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REVIEW ARTICLE

Gut Microbiota and Diabetes Mellitus: Insights into Type 1 and Type 2 Diabetes: A Review

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ABSTRACT

Type 1 diabetes T1D and type 2 diabetes T2D, two metabolic disorders that are common throughout the world, have different pathophysiologies but increasingly similar characteristics. Recent findings demonstrate how important the gut microbiota is in regulating immunological and metabolic processes associated with both forms of diabetes. In light of host-related variables such age, sex, genetics, method of birth, nutrition, and antibiotic use, this study investigates the role that microbial dysbiosis plays in the development and progression of T1D and T2D. Different microbial signatures linked to diabetes states have been found in both human and animal investigations. These signatures are frequently characterized by decreased diversity and an imbalance between pro-inflammatory and beneficial bacteria. Additionally, included are the potential therapeutic benefits of microbiota-targeted therapies, such as dietary modification, fecal microbiota transplantation [FMT], probiotics, prebiotics, symbiotic, and medications like metformin. Novel approaches to diabetes prevention, diagnosis, and treatment through microbial manipulation are made possible by an understanding of the gut-diabetes axis.

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1- INTRODUCTION

According to the World Health Organization [WHO], diabetes is a collection of metabolic illnesses marked by persistently high blood sugar levels that can harm many organs, particularly the kidneys, heart, blood vessels, nerves, and eyes, and ultimately cause malfunction [1]. Type 1 diabetes (T1D) and type 2 diabetes (T2D) are two types of diabetes that can be identified. Patients with T1D and T2D are classified according to different criteria, including age at disease onset, excess weight, level of insulin resistance IR, metabolic syndrome (MS), degree of pancreatic β-cell function loss, presence of specific autoantibodies linked to β-cell destruction, presence of a systematic subclinical inflammatory state, blood levels of C-peptide, and need for insulin treatment to survive [2]. T1D patients are usually described as young, thin individuals who have lost 90–100% of their β-cell activity and require insulin therapy from the beginning of the disease due to a direct absence of insulin secretion and synthesis. Nonetheless, a person with type2 diabetes is typically described as an older, obese, or overweight person who develops insulin resistance, which results in β-cell dysfunction and, ultimately, insulin insufficiency. Oral antidiabetic medications can be used to treat patients with various conditions, particularly cardiovascular disorders, early in the course of the disease, while insulin treatment is required later. However, it is currently difficult to

differentiate between these two main forms of diabetes, and a person may have both T1D and T2D characteristics [3]. Both T1D and T2D have been linked to dysbiosis, a disruption in microbial composition, which raises the possibility that the gut microbiota could be a useful diagnostic marker and treatment target [4,5].

1. Overview of gut microbiota

1.1 What is microbiota

A varied colony of bacteria known as the gut microbiota performs a number of tasks that affect the host's general health. These include natural defense against infection, immune system modulation, and nutrient metabolism. Certain bacteria are linked to inflammatory chemicals that can cause inflammation in different bodily tissues. Numerous chronic multisystem diseases, such as obesity, atherosclerosis, and type 2 diabetes mellitus, are caused by inflammation. [6]

The bacteria that live in the human gastrointestinal system are referred to as the gut microbiota. Prokaryotic species—which include bacteria and other unicellular organisms without specialized organelles—make up the majority of this dynamic ecosystem. Fungi, parasites, and archaea are also present, albeit to a lesser degree. Another component of this environment is viruses. The gut microbiome is the term used to describe the genetic and functional characteristics of microbial species [7]. Humans can be categorized more broadly based on their enterotypes, which are distinctively similar gut microbial assemblages found in particular groups [8]. Comparing the 3.3 million genes found in a single individual's gut microbial species to the roughly 23,000 genes found in the human genome highlights the size and potential impact of these species on human health [9].

1.2 Factors associated with microbiota composition

During infancy, the gastro-intestinal [GI] tract's gut microbiota develops. Microbial colonization depends on several host variables, which are explained in more depth below. Furthermore, the GI tract's various sections have distinct chemical conditions that favor the growth of particular microorganisms over others. Throughout early development, diversity and stability keep growing until they attain a specific composition as people age [10]. Numerous variables and life events may influence its makeup, which could work in concert to increase diversity [11]. Although there have been occasional instances of increasing diversity in specific disease states, it is crucial to remember that in this case, greater diversity is typically linked to better health outcomes [12,13]. The inter-individual variance we see is caused by factors that affect the gut environment, which in turn contribute to the distinctive gut microbial community. In newborns, the initial colonization of the gut microbiota is greatly influenced by the route of delivery. The newborn is exposed to the mother's vaginal bacteria during vaginal births, resulting in a microbiota composition that reflects the vaginal area and includes genera such as Lactobacillus, Prevotella, and Sneathia [14]. Babies born by caesarean section [C-section], on the other hand, are exposed to the skin germs of their mothers, which results in a less varied microbiota with a higher concentration of skin-associated bacteria, including species of Staphylococcus, Corynebacterium, and Propionibacterium [14,15]. Furthermore, the neonatal gut microbiota is further impacted by the administration of antibiotics during C-section delivery [16]. In general, newborns born vaginally have a microbiota that is more comparable to that of their mothers than those born via cesarean section [17]. These variations in microbiota could contribute to the explanation of why infants born via cesarean section are more susceptible to allergies and illnesses [18, 19].

Early milk consumption has an effect on gut microbiota as well. Bifidobacterium species, which flourish on dietary fiber, dominate the microbiota supported by breast milk, which has its own bacteria and beneficial chemicals [20]. Infants who are fed formula, on the other hand, usually have more varied microbiotas with higher levels of Enterobacteriaceae species [15]. The advantages of breast milk may come from sources other than the microbiota itself, even if formula-fed babies have a wider variety [17]. Crucially, during the neonatal period, the gut microbiota experiences several changes, and the effects of feeding and delivery methods may only cause brief changes in the microbial makeup [21].

1.2.1 Diet

The gut microbiota's makeup is greatly influenced by dietary behavior; depending on the nutrients available, some bacterial strains may become dominant and have particular downstream consequences on the host[22]. The gut microbial community reorganizes when diets change because specific nutrients become unavailable. Bacteroides species, which flourish in environments high in protein, typically dominate the microbiomes of people who consume large amounts of animal protein. In contrast, people who eat a plant-based diet frequently have higher amounts of Prevotella, which is effective in breaking down complex fibers and carbs [23,

24]. This link is supported by ethnographic research, which demonstrates how collective enterotypes are influenced by cultural food trends. For instance, Bifidobacterium, a genus recognized for its capacity to create enzymes that break down starch, is found in higher concentrations in many Asian people whose diets are high in starch-heavy foods like rice. [25, 26]. The effects of the Western diet, which is marked by a high consumption of saturated fats, little fiber, and few unsaturated fats, on gut flora have been thoroughly investigated. It is connected to an increase in anaerobic bacteria, particularly Bilophila and Bacteroides, the latter of which has been linked to gut inflammation [27]. In addition to macronutrients, animal studies have demonstrated that chemicals like emulsifiers, artificial sweeteners, and preservatives—which are frequently included in Western processed foods—can change the composition of the microbiota [28]. These chemicals may raise the gut's inflammatory potential and decrease microbial diversity, highlighting the close relationship between gut health and nutrition [29].

1.2.2 Age

Age groups clearly differ in the composition of their gut flora. While major microbial phyla were present in all ages, their proportions varied, according to a cross-sectional study of 367 healthy people in Japan [ages 0–104]. Actinobacteria levels decreased with age, while Firmicutes levels increased after weaning, especially after age 4, indicating an inverse relationship. Although Bacteroidetes stayed mostly unchanged, there was a minor rise in those above 70 [30]. Lifelong factors that complicate the straightforward link between age and gut microbiota include immunological development, disease history, environmental microbial exposure, and hormone changes, all of which may have an impact on these patterns [30].

2.2.3 Sex

Despite being less researched, sex has also been found to have an impact on the composition of the microbiota. At the genus or species level, these distinctions are more apparent. A study conducted by Baars *et al*. Mice showed sex-specific variations in gene expression and microbial composition, which affected the metabolism of host lipids. Furthermore, Haro *et al*. discovered that whereas Bacteroides plebeius was more common in males, Bacteroides caccae was more common in females. Although further study is required to fully understand the causes, these sex-based changes in microbial populations seem to be related to hormonal impacts.

2.2.4 Body Mass Index [BMI]

Variations in body mass index frequently make sex-related microbiome differences more noticeable. In the same Haro *et al*. investigation, males with a BMI over 33 were shown to have lower Firmicutes levels than males with lower BMIs, but women's Firmicutes levels were consistently high regardless of BMI. Gut microbial diversity and BMI have generally been found to be strongly inversely correlated, with higher BMI being linked to lower diversity. Additionally, a greater Firmicutes-to-Bacteroidetes ratio is frequently associated with obesity and tends to equalize with weight loss.

2.2.5 Host Genotype

The diversity and organization of the gut microbiota are significantly impacted by host genetics. Microbial composition is influenced by immunological responses and disease susceptibility, which are influenced by specific gene variants. For example, people who have the APOA5 gene's rs651821 variant are more likely to have bacteria from the genera Lactobacillus, Sutterella, and Methanobrevibacter, which are associated with a higher risk of metabolic diseases. Similarly, through their impact on the composition of the gut microbiota, genome-wide association studies have connected genetic variations to a number of illnesses, such as obesity, schizophrenia, type 2 diabetes, amyotrophic lateral sclerosis [ALS], and inflammatory bowel disease [IBD]. The connection between microbial activity and obesity may be mediated by SLIT3, a gene of interest that has been linked to BMI and has been demonstrated to influence inflammation caused by microbes. Additionally, autoimmune conditions like ankylosing spondylitis are linked to variations in the human leukocyte antigen [HLA] system, such as HLA-B27. Distinct microbial profiles were seen in individuals with HLA-B27, suggesting a potential connection between microbiota-driven inflammation and genetic susceptibility. Current research indicates that non-genetic variables, such nutrition and environment, have a more significant impact on the gut microbiome, even though genetics does play a role. Large-scale research is still being conducted in this field to elucidate the link between genes and microbiomes.

2.2.6 Antibiotic Use

The gut microbiota can be severely disrupted by antibiotics, changing its makeup quickly and occasionally permanently. Gut microbiota dysbiosis, a condition of microbial imbalance that reduces microbial diversity and hinders proper function, is the most frequent result. The gut's capacity to control immunity, fight off infections, and preserve metabolic balance is weakened by this disturbance. Infections, chronic illnesses, poor diet, and antibiotic use are not the primary causes of dysbiosis. Antibiotic usage during the first year of life was linked to higher BMI in male children, according to a noteworthy multinational study with 74,946 participants. This suggests that early microbial disruption may have an impact on long-term metabolic health. It's interesting to note that females did not exhibit this connection, suggesting that antibiotics may have a sex-specific effect. The results emphasize the necessity of using antibiotics with caution, particularly in the early stages of development, in order to maintain microbial integrity and avoid long-term health effects.

3 Gut Microbiota and Type 1 Diabetes [T1D]

T-cell-mediated death of pancreatic beta cells is a hallmark of type 1 diabetes [T1D], an autoimmune disease that results in inadequate insulin production and poor glucose absorption. This leads to chronic hyperglycemia, which is strongly associated with long-term consequences like cardiovascular disease, kidney failure, and nerve damage. Over 542,000 children have type 1 diabetes in 2015, and the number of new cases diagnosed each year is about 86,000, with a 3% annual increase worldwide, according to the International Diabetes Federation. T1D has a complex etiology that includes gut microbiota, nutrition, environmental factors, and genetics. One of the body's most densely populated microbial ecosystems is the human gut, which is home to 500–1000 bacterial species and over 100 trillion cells. These microorganisms are necessary for immunological development, metabolic regulation, and digestion as well as food absorption. Bioactive metabolites produced by the gut bacteria affect immunological and systemic inflammation. Heart failure, chronic kidney disease, obesity, and diabetes are among the illnesses linked to dysbiosis, or disruptions in the microbial makeup.

Growing research suggests that early-life changes in gut microbiota may be a factor in autoimmune reactions that harm beta cells in the context of T1D. For instance, people with or at risk for T1D have been found to have higher levels of pro-inflammatory microorganisms, decreased microbial diversity, and decreased quantity of good bacteria. Additionally, Carmody *et al*. showed that nutrition has a significant impact on the gut microbiota, indicating that early dietary patterns may affect a person's vulnerability to disease. These results highlight the intricate, reciprocal interaction between gut microbiota and the development of type 1 diabetes and suggest that microbiome-targeted treatments may be useful in managing and preventing the condition.

4 Gut Microbial Composition and Type 1 Diabetes [T1D]

The gut microbial mix of healthy people and those with or at risk for T1D vary dramatically, according to numerous research. Non-obese diabetic [NOD] mice and Bio-Breeding diabetes-prone [BB-DP] rats are two animal models that resemble human type 1 diabetes and have early microbial abnormalities prior to the beginning of the disease. Comparing BB-DP rats to their diabetes-resistant counterparts, future T1D development is associated with higher levels of Bacteroides, Eubacterium, and Ruminococcus and lower levels of Lactobacillus, Bryantella, Bifidobacterium, and Turicibacter.

Similar trends are seen in human studies. T1D patients had lower levels of Actinobacteria and Firmicutes, as well as a poorer Firmicutes/Bacteroidetes ratio, according to a case-control research involving 16 children with T1D and 16 healthy controls. At the genus level, T1D children had higher levels of Clostridium, Bacteroides, and Veillonella, while healthy children had higher levels of Lactobacillus, Bifidobacterium, Blautia coccoides/Eubacterium rectale, and Prevotella. In children who subsequently acquired T1D, a different matched study revealed decreased microbial diversity and stability, with an increase in Bacteroidetes and a decrease in Firmicutes as the autoimmune process progressed. Additionally, following the emergence of T1D autoantibodies, there was a decrease in the number of butyrate-producing bacteria and a decrease in microbial stability.

Although some treatments, such as taking Lactobacillus reuteri every day, helped glucose-tolerant people secrete more insulin and incretin, this benefit was probably caused by increased beta-cell activity rather than modifications in the gut microbiota. Children with and without anti-islet cell autoantibodies did not significantly differ in terms of overall microbial diversity or composition, according to another study that examined 298 stool samples. But according to network research, children who tested positive for antibodies had more isolated nodes and intermediate eigenvector centrality, which may have decreased microbial communication and adaptation.

All things considered, the development of T1D is linked to decreased gut microbiota quantity, diversity, stability, and connection. The generation of microbial metabolites and food fermentation may be hampered by these inadequacies, which could lead to autoimmune and metabolic disorders including type 1 diabetes.

5 Gut Microbiota and Type 2 Diabetes [T2D]

A complex metabolic disease, type 2 diabetes mellitus [T2DM] is typified by persistent hyperglycemia brought on by insulin resistance and β-cell dysfunction. The gut microbiota has become a key player in the pathophysiology of type 2 diabetes in recent years. Significant changes in gut microbial composition are seen in patients with type 2 diabetes, including an increase in opportunistic pathogens like Escherichia shigella and a decrease in helpful commensals such Akkermansia muciniphila and Faecalibacterium prausnitzii. Increased intestinal permeability and improved systemic translocation of microbial products, such as lipopolysaccharides [LPS], are caused by these alterations, and they worsen insulin resistance and chronic low-grade inflammation. Additionally, decreased production of short-chain fatty acids [SCFAs], especially butyrate, which are known to maintain gut barrier function, control host immunological responses, and enhance insulin sensitivity, is linked to dysbiosis in type 2 diabetes. Recent metagenomic research has revealed metabolic pathways and microbial gene signatures that have a substantial correlation with glycemic characteristics, underscoring the microbiome's potential as a therapeutic target and diagnostic biomarker in type 2 diabetes . Restoring microbial balance with interventions such dietary fiber supplements, probiotics, prebiotics, and fecal microbiota transplantation is being researched and could provide new adjunctive therapies for bettering metabolic management.

6 Therapeutic Approaches Targeting Gut Microbiota in Diabetic Patients

The immune system, glucose homeostasis, and host metabolism are all significantly impacted by the gut bacteria. Reduced variety and quantity of beneficial bacteria, such as Faecalibacterium prausnitzii and Akkermansia muciniphila, are among the altered microbiota compositions observed in diabetic individuals.

6.1 Probiotics and Prebiotics

By restoring microbial balance, improving gut barrier function, and lowering inflammation, probiotics such Lactobacillus and Bifidobacterium species have demonstrated promise in improving glycemic control. Inulin and fructooligosaccharides are examples of prebiotics that promote the growth of good bacteria and boost the synthesis of short-chain fatty acids [SCFAs], which aid in immunological control and glucose metabolism.

6.2 Synbiotics

Synbiotics optimize beneficial microbial activity by combining prebiotics and probiotics. According to clinical research, using a synbiotic pill can help diabetes individuals have a healthier gut microbial profile while lowering their fasting blood glucose, HbA1c, and inflammatory markers.

6.3 Dietary Interventions

One fundamental strategy for influencing gut flora is dietary modification. Mediterranean, plant-based, and high-fiber diets are abundant in substrates for good bacteria and encourage the synthesis of SCFAs, including butyrate, which lower inflammation and increase insulin sensitivity. Western diets' low consumption of fermentable fibers leads to metabolic imbalance and microbial depletion.

6.4 Fecal Microbiota Transplantation [FMT]

FMT restores gut microbial diversity in a recipient by transferring fecal material from a healthy donor. Improved insulin sensitivity after FMT from lean donors has been shown in preliminary studies in people with metabolic syndrome. Even though FMT is currently in the experimental stage for diabetes, it has potential as a targeted microbiota-based treatment.

6.5 Pharmaceutical Modulators

The goal of emerging treatments is to alter the gut microbiota or its byproducts pharmacologically. These consist of bile acid modulators, microbial enzyme inhibitors, and SCFA analogs. Although first results are promising, more human research is necessary to confirm their efficacy and safety.

6.6 Metformin and Gut Microbiota

The gut microbiota76 is one way that metformin, the first-line treatment for type 2 diabetes, works. It has been demonstrated to boost the number of bacteria that produce SCFA and Akkermansia muciniphila, which are linked to better glucose management and insulin sensitivity. Along with lowering inflammation and endotoxemia, metformin also improves the integrity of the intestinal barrier and mucin formation. Moreover, microbial alterations and modified bile acid metabolism may be the mechanism underlying the gastrointestinal adverse effects frequently linked to metformin. Co-administration of probiotics or gradual dose adjustments may help lessen these side effects. Knowing how metformin and gut microbes interact can help identify new approaches to managing diabetes.

7 Future Prospets

Future studies are set to reveal customized, microbiota-based approaches to diabetes care as our knowledge of the gut microbiota–diabetes link grows. The identification of causal microbial taxa and metabolites that affect disease risk will require extensive, longitudinal investigations that integrate metagenomics, metabolomics, and immunological profiling. Glycemic management may be maximized and complications avoided with personalized microbiome therapies, which are based on a person's genetic composition, lifestyle, and current microbial environment. Next-generation probiotics and developments in microbial engineering may make it possible to precisely modify microbial ecosystems. Additionally, early diabetes diagnosis and risk stratification may be made easier by integrating gut microbiota evaluations into standard clinical practice. Lastly, the clinical translation of microbiome-based medicines, particularly FMT, will depend heavily on ethical and regulatory frameworks.

2- CONCLUSION

Because it affects immunological responses, inflammation, and metabolic homeostasis, the gut microbiota is crucial to the pathophysiology and development of both T1D and T2D. Novel approaches to diagnosis and treatment are made possible by a deeper comprehension of host–microbe interactions. Although there is currently evidence that food, probiotics, prebiotics, and pharmaceutical treatments can modify gut microbiota, further study is required to demonstrate long-term efficacy, safety, and application. Personalized therapies that target the underlying causes of metabolic dysfunction could change the approach to diabetes care from reactive to proactive by incorporating microbiome science into standard practices.

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