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GUT MICROBIOME DYNAMICS IN DIABETES MELLITUS: UNLOCKING THERAPEUTIC STRATEGIES WITH PROBIOTIC MANAGEMENT

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ABSTRACT

Diabetes Mellitus (DM) poses a global health challenge, necessitating innovative therapeutic approaches. Recent research highlights a pivotal connection between the gut microbiome and diabetes pathogenesis, unveiling potential avenues for intervention. This abstract provides an overview of the intricate interplay between the gut microbiome and diabetes, underscoring probiotic management as a promising therapeutic strategy. The human gut houses a diverse microbiome crucial for metabolic homeostasis. Alterations in microbiome composition correlate with insulin resistance, inflammation, and diabetes development. Understanding these dynamics offers opportunities for targeted diabetes interventions. Probiotics, live microorganisms conferring health benefits, emerge as tools to modulate the gut microbiome in diabetes. Specific strains positively impact glucose metabolism, enhance insulin sensitivity, and mitigate inflammation through bioactive compound production, gut barrier modulation, and interaction with immune pathways. The gut microbiome serves as a crucial determinant in diagnosing and predicting the prognosis of diabetes, offering insights into novel diagnostic markers and prognostic indicators for personalized management. Probiotic management offers a feasible strategy, opening innovative, personalized diabetes care approaches. Fecal microbiota transplantation (FMT)

emerges as a potential avenue for innovative diabetes management, leveraging the gut microbiome's role in metabolic health.

KEYWORDS: Gut microbiome, probiotics, diagnostic and prognostic markers, insulin resistance, Fecal microbiota transplantation.

INTRODUCTION

Diabetes arises either when the pancreas fails to produce sufficient insulin or when the body cannot effectively utilize the insulin it produces. Insulin, a crucial hormone, regulates blood sugar levels. Uncontrolled diabetes leads to hyperglycaemia, causing significant damage to various bodily systems, particularly the nerves and blood vessels.[1] In India, diabetes poses a significant public health challenge, with an estimated 77 million adults afflicted with type 2 diabetes and approximately 25 million at risk of developing the condition. Alarmingly, more than half of individuals with diabetes are unaware of their status, underscoring the importance of early detection and treatment. The consequences of untreated diabetes are grave, with adults facing a heightened risk of heart attacks, strokes, neuropathy, foot ulcers, blindness, and kidney failure.[2]

Diabetes mellitus includes various classifications, such as type 1 and type 2 diabetes, maturity-onset diabetes of the young (MODY), gestational diabetes, neonatal diabetes, and secondary forms associated with endocrinopathies or steroid usage. The main subtypes, type 1 and type 2 diabetes, result from defective insulin secretion or action. Type 1 diabetes typically manifests in children or adolescents, whereas type 2 diabetes predominantly affects middle-aged and older adults, often stemming from prolonged hyperglycemia due to poor lifestyle and dietary choices. Notably, unhealthy diets and sedentary lifestyles are major contributors to the global obesity epidemic, a significant risk factor for type 2 diabetes. While obesity has traditionally been attributed to external factors, emerging research suggests a potential link between gut microbiota composition and obesity.[3] The human microbiome, comprising primarily Bacteroidetes and Firmicutes, plays a crucial role in energy metabolism. Alterations in the gut microbiome, such as an increased Firmicutes to Bacteroidetes ratio, have been observed in obese individuals. Transplanting microbiota from obese mice to germ-free mice resulted in significant weight gain, highlighting the role of gut microbiota in obesity. The gut microbiome's specific demography in obese individuals may enhance energy harvest, contributing to adiposity. Firmicutes, with their ability to metabolize insoluble carbohydrates, are implicated in this process; While the exact mechanisms linking

the microbiome to obesity and type 2 diabetes remain elusive, the evidence supporting their association is substantial. Investigating the microbiome's role in type 2 diabetes holds promise for therapeutic interventions targeting obesity, inflammation, and insulin resistance.[4] Diabetes mellitus presents a significant global health burden, necessitating continuous research and innovative management strategies. Understanding the interplay between the microbiome, obesity, and type 2 diabetes offers potential avenues for preventative and therapeutic interventions, paving the way for improved health outcomes in affected individuals.

Dysbiosis and diabetes mellitius

Dysbiosis refers to an imbalance of microorganisms within our bodies, which can lead to unwanted symptoms when the colonies of microorganisms are not in harmony. This imbalance occurs when unhelpful microbes dominate those that are beneficial, resulting in a snowball effect where the beneficial microbes diminish, allowing the unfriendly ones to multiply unchecked. Various factors contribute to dysbiosis, including antibiotic use, an unhealthy diet lacking in nutrients and fiber, alcohol abuse, medical illnesses such as cancer treated with chemotherapy, and high levels of stress. Dysbiosis of the gut microbiota is primarily characterized by a decrease in the diversity and abundance of bacteria and fungi, particularly those associated with dysfunction and various pathologies. This imbalance can lead to a range of disorders including cardiovascular, neuronal, immune, and metabolic disorders due to factors such as bile acid metabolism, inflammatory status, insulin resistance, and incretin secretion.[5] The gut microbiota also plays a significant role in chronic systemic inflammation, which is secondary to endotoxemia caused by the release of endotoxins following bacterial death. Although the link between the gut microbiota and the onset and progression of diabetes is still being investigated, several studies have focused on the pathophysiology of diabetes, including its complications. Type 2 diabetes, considered a chronic and noncommunicable disease responsible for a significant portion of premature deaths worldwide, is associated with poorly controlled diabetes and metabolic disorders such as impaired lipid metabolism, oxidative stress, and hypertension, leading to microvascular and macrovascular complications. Studies have shown a significant association between changes in the composition profile of gut microbiota and the development of diabetes, with perturbed Bacteroidetes/Firmicutes phylum eubiosis linked to increased intestinal permeability and subsequent inflammatory responses. However, certain bacteria have been found to have a protective role in reducing the risk of diabetes development by improving

glucose metabolism and insulin sensitivity while suppressing inflammation.[6] Additionally, drugs such as metformin, commonly used for diabetes treatment, have been shown to alter the composition of the gut microbiota, suggesting a potential interaction between metformin and the gut microbiota in modulating inflammation, glucose homeostasis, and gut permeability. These findings suggest that metabolic factors associated with chronic low-grade inflammation and oxidative stress, which link gut microbiota dysbiosis and type 2 diabetes, also influence the onset and progression of diabetic complications.[7]

Microbiota and Diabetes Complications

Diabetic retinopathy

Diabetic retinopathy is a complication of diabetes that impacts the eyes by damaging the blood vessels within the retina, the light-sensitive tissue located at the back of the eye. Initially, diabetic retinopathy may not manifest any symptoms or only cause mild vision issues. However, if left untreated, it can eventually lead to blindness.[8] This condition can arise in individuals with type 2 diabetes with the duration of diabetes and poor blood sugar control, the risk of developing diabetic retinopathy increases. Diabetic retinopathy is prevalent, accounting for over 60% of cases in Type 2 Diabetes Mellitus (T2DM). Studies indicate a higher presence of Gram-positive bacteria, particularly coagulase-negative staphylococci, in diabetic patients, particularly those with retinopathy. This finding is consistent with research by Bilen et al., who identified Staphylococcus epidermidis and Staphylococcus aureus as predominant conjunctival organisms in T2DM patients, with higher S. aureus frequency compared to T1DM patients and healthy individuals. Parkinson's disease, a complication associated with chronic diabetic neuropathy in T2DM, also exhibits increased occurrence of S. aureus in conjunctival flora compared to healthy controls, as observed by Kusbeci.[9]

Diabetic Nephropathy

Many Type 2 Diabetes Mellitus (T2DM) patients also experience chronic kidney toxicity, including kidney stone formation, possibly due to disturbances in colonic epithelial permeability linked to T2DM and renal toxicity pathogenesis. Studies employing phylogenetic microarray analysis have revealed significant alterations in microbial operational taxonomic units (OTUs), notably an elevated abundance of Enterobacteriaceae in chronic kidney disease patients compared to healthy controls. Additionally, recent research by Zheng et al. has underscored the role of gut microbiota, particularly Klebsiella, in melamine

bioconversion, contributing to renal toxicity and crystal stone formation. Oxalobacter formigenes, a commensal gut microbe, has demonstrated efficacy in improving the clinical condition of kidney stone patients.[10] However, conventional probiotics such as Lactobacillus and Bifidobacterium species lack sufficient capacity to degrade oxalate for kidney stone treatment. Moreover, the microbiota's influence on calcium oxalate stone formation is evident from the reduced expression of vitamin K epoxide reductase complex subunit 1 (VKORC1) in patients with calcium oxalate urolithiasis, potentially modulated by vitamin K2-producing gut bacteria.[11]

Diabetic Foot Ulcer

Age-related diabetes poses an escalating risk for diabetic foot ulcers, which often lead to infections and potential limb loss. Various well-studied microorganisms, including Staphylococcus species, Pseudomonas aeruginosa, and Escherichia coli, have been identified in infected diabetic foot ulcers. The typical plantar foot flora primarily consists of coagulase-negative Staphylococcus species, known to competitively inhibit infection-associated Staphylococcus aureus.[12] Recent research by Redel et al. observed a diminished ratio of non-pathogenic Staphylococcus to pathogenic S. aureus on the feet of diabetic men compared to healthy individuals. Using bacterial 16S rRNA gene pyrosequencing, Gardner et al. examined the microbiomes of diabetic foot ulcers, revealing a negative correlation between ulcer depth and duration with the abundance of Staphylococcus, while ulcer duration showed a positive correlation with the presence of Proteobacteria.[13]

Diabetic Cardiomyopathy

Diabetic cardiomyopathy refers to cardiac dysfunction characterized by structural, functional, and metabolic changes occurring independently of coronary artery disease (CAD). This distinct clinical condition was initially proposed by Lundbaek in 1954 as diabetic heart disease, which can occur in the absence of hypertension and CAD, often coexisting with type 2 diabetes (T2D). Rubler et al. further supported these observations in 1972 by documenting post-mortem findings in four T2D patients with glomerulosclerosis and heart failure with reduced ejection fraction (HFrEF), despite normal epicardial coronary arteries and the absence of hypertension, CAD, valvular, or congenital heart disease.[14] A comprehensive nationwide case-control study conducted by Bertoni et al. in 1995 provided additional evidence of an association between non-ischemic idiopathic cardiomyopathy and diabetes. Furthermore, recent large-scale population studies have demonstrated that even with optimal

management of cardiovascular risk factors, individuals with T2D still face a consistently higher risk of hospitalization due to heart failure, without a significant increase in the risk of mortality, myocardial infarction, or stroke compared to the general population.[15]

Gut Microbiota Interaction in Insulin Metabolism

Insulin resistance serves as the primary pathophysiological basis for metabolic syndrome and Type 2 diabetes Previous metagenomic studies have outlined the features of gut microbiota and their role in metabolizing key nutrients in relation to insulin resistance. Notably, commensal bacteria's carbohydrate metabolism has been suggested to contribute substantially to the host's overall energy extraction, potentially influencing the development of obesity and prediabetes. However, the precise underlying mechanisms remain unclear. To address this, we employ a comprehensive multi-omics approach in human subjects. By integrating unbiased fecal metabolomics with metagenomics, host metabolomics, and transcriptomics data, we elucidate the microbiome's role in insulin resistance.[16] Research findings indicate elevated levels of fecal carbohydrates, particularly host-accessible monosaccharides, in individuals with insulin resistance, correlating with microbial carbohydrate metabolisms and host inflammatory cytokines. Furthermore, specific gut bacteria associated with insulin resistance and sensitivity are identified, exhibiting distinct patterns of carbohydrate metabolism.[17] Importantly, we demonstrate in a mouse model that bacteria linked to insulin sensitivity can mitigate host phenotypes of insulin resistance. This study offers a comprehensive understanding of host-microorganism interactions in insulin resistance, highlighting the significance of microbiota-mediated carbohydrate metabolism as a potential therapeutic target for addressing insulin resistance.[18]

Gut Microbiota as Dignostic and Prognotic Biomarker

The 5-year survival rate for localized colorectal cancer (CRC) stands at 90%, but sharply declines to less than 15% for metastatic disease. Consequently, the quest for early, noninvasive, and accessible CRC screening methods is paramount. While the fecal immunochemical test (FIT) serves as the primary screening strategy globally, its sensitivity for CRC diagnosis is limited to 79%. Though the multitarget stool DNA test boasts high sensitivity (92.3%), its elevated false-positive rate poses challenges. Hence, there persists a pressing need for more precise and noninvasive biomarkers for early CRC detection Microbial markers have emerged as promising indicators for early screening in light of the recognized link between gut microbiota dysbiosis and CRC.[19]

Various oncogenic gut bacteria, including Bacteroides fragilis, Escherichia coli, Enterococcus faecalis, Streptococcus gallolyticus, and Fusobacterium nucleatum, have been individually assessed from fecal and mucosal samples of CRC patients. Furthermore, sequencing methods such as whole-genome shotgun (WGS) and 16S rRNA DNA sequencing have identified distinct signatures between individuals with CRC and healthy controls, revealing a global shift in microbiota composition. While no single oncomicrobe universally characterizes all CRC patients, enriched and depleted fecal microorganisms have been discerned between CRC and control groups.[20] Fusobacterium nucleatum, in particular, has emerged as a fecal marker for CRC, with potential to enhance FIT performance in detecting CRC and advanced adenoma. Additionally, the oral-gut microbiome relationship suggests that microbial CRC biomarkers could also be explored in oral samples. Recent discoveries of endogenous microbial DNA signatures in blood samples from CRC patients further underscore the potential of microbial biomarkers in CRC diagnosis.[21]

Role of Probiotics in Diabetes Management

The proposed mechanisms for how probiotics or prebiotics work are not fully understood yet; however, several have been suggested. Improvement or control of the lipid profile associated with prebiotics and probiotics has been studied, with suggestions that this improvement is achieved by the production of short-chain fatty acids (SCFA), which act as inhibitors of lipid synthesis in the liver .Probiotics have also demonstrated the ability to reduce reactive oxygen species (ROS), which, among other harmful effects, damage the intestinal barrier and allow bacterial translocation, potentially leading to different infections and inflammation.[22] It has been associated with the control of mild chronic inflammation, as Bifidobacterium levels decreasing have been linked to increased bacterial lipopolysaccharides (LPS), characteristic of endotoxemia leading to higher concentrations of pro-inflammatory cytokines.[10] Additionally, probiotic Lactobacillus casei has been found to regulate the release of LPS into the blood via liver GlyRs upregulation. Another proposed mechanism is the modulation of Th1 and Th2 pro-inflammatory responses by probiotics, aiding in the prevention of type 2 diabetes development. Probiotic regulation of FoxA2 gene expression, affecting insulin sensitivity, has also been observed. Furthermore, probiotics have been shown to affect Cl secretion and chloride channel protein expression in the small intestine, maintaining the normal function of tight junction barriers and decreasing bacterial translocation.[23]

These effects were observed in L. casei, and further studies are needed to associate similar effects with other probiotic strains. These findings suggest possible molecular mechanisms through which probiotics act on immune responses. Moving on to prebiotics, inulin, the most widely studied prebiotic, has shown the effect of controlling glycemic index by reducing glucose absorption rates and controlling the lipid profile by decreasing serum triglyceride levels. This is achieved through the inhibition of glycerol-3-phosphate acyltransferase and fatty acid synthase, as well as key enzymes in de novo lipid synthesis.[24] Extensive work has been done to study the molecular mechanisms through which both prebiotics and probiotics function. However, further studies are needed to establish a better understanding of how these mechanisms enhance human health.

Fecal Transplantation and Diabetes Millitus

Fecal microbiota transplantation (FMT) significantly improved clinical indicators such as HOMA-IR and BMI in patients with type 2 diabetes mellitus (T2DM), in addition to the control of fasting blood glucose, postprandial blood glucose, and hemoglobin A1c levels, which were also managed by metformin. Within four weeks, the donor microbiota effectively colonized in T2DM patients, with a slightly higher colonization ratio observed in the FMT group compared to the FMT plus metformin group, resulting in increased microbial diversity and community changes compared to baseline after treatment. Notably, a total of 227 species and 441 species were significantly altered after FMT and FMT plus metformin, respectively. FMT was significantly associated with improvements in clinical parameters. Among them, Chlorobium phaeovibrioides, Bifidibacterium adolescentis, and Synechococcus sp. WH8103 were notable due to their significantly negative correlations with HOMA-IR levels.[25]

Research reported that at week 4 after FMT, microbial richness and Shannon diversity showed noticeable improvements compared to baseline, although statistical significance was marginal. The dominant taxa observed in such donor and patient samples were Bacteroidetes, Firmicutes, and Proteobacteria. Relative abundance of Bacteroidetes decreased, while Firmicutes increased after intervention in both FMT alone and FMT plus metformin groups. An unusual microbial composition characterized by a high proportion of Proteobacteria and nearly absent Bacteroidetes was observed at week four after metformin treatment.[26]

CONCLUSION

The symbiotic relationship between the microbiota and diabetes underscores the critical role of gut health in metabolic regulation. The dynamic interplay between microbial communities

and host physiology influences various aspects of glucose metabolism and insulin sensitivity, contributing to the onset and progression of diabetes. While the exact mechanisms remain incompletely understood, emerging evidence suggests that interventions targeting the microbiota hold promise for diabetes management and prevention. Probiotics, prebiotics, and dietary modifications offer potential strategies to modulate the gut microbiome and improve metabolic outcomes. However, translating these findings into clinically effective therapies requires further investigation into the complex interactions between the microbiota, host factors, and environmental influences. Moreover, personalized approaches tailored to individual microbial profiles and metabolic needs may enhance the efficacy of microbiota-based interventions. Ultimately, advancing our understanding of the microbiota-diabetes axis holds significant implications for developing innovative strategies to combat this prevalent metabolic disorder and improve global public health.

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