acids could stimulate TGR5 signaling on enteroendocrine cells, resulting in an elevation of circulating GLP-1, which might indirectly boost thermogenesis and more straightforwardly, activating TGR5 on adipocytes could raise cAMP levels and drive UCP1 - dependent heat generation; butyrate was also demonstrated to activate AMPK signaling and prompt the browning of WAT; consequently, a healthy and varied gut microbiota might augment energy expenditure by promoting thermogenesis and forestall diet - induced obesity, whereas a dysbiotic microbiota might be devoid of this protective function.

4. Conclusion

In the end, gut microbiota was a crucial controller of energy equilibrium and a vital element in the growth and spread of obesity, and the intricate ecosystem inside the gastrointestinal tract didn‘t just passively react to dietary intakes but took an active part in deciding the metabolic destiny of the host, and through strengthening the capacity to draw energy from non - digestible dietary parts, the gut microbiota directly influenced the calorie supply, and what‘s more, it coordinated a great number of systemic metabolic procedures by generating a set of metabolic products having strong signaling molecule impacts, and the disturbance of gut microbiota balance (dysbiosis) and the ensuing damage to intestinal barrier function could set off a string of occurrences such as metabolic endotoxemia and chronic low - level inflammation which played a substantial role in causing insulin resistance and spurring fat build - up in adipose tissue.

Moreover, the gut microbiota has functions like those of endocrine organs and can regulate the secretion of crucial hormones such as GLP - 1 and PYY which are in charge of controlling satiety and glucose metabolism; its function in affecting the browning of white adipose tissue and promoting thermogenesis further emphasizes its ability to regulate energy expenditure; the combined effect of these mechanisms - impacting two sides of the energy balance equation - strengthens the status of the microbiota as a vital element in obesity; the fact that the composition of the gut microbiota is transmissible and can directly influence the weight phenotype in animal models underlines its role in causing disease; thus, approaches aimed at reestablishing a healthy and diverse gut microbiota like personalized nutrition techniques, prebiotics, probiotics, and next - generation microbiota - based therapies hold great promise for the management of future obesity; subsequently, future research should concentrate on expanding these findings from animal models to humans, pinpointing specific harmful and beneficial bacterial species, and creating targeted, safe, and efficient interventions to regulate the gut microbiota so as to enhance metabolic health.

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