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Role of Gut Microbiota in the Development of Obesity and Diabetes

Preeti Verma[1]*

[1*]Shri Shankaracharya Institute of Pharmaceutical Sciences and Research, Shri Shankaracharya Professional University, Bhilai Durg, CG, Pin - 490040

***Corresponding Email: preeti.verma1110@gmail.com**

Abstract

The gut microbiota is a complex and dynamic community of trillions of microorganisms that live in the gastrointestinal tract, has turned out to be a key controller of metabolism, immunity, and general health of the host. There is a growing body of evidence that evidence of the dysbiosis or changes in the gut microbial composition is a major contributor to the onset and severity of obesity and diabetes in animal models. These disruptions affect energy collection, fat and glucose homeostasis, and systemic inflammation, which brings to focus the microbiota as an energetic determinant of metabolic well-being. Mechanistic studies prove the functions of short-chain fatty acids (SCFAs) in maintaining intestinal barrier integrity and glucose metabolism, and endotoxin-mediated metabolic endotoxemia and immune signaling cascade aggravate insulin resistance and chronic inflammation. Probiotics, prebiotics, fecal microbial transplantation (FMT), dietary therapy, and pharmacological agents are all experimental treatments used to restore microbial balance and improve the metabolic outcomes in controlled animal experiments. Although these findings are encouraging, it is difficult to apply them to humans because of inter-species variations, changes with environments and diets, and doubts over long-term effectiveness. The therapeutic potential of the gut microbiota in preventing and managing obesity and diabetes requires the integration of longitudinal human studies with multi-omics and personalized microbiome-based strategies in the future to tap into the potential of microbiota.

Key Words:

Gut Microbiota, Obesity, Diabetes, Dysbiosis, Short-Chain Fatty Acids, Fecal Microbiota Transplantation, Inflammation, Energy Metabolism.

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1. INTRODUCTION

The gut microbiota, a dynamic and intricate community of trillions of microorganisms that dwell in the gastrointestinal tract has become important in the regulation of host metabolism, immunity and general wellbeing. The importance of gut microbiota in the pathogenesis of metabolic diseases, especially obesity and diabetes, in the last 20 years has been emphasized[1]. In addition to genetic predisposition and other lifestyle factors including diet and exercise, the structure and functionality of gut microbes have been reported to play major roles on energy balance, glucose

regulation, fat storage, and systemic inflammation. It has been discovered through animal research and human clinical studies that disruptions in the intestinal microbial ecosystem, commonly known as dysbiosis, may predispose an individual to metabolic illnesses by modifying host-microbe interactions and facilitating metabolic endotoxemia.

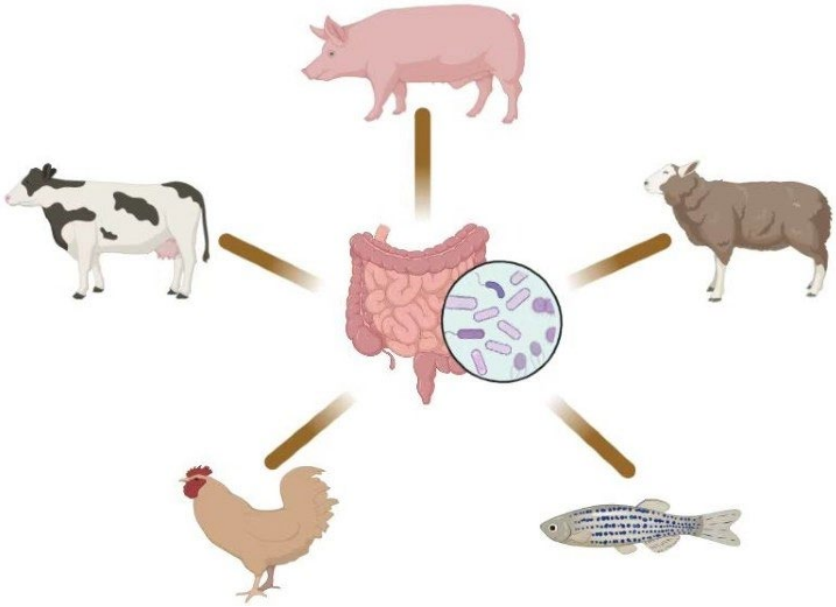

Figure 1: Gut Microbiota[2].

These findings represent a paradigm shift in the perception of obesity and diabetes, as they are more than the diseases of energy imbalance, but to the close association of the two with the gut microbial composition and activity. As these metabolic diseases and their attendant complications continue to increase all around the world, there has been some need to consider new approaches beyond the orthodox dietary and pharmacological measures[3]. The microbiota of the gut provides a promising target therapy, because its composition is very much dynamic and can be altered by diet, probiotics, prebiotics, fecal microbiota transplantation, and pharmaceuticals. Therefore, the exploration of the connection between the microbiota of the gut and the development of the metabolic diseases has the potential to contribute to the further growth of scientific knowledge as well as to the creation of new therapeutic methods.

1.1. Background Information and Context

Obesity and diabetes are two of the most urgent health issues facing the world and the prevalence rates of the two are increasing at disastrous rates in developed and developing world. Conventionally, lifestyle behaviors, genetic predisposition, and environmental factors have been identified as their pathogenesis[4]. Recent studies have however shown that gut microbiota plays a key role in the regulation of metabolism affecting energy harvesting of the diet, lipid metabolism, glucose homeostasis and immune regulation. Changes of microbial diversity and composition have been systematically connected to weight gain, insulin resistance, and systemic inflammation which can inform about the underlying mechanisms of those conditions.

1.2. Objectives of the Review

This review aims to:

- To examine the role of gut microbiota in regulating host energy metabolism, fat storage, and glucose homeostasis in obesity and diabetes.
- To analyze how microbial dysbiosis and altered microbial diversity contribute to insulin resistance, systemic inflammation, and metabolic dysfunction.
- To investigate the impact of microbial metabolites, particularly short-chain fatty acids (SCFAs), on gut barrier integrity, immune modulation, and metabolic regulation.
- To evaluate the effectiveness of therapeutic interventions, including probiotics, prebiotics, fecal microbiota transplantation, diet, and pharmacological agents, in modulating gut microbiota and improving metabolic outcomes in animal models.
- To identify gaps in current animal model research and suggest future directions for translating microbiota-based therapies to human obesity and diabetes management.

1.3. Importance of the Topic

Due to the growing problem of obesity and diabetes in the global population, the role of gut microbiota in the pathogenesis of the mentioned diseases is of utmost significance. The gut microbiota is a changeable aspect of human biology unlike fixed genetic factors, thus presenting a distinct chance of prevention and treatment[5]. By explaining the mechanisms of microbiota in animal models, researchers can open the road to specific interventions that can be used to supplement the current lifestyle and pharmacological therapies. So, it is possible to conclude that this review emphasizes the paramount role of gut microbiota as a diagnostic indicator, as well as a therapeutic objective in combating metabolic diseases.

2. ANIMAL MODELS AND EXPERIMENTAL APPROACHES IN GUT MICROBIOTA–METABOLISM RESEARCH

The animal models have played a central role in the discovery of the role of gut microbiota in obesity and diabetes with various studies examining the effects of microbial composition and products directly in relation to fat storage, glucose regulation, and systemic inflammation using germ-free mice, fecal microbiota transplantation (FMT), feeding on a high-fat diet and exposure to endotoxins[6]. Other methodologies that include 16S rRNA sequencing, metabolomic profiling and knock out models can further explain the important role of particular bacteria taxa, metabolites such as SCFAs, and immune pathways in promoting metabolic health and disease progression. These methods offer manipulable and controlled structures of causal inference but cannot be directly translated to humans because of differences in microbiome composition, functional diversity, and inability of the laboratory environment to recreate all the complexities of the human environment, diet, and lifestyle and require careful interpretation of results.

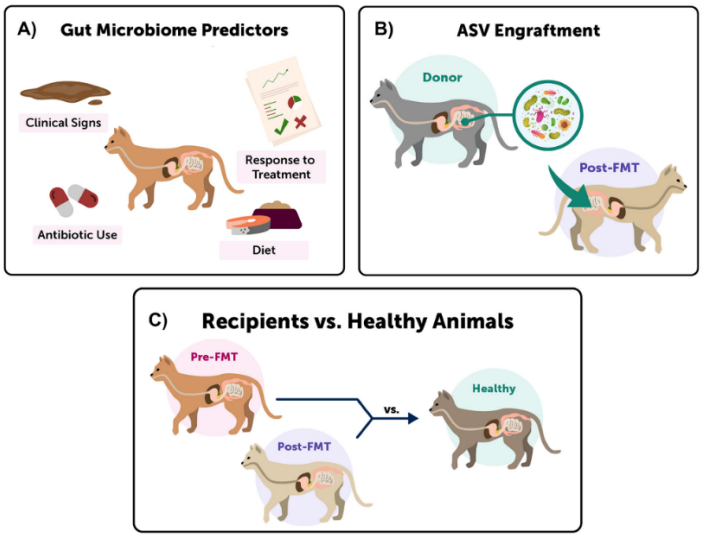

Figure 2: Fecal Microbiota Transplantation (FMT)[7].

2.1. Key Research Studies

Rodent models, such as endotoxin exposure, germ-free studies, FMT and feeding on a high-fat diet demonstrate that gut microbiota closely determine fat storage, glucose metabolism and inflammation. These results indicate dysbiosis of the gut and microbial products as causes of obesity and diabetes.

- **Germ-free mouse studies:** Germ-free mice have been used to give important insights on the role of gut microbiota in obesity and diabetes. Examining these mice colonized using microbiota of obese donors, it is observed that these mice have much higher fat accumulation than mice colonized using microbiota of lean donors[8]. Interestingly, this is done despite the fact that the caloric intake is similar between groups and therefore it can be noted that the composition of the gut microbiota is capable of having an independent effect on the energy harvest, fat storage and metabolic control.
- **Fecal microbiota transplantation (FMT) in rodents:** FMT experiments also presuppose the causal role of gut microbiota in metabolic disorders. Transplantation of microbiota of diabetic or obese rodents to germ-free mice results in defective glucose tolerance and insulin resistance in the recipient[9]. This is good evidence that microbial communities in themselves have the power to transmit metabolic disease phenotypes, which makes gut dysbiosis an important cause of obesity and diabetes.
- **High-fat diet (HFD) models:** Animals on high-fat diets (HFD) exhibit distinctive changes in the gut microbial composition, which consist of an influx of Firmicutes and a decline of Bacteroidetes. Such shifts of microbes are always associated with excessive weight gain, chronic grade-low inflammation, and glucose metabolism impairment[10]. These models are highly similar to changes in microbiota in obese and diabetic humans, and they are accordingly applicable to mechanistic studies.
- **Endotoxin research:** Lipopolysaccharides (LPS), which is one of the significant products of the outer membrane of Gram-negative gut bacteria, is a significant factor in

metabolic dysfunction. In mouse studies it has been shown that LPS has the capability of inducing metabolic endotoxemia that allows systemic inflammation, disturbs the insulin signaling and increases the speed of insulin resistance[11]. This is an indication of the significance of gut-derived inflammatory mediators in complications of obesity.

2.2. Methodologies and Findings

Such methods as 16S rRNA sequencing, metabolomic profiling, and knockout models in rodents demonstrate how gut microorganisms and their metabolites affect obesity, diabetes and inflammation. These ways connect microbial population and host immune pathology to metabolic health.

- **16S rRNA sequencing:** 16S rRNA gene sequencing is one of the most popular techniques in rodent research studies that has allowed the community to describe changes in gut microbial communities of different conditions (high-fat diets, germ-free)[12]. The method can be used to identify the taxa of bacteria related to obesity, diabetes, and metabolic inflammation.
- **Metabolomic profiling:** Metabolomic studies have shown that there is major change of microbial metabolites, especially short-chain fatty acids (SCFAs). The rodent models prone to diabetes usually exhibit low butyrate concentration, a metabolite that has been shown to have anti-inflammatory and insulin-sensitizing effects[13]. These results relate the microbial functional capacity to the metabolic health of the host.
- **Knockout models:** Knockout models Genetically modified mice including TLR4-deficient models have been used to an important role in the study of host microbe interactions. Such mice have lower systemic inflammation and are resistant to insulin resistance caused by diet[14]. These kinds of results indicate that microbial products influence innate immune pathways which play a crucial part in the development of metabolic disorders.

2.3. Strengths and Weaknesses

The animal models offer the control and manipulation of microbiota and allow the robust causal understanding of metabolism. Nonetheless, the discrepancy between human microbiomes and simplified laboratory conditions makes it impossible to apply findings directly to human beings.

- **Strengths:** Animal models have special benefits to the investigation of gut microbiota and metabolic disease. There is less variability in controlled diets and genetic homogeneity, which enable researchers to isolate the effects of microbiota composition on host metabolism[15]. Moreover, the control of microbial communities via germ-free, FMT or antibiotics gives an effective experimental base to cause inference.
- **Weaknesses:** Animal models have weaknesses because they have been shown to be limited in extrapolating findings to humans despite their value. Microbiomes of rodents and human gut differ significantly in terms of composition and functional diversity, and

this may need to be considered when extrapolating to human populations[16]. Moreover, laboratory settings cannot model the complexity of human environmental, dietary and lifestyle factors, and simplify the processes of disease. Such restrictions indicate that care should be taken when generalizing animal model results to human settings.

3. MECHANISTIC INSIGHTS INTO GUT MICROBIOTA–MEDIATED METABOLIC DYSFUNCTION

Microbiota in the guts have shown to play a major role in obesity and diabetes by a variety of mechanisms, such as increasing energy availability, maladaptive microbial ecology, control of SCFA, endotoxemia, and host/microbe immunology. Experiments of germ-free and donor colonization demonstrate that microbiota derived out of obese enhance fat storage efficiency, and models of a high-fat diet demonstrate dysbiosis with fewer beneficial SCFA-producing species and more pro-inflammatory bacteria, inducing insulin resistance and malfunctioning metabolism[17]. SCFAs like butyrate are protective in glucose metabolism and gut barrier integrity, but endotoxin -mediated metabolic endotoxemia is pro-inflammatory and destructive of insulin signaling. Genetic and knockout models also support the fact that innate immune pathways, gut barrier functionality and host defense mechanisms play a critically important role in shaping microbiota host interactions. All these results put gut microbiota at the center of metabolic health regulation, and there is a therapeutic potential of microbial regulation of obesity and diabetes.

3.1. Gut Microbiota and Energy Harvest in Obesity

Gut microbiota has been proven in rodent studies as having a direct effect in the host energy metabolism and fat storage. Making the germ-free mice colonized with microbiota of an obese donor demonstrate increased ability to extract the energy of the same diet compared to the mice colonized with microbiota of a lean donor[18]. This enhancement of efficiency is attributed to the induction of microbial genes that cause degradation and fermentation of heavy carbohydrates resulting in the generation of more metabolites that may be used by the host. Consequently, obese-derived microbiota cause mice to gain more fat mass even when they gain the same amount of food, thus the microbiota being a determinant of metabolic efficiency.

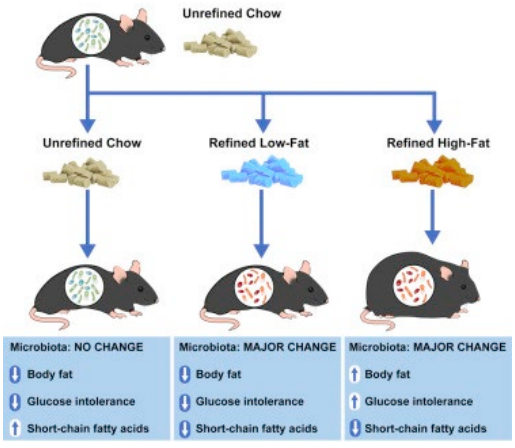

Figure 3: Gut Microbiota and Energy Harvest in Obesity[19].

Mechanistic research has found that certain bacterial phyla, including Firmicutes, are increased in obese microbiomes and are implicated in an increased production of absorbable energy sources. Such microbial communities do not only augment the energy collection however they also modify lipid catabolism, altering the fat storage ways in the liver and adipose tissue. Altogether, these results highlight the idea that obesity is not a mere effect of a caloric composition and physical operations but also largely contributed to the microbiota-mediated metabolic processes. This makes the microbiome a key contributor to the obesity pathogenesis and opens the prospects of microbiota-directed therapeutics.

3.2. Microbial Dysbiosis and Insulin Resistance

Obesity and diabetes animal models have shown that insulin resistance is closely related to microbial dysbiosis or the lack of balance and diversity in the gut microbiota. Rodent studies of high-fat diet (HFD) show the same changes in the composition of the gut microbial pool, where beneficial commensal species are decreased, and pro-inflammatory microbes are increased. Such changes are also linked to the decline in microbial diversity which undermines ecosystem stability and resilience[20]. Dysbiotic microbiota worsen the metabolic dysfunction by modifying the intestinal permeability, facilitating systemic inflammation, and disrupting the insulin signaling pathways.

Moreover, dysbiosis seems to establish a feed-forward mechanism that carries on metabolic malfunction. Growth of Gram-negative endotoxin-producing microbes increases the amount of lipopolysaccharides (LPS) circulating and stimulates innate immune responses and causes chronic low-grade inflammation - a major contributor to insulin resistance[21]. Concurrently, the microbes that are beneficial and help bring about metabolic balance are reduced including the SCFA-producing bacteria. Accordingly, the microbial imbalance of HFD-fed rodents does not only reflect clinical evidence in humans with metabolic syndrome, but also offers a mechanism of explanation of how dysbiosis contributes to the development of obese mice into diabetic mice.

3.3. Role of SCFAs in Metabolic Regulation

The microbial product of fermentation produced by microbes (mostly acetate, propionate, and butyrate) are referred to as short-chain fatty acids (SCFAs). SCFA is vital in sustaining metabolic wellness. SCFAs were found to control glucose homeostasis in rodent models by various pathways which include the release of gut hormones like GLP-1 and PYY, insulin sensitivity, and the hepatic gluconeogenesis. Particularly, butyrate is a vital energy source of colonocytes, and it is important to maintain the integrity of the intestinal barrier[22]. Reduction in SCFA production, as seen in diabetic or dysbiotic rodent models, impairment of gut barrier functions, and consequently allowing bacteria components into circulation (and leading to inflammation) takes place.

Immune responses are also protective functions of SCFAs. They are signaling molecules, which control the expression of genes by inhibiting histone deacetylase and activating G-protein-coupled receptors. A lower abundance of butyrate-producing bacteria like Faecali bacterium

prausnitzii in diabetic-sensitive rodents is associated with an increase in inflammation and poor insulin sensitivity[23]. On the other hand, dietary treatment or probiotic supplements which reestablishes SCFA generation have been linked to enhanced glucose metabolism and diminished adiposity. Such results underscore the role of SCFAs as major mediators between the activities of microbes of the gut and host energy regulation and emphasize their role in the treatment of obesity and diabetes.

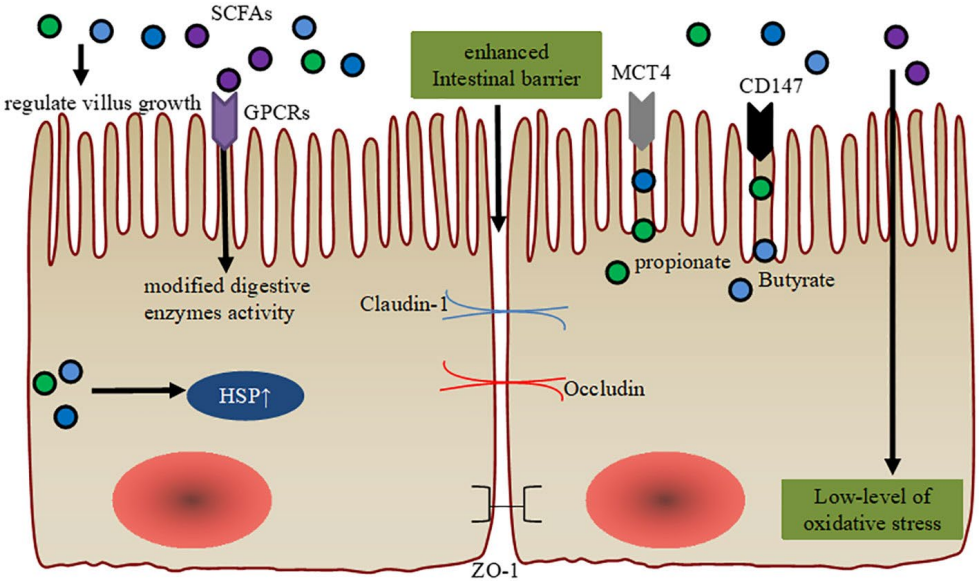

Figure 4: Short-chain fatty acids (SCFAs)

3.4. Endotoxemia and Inflammation

The lipopolysaccharides (LPS) of Gram-negative intestinal microbes have become a key mediator of the connection between gut microbiota and systemic metabolic dysfunction via endotoxemia. Chronic exposure to low concentrations of LPS in rodent studies leaves behind metabolic endotoxemia, which is a low-grade inflammation[24]. This inflammation disrupts insulin signaling in major metabolic tissues such as liver, muscle, and adipose tissue, which results in the insulin resistance and the weight gain. In addition, LPS may trigger Toll-like receptors (in particular, TLR4), which leads to the onset of inflammatory cascades only worsening metabolic disorders.

The effects of LPS are also supported by experimental studies to cause the progression of obesity and diabetes. An example of this is the use of antibiotics to cause a decrease in Gram-negative populations or the use of interventions that promote the integrity of the gut barrier that will effectively reduce the levels of circulating LPS. These modifications are linked to decreased inflammation, augmented glucose tolerance as well as augmented insulin sensitivity in rodents. Taken together, these findings put endotoxemia as a high-order relationship between microbial dysbiosis, immune activation, and metabolic disease into the limelight, which makes strategies that decrease the LPS burden or block its signal transduction a potentially useful therapeutic option.

3.5. Genetic and Knockout Models

Genetic and knockout mouse models have helped give potent instruments to unravel the host-microbe interactions in obesity and diabetes. Mice with defects in innate immune signaling (TLR4, MyD88) have shown unprecedented resilience to the breakdown in metabolic function induced by high-fat diet[25]. It is found that these mice have less inflammatory response, better insulin responses, and less susceptible to weight gain proving that microbial recognition pathways are key mediators of host metabolic outcomes. The results of such findings can be used as a direct indication that immune system-microbiota crosstalk promotes the development of obesity-associated metabolic conditions.

In addition to immune signaling, other genetic models have enlightened the complicated roles of host factors in the determination of the outcomes related to the microbiota. Mice that lack gut barrier proteins or antimicrobial peptides, as an example, respond excessively to microbial dysbiosis, and these observations point to the significance of host defense in supporting metabolic equilibrium[26]. All these models focus on the fact that interaction between microbiota and host is a two-way process: microbes can have the effect on metabolic health, but the degree of predisposition or resistance to obesity and diabetes depends on the genetic and immune characteristics of the host. These understandings lead to accuracy medicine strategies that address host-microbiome dynamics.

4. THERAPEUTIC MODULATION OF GUT MICROBIOTA IN ANIMAL MODELS

The therapeutic interventions on gut microbiota have become the focus of considerable attention as the possible means of obesity and diabetes prevention and treatment. Probiotics and prebiotics are one of the commonest methods of study in the animal model. Probiotics are live beneficial bacteria strains (Lactobacillus and Bifidobacterium strains) demonstrated to maintain microbial equilibrium, increase gut barrier functions, and decrease systemic inflammation[27]. Prebiotics on the other hand are nondigestible fiber that promotes the growth of the useful microbes and the consequent growth of short-chain fatty acid (SCFA) such as butyrate which enhances glucose metabolism and insulin sensitivity. Probiotics and prebiotics have been tested to have reduced fat accumulation, lower inflammatory markers and better glycemic control and have proven to have therapeutic effects on rodents.

The other therapeutic option that is likely to succeed is fecal microbiota transplantation (FMT), in which microbiota of healthy or lean individuals are transplanted into obese or diabetic individuals. In rodents, FMT has been reported to correct dysbiosis, recoup microbial diversity as well as to improve metabolic parameters, such as glucose tolerance and insulin sensitivity. These findings are very strong indicators that the direct effect of modifying the microbial ecosystem on host metabolic health can be achieved[28]. Moreover, controlled doses of antibiotics have been implemented to decrease the population of harmful bacteria, including Gram-negative LPS-producing bacteria to reduce endotoxemia and enhance the metabolic outcomes. Nevertheless, these influences are frequently short-term, and it is necessary to find methods that will re-model the gut microbiome in a sustainable manner.

The dietary and pharmacological approaches also have significant roles in the gut microbiota modulation in animal models. Diets supplemented with polyphenols, omega-3 fatty acids, or resistant starches were reported to increase the diversity of microbes, improve beneficial metabolites, and increase insulin sensitivity[29]. On the same note, certain medications like metformin which is widely used to treat type 2 diabetes have been observed to have section of their therapeutic effect on gut microbiota modification. Spurring the development of SCFA-forming bacteria and inhibiting pathogenic microorganisms, metformin proves how conventional pharmacological interventions can be used in combination with microbiota-mediated processes.

All of these studies on animals lead to the conclusion that gut microbiota is a promising target in suppressing obesity and diabetes. Researchers can be able to control the metabolic results in a controlled environment by manipulating microbial composition and activity using probiotics, prebiotics, FMT, antibiotics, diet, or drugs[30]. Although caution is needed when applying complex and variable human microbiome to animal models, there is substantial evidence that the gut microbiota is a controllable variable with extensive clinical consequences on the treatment of metabolic diseases.

Table 1: Summary of Key Studies on Gut Microbiota, Obesity, and Diabetes

Author(s) & Year	Study	Focus Area	Methodology	Key Findings
Singer-Englar, Barlow & Mathur (2019)[31]	Updated review on gut microbiome, obesity, and diabetes	Gut microbiota alterations in obesity and diabetes	Literature review of human and animal studies	Dysbiosis contributed to energy imbalance, insulin resistance, and low-grade inflammation; microbiome-targeted interventions (probiotics, dietary modifications) have therapeutic potential
Stephens, Arhire & Covasa (2018)[32]	Gut microbiota as a metabolic organ	Role of gut microbiota in energy metabolism and obesity	Review of experimental studies in humans and rodents	Gut bacteria actively influence digestion, energy harvest, and fat storage; shifts in microbiota predispose to weight gain and metabolic disorders
Sun et al. (2018)[33]	Insights into gut microbiota in obesity	Mechanisms and therapeutic perspectives	Literature review on pathogenesis and interventions	Dysbiosis disrupted metabolic pathways and gut barrier integrity, triggered systemic inflammation; dietary interventions, prebiotics, probiotics, and FMT could restore microbial balance and improve metabolic health
Tanase et al. (2020)[34]	Gut microbiota and microvascular complications in T2DM	Microbiota's role in diabetic complications	Review of human and experimental studies	Dysbiosis exacerbated nephropathy, retinopathy, and neuropathy via inflammation, oxidative stress, and impaired glucose metabolism; microbiota modulation may prevent or slow T2DM complications
Vallianou et al. (2019)[35]	Gut microbiome and microbial metabolites in	Role of microbial metabolites	Literature review	SCFAs regulate appetite, insulin sensitivity, and inflammation; evidence supports microbiota's role in obesity but

	obesity	(SCFAs) in metabolic regulation		further mechanistic studies are needed for causality and therapy optimization

5. DISCUSSION

Cumulative proof supplied by the animal model and studies indicate that gut microbiota are essential in the pathogenesis of obesity and diabetes, energy metabolism, glucose, and systemic inflammation. The findings do not only further our knowledge of host microbe interactions, but they also offer a model, which can be used to treat the microbiome[36]. Yet in the face of these developments, there are some fundamental questions as to how the microbial effects are produced, whether these effects differ across species, and how far animal results can be extrapolated to human clinical practice.

5.1. Interpret and Analyze the Findings

The analysed articles collectively demonstrate that gut microbiota have a metabolic health impact via various mechanisms, such as increased energy harvesting, SCFA homeostasis, endotoxin elicited inflammation, and immune crosstalk. Germ-free and FMT experiments give causal support to the fact that microbiota composition could be able to transmit phenotypes of metabolic diseases[37]. The models of high-fat diets always emphasize microbial dysbiosis as a cause of insulin resistance, and the role of innate immune signals in mediating the effect of these interventions has been verified by knockout models. Notably, therapeutic measures, like probiotics, prebiotics, FMT, and pharmacological control of the microbiota, were promising in the restoration of the microbial balance and the enhancement of metabolic parameters in animal models.

5.2. Discuss Implications and Significance:

These data indicate that intestinal microbiota is not just a passive observer, but rather an active controller of host metabolism, and it has a potential as a therapeutic object. As the microbiome is dynamic and receptive to interventions, restorative interventions to microbial homeostasis may be used as complementary to traditional lifestyle and pharmacological management of obesity and diabetes[38]. It has great public health consequences, as the metabolic disorders are increasing worldwide. Besides, the discovery of microbial biomarkers may contribute to the early diagnosis and individualized treatment plans.

5.3. Highlight Gaps and Suggest Future Research Directions:

Although these findings are promising, there are a number of gaps that restrict the use of animal research on human beings[39]. The variability of the microbiome composition, foods, and environmental contact of rodents and humans makes direct comparison difficult. In most research, it has been conducted in a controlled laboratory, which simplifies the metabolic environment of the human body. Moreover, the microbiota-targeted therapies have not been studied in terms of their long-term outcomes and sustainability. Future science must be based on the large-scale, longitudinal human studies, and the multi-omics approach should be integrated

to reveal the functional pathways between microbiota and metabolism[40]. Also, investigations of host genetic variation, individual-based microbiome-based treatment, the safety and efficacy of treatment, including FMT, in the clinical population will also be essential in developing this area.

6. CONCLUSION

The accumulating evidence in the literature of animal model studies highlights the primary role of gut microbiota in the pathogenesis and course of obesity and diabetes. The gut microbial composition and diversity have effects on host energy harvesting, fat storage, glucose regulation, and systemic inflammation, where dysbiosis and decreased production of SCFA promote insulin resistance and metabolic dysfunction. The experimental methods in the form of probiotics, prebiotics, fecal microbiota transplantation, dietary manipulations, and pharmacological agents demonstrate that it is possible to control the microbiome and achieve better metabolic results. Although these data are very informative regarding the mechanistic insights and should position gut microbiota as an effective therapeutic option, translation to human should consider the interspecies differences, environmental variability and sustained effectiveness. The combination of longitudinal human research, multi-omics, and individual-level use of microbiomes will be crucial in terms of achieving the complete potential of gut microbiota to prevent and treat metabolic disorders.

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