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Dual Mechanisms Regulating Obesity: DNA Methylation and Gut Microbiota

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Description

Obesity is a global health crisis, contributing to a range of chronic diseases such as diabetes, cardiovascular disorders, and certain cancers. The prevalence of obesity has been escalating at an alarming rate, making it imperative to understand its underlying mechanisms. Recent research has illuminated two critical factors in the regulation of obesity: DNA methylation and gut microbiota. These dual mechanisms offer promising insights into potential therapeutic strategies to combat obesity. Obesity is a complex condition influenced by a myriad of factors, including genetics, environment, lifestyle, and metabolic processes. Traditional views of obesity focused primarily on caloric intake and physical activity. However, emerging evidence suggests that biological mechanisms at the molecular level play a significant role in the development and persistence of obesity [1].

DNA methylation is an epigenetic modification involving the addition of a methyl group to the DNA molecule, typically at cytosine bases in the context of CpG dinucleotides. This modification can influence gene expression without altering the underlying DNA sequence. Methylation can either repress or activate gene transcription, depending on the context and location within the genome. DNA methylation has been linked to obesity through its impact on genes involved in metabolic processes, appetite regulation, and fat storage. Epigenetic changes can be influenced by various factors, including diet, physical activity, and environmental exposures. These changes can be heritable, potentially passing on obesity-related traits across generations. DNA methylation patterns in the genes encoding leptin and adiponectin, hormones involved in appetite regulation and insulin sensitivity, are altered in obese individuals. Hypermethylation of these genes can lead to reduced expression, disrupting normal metabolic functions. The proopiomelanocortin gene, which plays a crucial role in energy homeostasis and appetite control, has also been found to be differentially methylated in obese individuals [2].

Hypomethylation of the POMC gene can result in increased gene expression, promoting hyperphagia (excessive eating) and weight gain. Studies have demonstrated that early-life environmental factors, such as maternal diet during pregnancy, can influence DNA methylation patterns in offspring, predisposing them to obesity later in life. These findings underscore the importance of maternal health and nutrition in preventing obesity in future generations. The gut microbiota refers to the trillions of microorganisms residing in the human gastrointestinal tract. These microbes play a crucial role in digestion, nutrient absorption, immune function, and overall health. The composition and diversity of gut microbiota can be influenced by diet, lifestyle, antibiotics, and other factors. Gut microbiota has emerged as a key player in the development of obesity. The microbial composition in the gut can influence energy balance, fat storage, and metabolic processes. Dysbiosis, an imbalance in the microbial community, has been linked to obesity and related metabolic disorders. Obese individuals often exhibit an altered ratio of Firmicutes to Bacteroidetes, two dominant bacterial phyla in the gut. An increased Firmicutes/Bacteroidetes ratio has been associated with enhanced capacity to harvest energy from the diet, contributing to weight gain [3].

Gut microbes ferment dietary fibers to produce short-chain fatty acids such as acetate, propionate, and butyrate. SCFAs play a role in regulating appetite, glucose metabolism, and fat storage. Altered SCFA production has been observed in obese individuals, potentially impacting metabolic health. The gut microbiota communicates with the brain through the gut-brain axis, influencing appetite and food preferences. Certain gut bacteria can produce neurotransmitters and other signaling molecules that affect brain function and behavior. Dysbiosis may disrupt this communication, leading to overeating and weight gain. The relationship between DNA methylation and gut microbiota is bidirectional and complex. Gut microbiota can influence DNA methylation patterns through the production of metabolites that act as substrates or inhibitors of enzymes involved in epigenetic modifications. Conversely, epigenetic changes can affect the composition and function of the gut microbiota [4].

Short-chain fatty acids produced by gut bacteria can serve as substrates for histone acetylation and inhibitors of histone deacetylases influencing gene expression. Butyrate, in particular, has been shown to have anti-inflammatory and anti-obesity effects through its impact on epigenetic regulation. Dietary components can modulate both gut microbiota and DNA methylation. For example, a high-fat diet can induce changes in gut microbiota composition and promote epigenetic alterations associated with obesity. Conversely, a diet rich in fruits, vegetables, and fiber can support a healthy microbiota and favorable epigenetic profiles. Maternal diet and gut microbiota during pregnancy can shape the offspring's microbiota and epigenetic landscape, affecting their susceptibility to obesity. Interventions targeting maternal health and nutrition may offer a strategy to prevent obesity in future generations.

Understanding the dual mechanisms of DNA methylation and gut microbiota in regulating obesity opens up new avenues for therapeutic interventions. Potential strategies include, modulating the gut microbiota through probiotics (beneficial bacteria) and prebiotics (nondigestible fibers that promote the growth of beneficial bacteria) can help restore microbial balance and improve metabolic health. Drugs targeting epigenetic modifications, such as DNA methyltransferase inhibitors or histone deacetylase inhibitors, hold promise for treating obesity by reversing adverse epigenetic changes. Tailoring dietary recommendations based on an individual's genetic and epigenetic profile, as well as their gut microbiota composition, can optimize weight management and metabolic health. Promoting healthy lifestyle habits, including regular physical activity, stress management, and adequate sleep, can positively influence both DNA methylation and gut microbiota, contributing to obesity prevention and management [5].

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Conflict of Interest

None.

References

1. Jebeile, Hiba, Aaron S. Kelly, Grace O'Malley and Louise A. Baur. "Obesity in children and adolescents: Epidemiology, causes, assessment, and management." *Lancet Diabetes Endocrinol* 10 (2022): 351-365.
2. Cai, Dongsheng and Sinan Khor. ""Hypothalamic microinflammation" paradigm in aging and metabolic diseases." *Cell Metab* 30 (2019): 19-35.
3. Miller, Alison L., Hannah J. Lee and Julie C. Lumeng. "Obesity-associated biomarkers and executive function in children." *Pediatr Res* 77 (2015): 143-147.
4. Phillips, B. Allyson, Shari Gaudette, Andy McCracken and Samiya Razzaq, et al. "Psychosocial functioning in children and adolescents with extreme obesity." *J Clin Psychol Med Settings* 19 (2012): 277-284.
5. Lung, Thomas, Stephen Jan, Eng Joo Tan and Anagha Killedar, et al. "Impact of overweight, obesity and severe obesity on life expectancy of Australian adults." *Int J Obes* 43 (2019): 782-789.

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