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Obesity and Gut Microbiome: Current Evidence

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ABSTRACT

Obesity, a multifactorial metabolic disorder characterized by excessive fat accumulation, has become a leading global health challenge. Recent research underscores the gut microbiome, a complex community of microorganisms inhabiting the human gastrointestinal tract as a crucial regulator of host metabolism, energy homeostasis, and inflammation. Dysbiosis, defined as an imbalance in microbial composition, has been closely associated with obesity and its related metabolic complications. Current evidence suggests that gut microbial diversity, diet, genetics, and environmental factors interact dynamically to influence weight gain and metabolic efficiency. Alterations in the Firmicutes/Bacteroidetes ratio, changes in short-chain fatty acid (SCFA) production, and modulation of bile acid metabolism highlight key mechanistic links between gut microbiota and obesity pathogenesis. Emerging studies emphasize the promise of microbiome-targeted interventions such as probiotics, prebiotics, fecal microbiota transplantation (FMT), and dietary modifications to restore microbial balance and improve metabolic health. However, methodological challenges, including small sample sizes, confounding factors, and data variability, limit the reproducibility and interpretation of findings. Longitudinal studies demonstrate that gut microbial diversity fluctuates following interventions such as bariatric surgery, reflecting the microbiome's responsiveness to metabolic shifts. Personalized medicine approaches integrating gut microbial profiling, nutrigenetics, and omics-based analyses offer potential for individualized weight loss and disease prevention strategies. The public health implications of these findings are profound, as microbiome-informed interventions could provide low-cost, non-invasive strategies for obesity prevention and management. Nevertheless, ethical challenges, such as informed consent, data ownership, and commercialization of microbiome data demand careful governance to ensure equitable access and responsible innovation. To advance understanding, future research must prioritize large-scale, population-based longitudinal studies to establish causality, identify key microbial species, and evaluate the efficacy and safety of microbiome-based therapies. By bridging mechanistic insights with personalized and ethical applications, microbiome science holds transformative potential for obesity control and global metabolic health improvement.

Keywords: Gut Microbiome, Obesity Pathogenesis, Personalized Medicine, Microbiome-Based Therapy and Ethical Considerations in Microbiome Research.

INTRODUCTION

Obesity is a chronic metabolic disease characterized by excess fat accumulation resulting from an imbalance between energy intake and expenditure. Globally, it constitutes a major health problem, being associated with an increased risk of cardiovascular, respiratory, diabetic, and cancer-related diseases [1-5]. The prevalence of obesity has increased rapidly in recent years; about one-third of the global population is overweight, with 10% obese. Projections indicate that more than 1.12 billion people may suffer from obesity by 2030. The disorder has diverse aetiologies caused by multifactorial pathophysiological mechanisms, including sedentary lifestyles, unhealthy eating habits, genetic factors, environmental factors, and secondary causes such as endocrine and central nervous system disorders [6-8].

Understanding Gut Microbiome

Gut microbiota is a large and diverse microbe community inhabiting our intestine and consists of archaea, bacteria, viruses, protists, and fungi, capable of profoundly affecting human physiology as a whole [9-12]. All said, bacteria

situated in our gut represent the largest group and hence are greatly considered when defining gut microbiome diversity [13-15]. Gut microbiota is involved in several functions such as training the immune system, preventing the colonization of pathogens, fermenting unused energy substrates, producing vitamins, regulating the host energy balance, and maintaining the integrity of the gut mucosal barrier [16-19]. Alterations or imbalances in the microbiota can lead to several diseases, including cancer, cardiovascular diseases, diabetes, inflammatory bowel disease, bacterial infections, and obesity [20-26]. The association between obesity and gut microbiota is well documented [27-32]. The potential underlying mechanisms of this association include several metabolic, inflammatory, and immune pathways. The modulation of gut microbiota by diet, antibiotics, or host genetics can induce the risk of obesity development [33-35]. It has been observed that a high-fiber diet or probiotics/prebiotics can provide a protective function against the development of obesity, whereas a high-fat diet or the use of antibiotics or germ-free conditions may increase the risk of obesity [36-40]. Additionally, ongoing research is testing the potential of fecal microbiota transplantation for disease regulation. Current animal and human studies also suggest that the changed composition and diversity of the gut microbiota might affect the pathogenesis of obesity [18]. Further research in this area might help in preventing or regulating obesity and associated diseases [41-44].

The Link between Obesity and Gut Microbiome

The increasing prevalence of obesity has stimulated an extensive search for biological factors that (re)shape the aetiology of obesity [45-49]. One of the most remarkable biological factors during the last decade is the vastly complex gut microbiome, which interacts with obesity on multiple levels [12]. The influence of the gut microbiome on obesity is so profound that the microbiome can be regarded as an independent variable that not only shapes host metabolism but substantially modulates vital environmental triggers including diet [50-54]. This section therefore aims for a comprehensive overview of current evidence of the potential role that gut microbiome play in obesity [13]. Several clinical studies demonstrate a strong association between gut microbiome and obesity [2]. A widely recognised finding is that obesity drastically alters gut microbial ecology, which results in a profound variation in the composition and functionality of the gut microbiome[55-59]. Additional such studies provide evidence that the establishment of obesity-related type 2 diabetes, fatty liver disease and cardiovascular disease correlates directly with a substantial shift in the composition of the gut microbiome[15]. Fundamental to these scientific observations is the fact that the gut gut microbiota consist of a tremendous variety of microorganisms including bacteria, viruses, fungi, protozoa and archaea[19]. The abundance of each of these microbial clades in an individual depends on a myriad of environmental if not, enteric parameters that shape the gut microbiome, including; host genotypic composition, lifestyle aspects, hygienic standards, antibiotics and diet. Alcove equally critical to any epidemiological approach, the diversity of gut microbes in the obese is typically lower and overall functionality is also severely incognizant [60-65]. Loose correlations therefore stimulate the search into the intricacies that connect obesity and gut microbiome on more detailed levels and this section thus sets the stage for the following section since it subsequently considers the mechanisms and metabolic pathways mediating obesity associated with altered gut microbes [66-70]. Most studies relating the development of obesity with the composition of the gut microbiota remain largely correlative, and the exact causal role that microbiota and certain microbial species might play during the development and the establishment of obesity remains highly obscure[14]. The subsequent section addresses all known mechanisms of action through which the formation and the development of obesity and the gut microbiota intimately interact [71-75].

Mechanisms of Interaction

Multiple pathways mediate an interaction between obesity and gut microbiome. Metabolic and immune responses constitute two major mechanisms involved [18]. Adjustments in microbial products, such as peptide YY (PYY), glucagon-like peptide-1 (GLP-1), and endocannabinoids influence host physiology underpinning obesity microbiome crosstalk [4]. Excess energy supply also modifies gut microbiota to promote the development of fat storage through its suppression on the adenosine monophosphate–activated protein kinase (AMPK) and fasting induced adipocyte factor (FIAF), cf. Section [3]. Interactions between the microbiota and host also regulate adiposity and play a central role in obesity [5]. Gut microbes contribute to obesity by regulating fat storage. Bile acids inhibit growth of certain members of the microbiota by disturbing membrane integrity [76-79]. Although, some bacteria possess specific bile salt hydrolysing enzymes that can reduce detergent activity and increase bacterial survival in the gut[13]. Bile acids are endogenous ligands for the membrane-bound G-protein-coupled receptor (GPCR) such as the G-protein-coupled bile acid receptor-1 (Gpbar-1), also known as Takeda G-protein-coupled receptor-5 (TGR5), and the Farnesoid X receptor (FXR), among others. These receptors constitute an important link connecting the gut microbiota to obesity and glucose homeostasis [15]. An exposure to an obesogenic diet induces dramatic changes in microbial community structure in animal models followed by modulation of fecal and circulating bile acids pool [16]. Fecal mitochondria transfer on germ-free recipients

reproduces the phenotype observed in donor mice indicating that the gut microbiota acts as causative factors [2]. The gut microbiota contributes also to the regulation of body weight and adiposity through the production of short-chain fatty acids (SCFAs) by anaerobic fermentation of dietary fibers [80-81]. Colonic SCFAs, such as acetate, propionate and butyrate, are energy source that modulate the composition of the gut microbiota and regulate host signalling pathways through several mechanisms, including epigenetic regulatory activity. Murine models support the hypothesis that a low fibre diet, which is associated with a decline in SCFA levels in the intestine, increases host susceptibility to infection by the enteric pathogen Clostridioides difficile[12]. The influence of SCFAs on host physiology and obesity management is also mediated by their ability to signal through G-protein-coupled receptors (GPCRs) such as GPR41, GPR43 and GPR109A, which are expressed on the intestinal epithelial cells and immune cells located in the gut. Obesity is often associated with increased intestinal permeability and metabolic endotoxemia, which may be important in the context of the development of chronic low-grade inflammation and insulin resistance; cf. Section [80-83].

Metabolic Pathways

Numerous metabolic routes link gut microbiome alterations to obesity [1]. The development of germ-free animal models was instrumental in identifying and understanding these mechanisms [17]. For instance, germ-free rodents conventionally inoculated with caecal or fecal samples from conventional donors display enhanced body fat synthesis and weight gain. Early studies showed that conventionalization of germ-free mice were associated with a 60% increase in body fat content and insulin resistance, despite a reduction in food intake [4]. Gut microbes also influence several compounds regulating dietary intake, energy expenditure and fat storage, such as the incretin hormone GLP-1, peptide YY and endocannabinoids[20]. These observations demonstrate that the gut microbiome helps the host extract, store and utilize energy from the diet, contributing to the development of obesity [21].

Immune Response

Obesity is marked by an enhanced inflammatory state of adipose tissue, characterized by increased immune cell influx and upregulated cytokine production [3]. Altered gut microbiota composition also occurs in obesity and related diseases, such as type 2 diabetes, non-alcoholic fatty liver disease, and cardiovascular disease [6]. In obesity, gut bacteria may stimulate local intestinal inflammation, facilitating penetration of bacterial components from the gut lumen into the systemic circulation [7]. Circulating bacterial components then reach adipose tissue, where they activate local immune cells and induce further inflammation [8]. Translocation of bacteria from the gut lumen to adipose tissue has been documented [1].

Role of Diet in Modulating Gut Microbiome

Diet remains the strongest factor modulating gut microbial composition and functionality [9]. The gut microbiome has evolved by constantly adapting to different environments and, in particular, to different diets [8]. Moreover, the gut microbial composition changes rapidly in response to changes in diet. High-fiber diets are associated with gut microbiome profiles enriched in fiber-degrading Firmicutes, Bacteriodetes, and Actinobacteria, whereas, in Westernized societies, high-fat and high-sugar diets allow Firmicutes that degrade mucins to dominate [2]. Maintenance of a healthy gut microbial composition requires the continuous supply of fermentable substrates. Supplementation with pro- and prebiotics is another way to shape microbial composition by directly feeding the beneficial microbes [7]. Dietary intervention has become a promising approach to shift the composition of an altered gut microbial ecosystem towards a healthy gut microbiome[7].

High-Fiber Diet

Fiber is a nondigestible form of carbohydrate found in plant foods [1]. The term "dietary fibre" refers to the intrinsic fibre found within a plant matrix. "Functional fibre" refers to isolated fibre extracted from plants and added to supplements or foods. "Total fibre" refers to the combination of dietary fibre and functional fibre combined [8]. Dietary fibre is abundant in fruits, vegetables, legumes, and whole grains; it helps regulate hunger and maintain blood-sugar levels [9]. Fiber serves as a substrate for colonic bacterial fermentation, producing short-chain fatty acids (SCFAs), which serve as fuel for the gut epithelium and have an impact on energy balance [2].

High-Fat Diet

High Fat Diet (HFD) is associated with chronic diseases such as obesity, diabetes, gastrointestinal diseases, neurodegeneration, and cardiovascular diseases [13]. Common factors underlying HFD-induced chronic diseases include bile acids, lipopolysaccharide, short-chain fatty acids, and trimethylamine N-oxide [16]. Signaling pathways including FXR, TGR5, NF-κB, PPAR-γ, and PERK play critical roles in microbiota modulation and disease mechanisms [10]. Excessive consumption of saturated and trans fats, especially in Westernized diets, correlates with weight gain, organ fat accumulation, gut microbiota dysbiosis, insulin resistance, oxidative stress, and cognitive impairment[8]. Human studies document HFDs comprising 52–60% energy from fat [17]. The gut

microbiota encompasses over 1000 bacterial species, constituting a biological interface between the external environment and the organism that maintains host physiology and its ability to adapt to environmental changes. Dysbiosis, an imbalance of the intestinal microbial community, is involved in several health problems [19]. Key questions remain on gut microbiota development, its influence on host metabolism, and the shaping effect of dietary factors [4]. The composition of gut microbiota in obesity, type 2 diabetes, and related diseases and the impact of bacteria such as Bilophila and Akkermansia muciniphila have been extensively reviewed. Culture-based approaches from 1965 to 1992 suggested dietary fatty acids do not influence intestinal bacteria [18]. However, fluorescent in situ hybridization and denaturing gradient gel electrophoresis reveal a profound impact of HFD on gut microbiota composition in mice, affecting Bacteroides-like species, Enterobacteriaceae, and Bifidobacterium, with a decreased abundance of Bifidobacterium spp [11].

Probiotics and Prebiotics

Alongside continued efforts to understand the links between the gut microbiome and obesity, new therapeutic strategies are under investigation [12]. Restoring the microbiota's composition and richness may offset obesity and metabolic dysfunction [13]. Manipulations include dietary supplements with prebiotics and probiotics, which alter microbial composition and metabolism and reduce host metabolic dysregulation[6]. "Probiotics" denotes live microorganisms that, when administered in appropriate amounts, confer a health benefit to the host [12]. Probiotics have been associated with modest weight loss and reduction of hyperglycemia, insulin resistance and chronic inflammation, possibly through modulation of gut microbial functions such as energy metabolism [7].

Genetic Factors Influencing Gut Microbiome

Host genetics shape gut microbial diversity and composition, thereby influencing obesity susceptibility. Colonization of the human gut begins at birth and continues throughout life, resulting in widespread microbial variability among individuals [1, 3]. Exogenous factors such as diet, antibiotics, and infection affect microbial composition; however, the permanent sources of this imbalanced composition remain unclear [8]. Variations in the composition of the microbiota within an individual may originate from many factors, including the host's genetics [13]. Host genetics shape gut composition and microbial arrangement, indicating that the microbial ecosystem could be considered among the phenotypes influenced by an individual's genetics [6, 9]. Hence, the immune system and various physiological variables regulated by the host genotype appear to have a crucial role in defining microbial selection, thereby directly or indirectly affecting an individual's intestines [14].

Impact of Antibiotics on Gut Microbiome

Widespread antibiotic use facilitates pathogens and reduces diversity of commensal bacteria, with potentially long lasting consequences on human health [11]. Epidemiological evidence shows a relation between antibiotics use in early life and increased risk of childhood and adulthood obesity [13]. Other studies have linked antibiotic use with obesity, type 2 diabetes an immunological diseases [10]. One mechanism raises the possibility that antibiotics-induced changes in the gut microbiota favour energy harvest from the diet and consequently promote obesity and related metabolic complications [15]. Modulation of the gut microbiota has been proposed as an approach for treatment of obesity and related metabolic disorders [16].

Gut Microbiome and Inflammation

Inflammation is a probable mechanism by which microbiome perturbations affect obesity. Whereas low-grade inflammation in adipose tissue is driving obesity-related disorders such as insulin resistance [3], overt inflammation is one of the best-known triggers of weight loss [6]. Inflammation is invariably present in obesity and insulin resistance, but it remains often unresolved which triggers and mediators initiate and propagate the inflammatory process [25]. Recently, there has been mounting evidence for a central role of the gut microbiota in inflammation and obesity, and alterations in the intestinal microbiota are increasingly being implicated in the initiation and perpetuation of obesity-associated inflammation [26].

Current Research and Findings

Studies using experimental models provide evidence supporting the influence of intestinal microbiota on obesity and related disorders [3]. Interventions such as antimicrobials, fecal transplantation, prebiotics, and probiotics significantly modulate the composition and activity of these communities, reinforcing their potential in obesity management [2]. Data from both human and animal models like pigs indicate that microbiotal shifts impact host health, and diet-induced obesity correlates with alterations in microbial populations' effects that can be reversed, possibly through dietary adjustments [2]. These observations underline the gut microbiota as a major environmental factor associated with obesity [14]. Reviews highlight that while the microbiome is recognized as a key player, its precise contribution remains uncertain since most evidence derives from rodent studies and associative observations, especially in humans [17]. Moreover, gut microbiota can trigger low-grade inflammation, further implicating it in obesity etiology. Recent longitudinal human studies support a role of the gut microbiome in obesity pathophysiology [18].

Animal Studies

Obese and lean phenotypes can be transferred to recipient mice by means of microbiota transplantation [18]. Detailed comparisons of the gut microbiome among mouse models suggest that the administration of Christensenella minuta, which is strongly depleted in obese individuals, can reduce adiposity gain in treatment-naive mice[12, 18]. However, the sequence of microbiome alterations and fat accumulation remains unclear, and the impact of gut microbes and their metabolites on host energy metabolism has yet to be elucidated, indicating a need for further investigation [12].

Human Clinical Trials

Human clinical trials provide insights into the impact of interventions on the gut microbiome, obesity, and metabolic health [3]. These studies have established links between BMI and metabolic abnormalities, obesity-related cancer risk, and demonstrated that dietary fibers and resistant starches can modulate cholesterol levels via fermentation [3]. The relationship among the gut microbiota, diet, and obesity is complex, affecting nutrient absorption and metabolic outcomes [1]. Host genetics, such as mutations in the leptin receptor gene, influence obesity susceptibility, adding further complexity to the interplay between environmental factors and individual predisposition. Weight-loss surgeries and dietary interventions can modify the microbiota and thereby influence metabolic health [19]. Probiotic approaches are still maturing and require further long-term clinical evaluation; nevertheless, the administration of probiotics, prebiotics, and synbiotics represents a promising and inexpensive strategy to beneficially alter the intestinal microbiota[15]. Specific probiotic strains have demonstrated potential in limiting energy absorption, increasing energy expenditure, and improving barrier integrity, as well as preventing inflammation and metabolic endotoxemia [12].

Gut Microbiome as a Therapeutic Target

Therapeutic approaches to restore healthy microbial composition and improve metabolic health have been proposed [1, 6]. Among these strategies, fecal microbiota transplantation (FMT) presents a promising option [7]. FMT involves the delivery of faeces from a donor with the desired microbiota to a recipient who lacks it. This method has been successfully used to treat diarrhoea, irritable bowel syndrome, and some autoimmune diseases. The microbiota is deeply involved in personal metabolism and can serve as an ideal target to improve health [6]. Prebiotics and probiotics constitute part of the strategy to counter obesity because of their benefits and low risks [9]. Emerging techniques such as FMT could be important for weight loss and the improvement of clinical status in obese individuals [2]. As scientific advances continue, treatments aimed at microbiota recovery may become common options for diseases related to obesity and metabolic dysbiosis [20].

Fecal Microbiota Transplantation

Fecal microbiota transplantation (FMT) involves transferring fecal bacteria from a healthy donor into the intestinal tract of a recipient with altered gut microbiota, aiming to confer a health benefit [10]. A pilot study in human subjects evaluated the impact of metabolic syndrome through FMT from lean donors [16]. Improvement in peripheral and hepatic insulin sensitivity was found in recipients after six weeks, with an associated increase in butyrate-producing intestinal microbiota[12]. Another controlled-randomized trial examined the recolonization of the gut microbiota following antibiotic treatment [11]. It demonstrated that recolonization may differ depending on the origin of the bacteria present in the donor stool used for transplantation [26]. In the antibiotic-only group, an increased abundance of Firmicutes, particularly Blautia, was observed, whereas in the high-dose FMT group, higher proportions of Bacteroidetes and Parabacteroides were evident [23]. A more recent systematic meta-analysis underscored the significant influence of the donors' microbiota on the health and well-being of the recipients [12]. Other investigations have examined FMT in relation to inflammation-related diseases, providing important insights into treatment potentials [25]. Together, these findings open perspectives on the use of FMT in obesity and further elucidate the roles of gut microbiota and its products in obesity-related inflammation (cross-reference section "Gut Microbiome and Inflammation")[13].

Microbiome-Based Therapies

Among the novel therapeutics proposed for obesity are approaches that target the gut microbiome, including fecal microbiota transplantation (FMT) and various microbiome-based therapies [15]. FMT, already used to treat recurrent Clostridium difficile infection, has attracted interest for its potential to influence a range of conditions such as irritable bowel syndrome, inflammatory bowel disease, multiple sclerosis, cancer, and metabolic disorders [21]. This expanding repertoire underscores the microbiome's involvement in obesity, metabolic syndrome, and type 2 diabetes through mechanisms linked to inflammation and short-chain fatty acid signaling [13]. Given that dietary patterns can modulate gut microbiota, impacting metabolic health, alterations in the microbiome clearly represent intriguing targets for therapeutic intervention [18]. Prebiotics and probiotics are among the agents utilized to adjust microbial composition, offering a potential means to mitigate obesity while posing limited risks [7]. FMT and other microbiota modulation techniques are gaining importance as strategies for weight loss and clinical improvement in obese individuals, suggesting that microbiota-recovery treatments may become standard

approaches for managing dysbiosis-related diseases [16]. Although probiotics, prebiotics, and synbiotics show promise in regulating the intestinal microbiota and reducing obesity risk, existing evidence is primarily derived from animal models; further human studies are necessary to substantiate these effects and support their clinical adoption [22].

Future Directions in Research

To advance understanding of the links between obesity and gut microbiota, two research priorities have been identified [8]. First, extensive population-based longitudinal studies are needed to characterize temporal variations in microbial composition and to document precise mechanistic effects on host biology [2, 17]. Second, the potential of gut microbial reshaping as a therapeutic strategy in obese people should be evaluated [11]. With ongoing research into the connections between the gut microbiota and obesity, a platform is emerging for accelerated progress [7]. Therapeutic intervention in human metabolic disease through targeted manipulation of the gut microbiota, an especially relevant approach to obesity and other illnesses often exacerbated by obesity, is a promising area of scientific inquiry [1, 3]. Such a microbiome-based strategy could include (but is not limited to) dietary modification, probiotics, prebiotics, genetically engineered dominant species, tailored antibiotics, fecal microbiota transplantation and the colonization of technical microdevices [5]. Yet fundamental advances are needed before the vast potential of the microbiome can be realized. Establishing causality and identifying key organisms and underlying physiological mechanisms are fundamental steps on this path [5]. Additionally, it is essential to consider the argument that the gut microbiota may only play a minor role in mediating the effects of obesity and may be less tractable for therapeutic intervention than other pathways [22].

Longitudinal Studies

Human gut microbiota contains many thousands of bacterial strains, with the highest numbers of both bacteria and diversity found in the colon [17]. The microbiome is sensitive to environmental and host factors, whose influences can vary with time and can, co-vary and interact in complex ways [10]. Hence, compounding and independent confounding factors could drive the differences observed in longitudinal studies of the links between gut microbiota and obesity [17]. Longitudinal studies observed that gut microbial diversity increased within the first 3 months after bariatric surgery and remained high after one year [11]. Certain microbial genera, such as Gammaproteobacteria, were more abundant post-surgery, while others like specific Firmicutes species decreased. Changes in the Firmicutes/Bacteroidetes ratio correlated with BMI, increasing up to BMI >33 and decreasing thereafter [13]. Microbiota composition also varies with gender and BMI. Longitudinal studies highlight dynamic changes in gut microbiota associated with obesity and weight loss interventions [17].

Personalized Medicine

Weight loss treatments have a broad spectrum of results and are often ineffective. According to different studies, gut microbiome profile associates with weight changes and may, thus, be used as a therapeutic target [26]. The potential role of gut microbiota to predict the success of a weight loss program has been investigated in 500 elderly individuals with overweight/obesity and metabolic syndrome, revealing its usefulness in the design of individual strategies to induce a lasting weight loss [23]. Since gut microbiome profile and gene expression associate with weight loss trajectories, RCTs are needed to investigate whether therapeutic modulation of gut microbiota may be of value to improve the results of life-style interventions in weight management. Gut microbiome composition and activity associate with inter-individual differences in body-weight regulation, suggesting its potential effects as a target of preventive and treatment interventions for obesity and cardiovascular diseases [17]. The potential role of the gut microbiome as a determinant of success in a behavioral weight-loss intervention has also been investigated in an exploratory study with diet-induced obese rats [12]. Personalized weight loss interventions based on nutrigenetics consider genetic markers such as single-nucleotide polymorphisms and DNA methylation to better understand the genetic regulation of obesity development [25]. Gene-diet interactions provide key information for precision nutrition, which aims to help prevent and manage obesity-related chronic diseases [14]. Advances in genotyping technologies and bioinformatics have paved the way for the implementation of nutrigenetics-based personalized approaches and the design of preventive protocols, especially when used in conjunction with other omics strategies, for a comprehensive overview of biological processes [13].

Public Health Implications

Obesity has become a major global public health concern [24]. Understanding the role of the microbiome has important implications for public health measures that can be implemented to prevent or reduce obesity at the population level, in light of current evidence [7]. A better understanding of the relationship between obesity and gut microbiome may give rise to low-cost and non-invasive interventions for affected individuals [3].

Challenges in Research

Methodological limitations complicate clinical studies on obesity and gut microbiome. Sufficient statistical power and sample size must be carefully calculated [7]. Long-term studies are necessary to validate the associations between the gut microbiome and obesity [5]. Variability in results arises from data sparsity, experimental noise, measurement errors, and inaccuracies during data-processing and annotation [8]. Ethical concerns must be addressed before designing microbiome-based interventions [9]. These challenges slow the development of microbiome-based treatment strategies for obesity [2].

Variability in Microbiome Composition

Obesity is among the most prevalent public health problems worldwide [20]. Due to its recognized impact on physiopathology, lifespan, and population health, obesity has been thoroughly studied over the last decades, as unveiled by the growing number of research publications associated with this disorder [24]. Since 2006, the existence of a correlation between microbial composition and the development of obesity was proposed. Ever since, many efforts have focused on the comprehension of the mechanisms behind such association [25]. Meanwhile, gut microorganisms have been considered a crucial biological factor of obesity [17]. Global patterns of microbial gene expression within the human distal gut revealed an overrepresentation of metabolic functions related to the absorption of monosaccharides and lipids in obese individuals.

Ethical Considerations

The constant progression of gut microbiome research [19] continues to reveal new perspectives, which lead to complex challenges in defining ethical boundaries and accelerating technological development responsibly. Recent evidence on the link between the gut microbiome and obesity further emphasizes the urgency of tackling these ethical challenges. One major challenge lies in obtaining informed consent [25]. Most Americans already exhibit poor levels of understanding of microbiome science [7], which makes it difficult to communicate the level of complexity and uncertainty involved in commercial microbiome analyses. Regarding proprietary ownership, the Human Microbiome Project concluded that participants do not view microbiome data as their exclusive property and are willing to share it for the advancement of science [26]. However, it remains debated what an individual loses when microbiome data are commercialized.

CONCLUSION

The intricate interplay between obesity and the gut microbiome represents a frontier in metabolic and clinical research. The gut microbiota significantly influences host metabolism through mechanisms involving energy extraction, lipid metabolism, bile acid transformation, and immune modulation. Dysbiosis has emerged as a key contributor to obesity, with evidence linking altered microbial composition to inflammation and impaired metabolic regulation. Longitudinal and interventional studies demonstrate that modulating the gut microbiome through diet, probiotics, prebiotics, or FMT can improve metabolic outcomes and weight management, emphasizing its therapeutic potential. However, significant challenges persist. Variability in microbiome composition, methodological inconsistencies, small sample sizes, and lack of long-term data continue to hinder definitive conclusions. Furthermore, ethical and legal issues concerning consent, privacy, and data ownership demand urgent attention as microbiome research increasingly intersects with biotechnology and personalized health industries. Despite these barriers, the integration of nutrigenetics, microbiomics, and precision medicine is paving the way for tailored obesity management strategies. Future research must focus on identifying causal microbial pathways, standardizing analytical protocols, and evaluating the long-term safety of microbiome-based interventions. Public health policies should incorporate microbiome knowledge to develop preventive, low-cost, and non-invasive strategies against obesity. By uniting scientific rigor, technological innovation, and ethical responsibility, the study of the gut microbiome offers a transformative framework for understanding and combating obesity, one that moves from generalized treatments toward personalized, evidence-based care for global metabolic health.

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