Available online on 15.10.2025 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

Open Access Full Text Article

Review Article

Dietary Modulation of the Gut Microbiome: A Promising Approach for Management of Diabetes

Kingsley Ikechukwu Chukwudozie *[1], Izuchukwu Christopher Chukwudozie [2], Chidimma Precious Onyeka [1]

1. Department of Microbiology, University of Nigeria
2. Department of Science Laboratory Technology, University of Nigeria

Article Info:

Article History:

Received 17 July 2025
Reviewed 08 Sep 2025
Accepted 05 Oct 2025
Published 15 Oct 2025

Cite this article as:

Chukwudozie KI, Chukwudozie IC, Onyeka CP, Dietary Modulation of the Gut Microbiome: A Promising Approach for Management of Diabetes, Journal of Drug Delivery and Therapeutics. 2025; 15(10):256-264 *DOI: http://dx.doi.org/10.22270/jddt.v15i10.7423*

*For Correspondence:

Kingsley Ikechukwu Chukwudozie, Department of Microbiology, University of Nigeria

Abstract

The gut microbiome has emerged as a critical regulator of host metabolism, immune function, and energy homeostasis, offering novel opportunities for the prevention and management of metabolic disorders such as diabetes mellitus. Dietary modulation represents a promising, non-pharmacological strategy to reshape gut microbial composition and functionality, thereby improving glycemic control and metabolic outcomes. Diets rich in fiber, polyphenols, fermented foods, and prebiotic compounds have been shown to enhance the abundance of beneficial bacteria species such as *Bifidobacterium* promote short-chain fatty acid (SCFA) production, and reduce systemic inflammation and insulin resistance. Conversely, high-fat and high-sugar Western-style diets are associated with dysbiosis, impaired gut barrier integrity, and metabolic endotoxemia, which exacerbate hyperglycemia and insulin resistance. Emerging evidence from clinical and experimental studies indicates that targeted dietary interventions, including the Mediterranean diet, plant-based diets, and functional food supplementation, can modulate gut microbiota diversity and metabolic pathways, supporting their therapeutic potential in diabetes management. This review highlights current knowledge on relationship between gut microbiome and diabetes, and offers new insights into potential preventive or therapeutic approaches that uses dietary modulation of the gut microbiome as a safe and effective adjunct to the clinical management of diabetes.

Keywords: Diabetes, Diet, Gut, Microbiome, Modulation

1. Introduction and Literature Review

The role of the microbiome, particularly the gut microbiome, in health cannot be overemphasized. The gut microbiome refers to the diverse community of microorganisms residing in the gastrointestinal tract and has emerged as a critical player in host metabolism and health [1]. Emerging evidence indicates that a symbiotic relationship exists between the host and its gut microbes. Studies have shown that the microbes of the gut microbiome impact the general physiology of the host [2]. The normal microbial community in the gut contributes to host nutrient metabolism, xenobiotic and maintenance of structural integrity of the gut mucosal barrier, immunomodulation, protection against pathogens and production of various bioactive compounds, thereby leading to overall improvement of host health [3,4]. On the other hand, an imbalance of this microbial community (dysbiosis) may result in any of various diseases, including metabolic diseases such as obesity and diabetes [5–7] (**Fig. 1**)

Dysbiosis can impair the intestinal barrier, leading to increased gut permeability and the translocation of bacterial products like lipopolysaccharides (LPS) into the systemic circulation, triggering systemic inflammation, which promotes insulin resistance and impaired glucose metabolism [8,9]. This process can affect pancreatic beta-cell function, influence adipose tissue metabolism, and alter the production of beneficial microbial compounds like short-chain fatty acids (SCFAs), collectively contributing to the development and progression of diabetes [10,11]. Similarly, hematological parameters such as red and white blood cell counts and indices of anemia are often disrupted in diabetes, reflecting oxidative stress and impaired immune response [12,13]. Furthermore, the liver plays a central role in glucose homeostasis through processes like gluconeogenesis and glycogenolysis. In diabetes, the liver is often subjected to oxidative stress and metabolic strain, which can lead to hepatocellular damage and impaired function [14,15]. Elevated levels of liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), along with changes in total protein and albumin, are indicative of liver dysfunction and are commonly observed in diabetic patients [16,17].

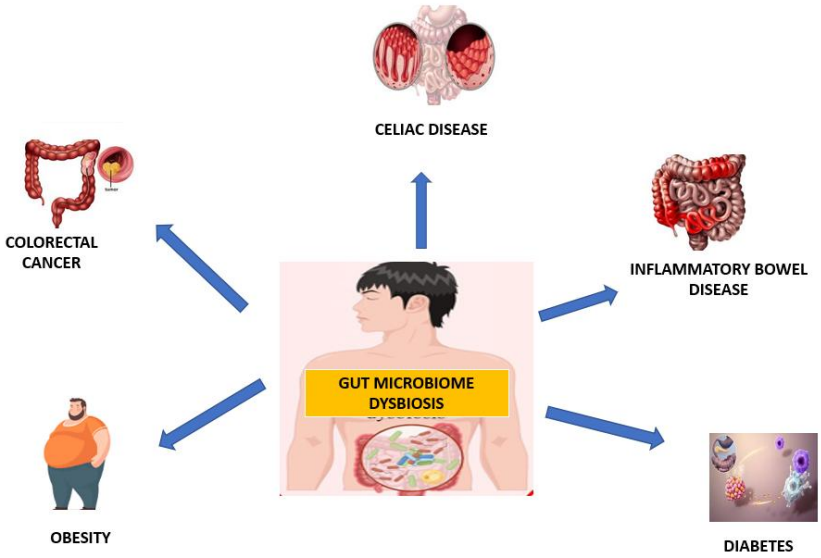

Figure 1: Dysbiosis in the gut microbiome can result to different diseases

There is a growing recognition of the role of diet in modulating the composition and metabolic activity of the human gut microbiota, which can impact health [18–20]. Several studies identified that changes in the quantity and diversity of gut microbiota have significant importance in the progression of many metabolic disorders, including diabetes and obesity [4,21,22]. The human gut microbiota is composed of different bacteria species taxonomically classified according to phyla, classes, orders, families, genera, and species. It is mostly composed by two dominant bacterial phyla, Firmicutes and Bacteroidetes that represent more than 90% of the total community, and by other subdominant phyla including Actinobacteria, Proteobacteria, Fusobacteria, and Verrucomicrobia [23,24].

Research has revealed that healthy individuals' gut microbiomes are more diverse and stable than individuals who developed autoimmunity and, eventually, diabetes. Diabetic individuals often have a gut microbial profile characterized by a reduction in beneficial bacteria populations and an increase in opportunistic or potential pathogenic bacteria [25–27]. Dietary interventions can modulate gut microbial composition to prevent and/or treat diabetes [28]. In the future, gut microbial signatures may serve as early diagnostic markers, and modulation of the gut microbiome may be widely applied to treat metabolic disorders such as diabetes. With more understanding of the gut microbiome and its relationship to health and disease, various interventions, including dietary interventions using prebiotics, probiotics and plant extracts, may restore the gut microbiota to health in patients with diabetes.

Current therapeutic approaches for diabetes include insulin therapy and oral hypoglycemic agents. While effective, these interventions are often expensive, associated with adverse side effects, limited accessibility in resource-poor settings, and do not fully address the multi-systemic complications of the disease. These complications highlight the need for therapeutic strategies that address the systemic consequences of diabetes rather than focusing solely on glycemic control. Consequently, there is a growing need for alternative or complementary therapies that are affordable, safe, and capable of addressing the multifaceted complications of diabetes. Restoration of gut microbial balance in diabetic patients through dietary modulation of the gut microbiome is now being rapidly explored as an emerging promising novel therapeutic strategy for diabetes management and in the treatment of other ailments promoted by dysbiosis in the gut microbiome [29–31]. Promising results from several animal studies indicate a potential for gut microbiome modulation in preventing and treating diabetes. Restoring gut microbial balance in diabetic patients can be achieved through dietary modulation by increasing intake of high-fiber foods (fruits, vegetables, whole grains, legumes) and probiotic foods (yogurt, kimchi, sauerkraut) while reducing processed foods. These changes promote beneficial gut bacteria, which increase short-chain fatty acid (SCFA) production, improve gut barrier function, enhance insulin sensitivity, and reduce inflammation, leading to better glycemic control and metabolic health.

2. Functions of the Gut Microbiome

The gut microbiota plays an important role in the normal functioning of the host organism. The relationship between the gut microbiota and the host organism is mutual. The gut microorganisms are supported by the host's food and play a key role in keeping the host healthy. Growing evidence from several studies on humans and animal models have reported the beneficial role of gut microbiota in human health [32,33]. The gut microbiota has many important functions in the human host, including assisting in providing resistance to pathogens, modulating the immune system[34], and playing a role in digestion and metabolism [35].

2.1. Metabolic Function of the Gut Microbiome

The Gut microorganisms play a major role in energy extraction from food through a variety of mechanisms. The gut microbiota encodes some genes that are not encoded by their host, thereby enabling them to

undertake various metabolic functions that the host is unable to perform, such as the digestion of substrates that the host cannot digest [36]. This ability results in the recovery of energy and absorbable substrates for the host and a supply of energy and nutrients for gut bacterial growth and proliferation. The gut bacteria can produce various vitamins, notably vitamin K, and a range of B group vitamins such as folate, riboflavin, biotin, niacin and thiamine, synthesize amino acids and short-chain fatty acids (SCFAs) such as butyrate, propionate and acetate [37]. Butyrate is the main energy source for colonocytes and helps strengthen the epithelial barrier against invasion and colonization by pathogenic microbes [38]. Butyrate can also activate intestinal gluconeogenesis and improve insulin sensitivity. It also has anti-inflammatory potential and potential anti-cancer activity via the ability to induce apoptosis of colon cancer cells [39–41]. Propionate is transferred to the liver, where it regulates gluconeogenesis and satiety signalling through interaction with the gut fatty acid receptors [42]. Acetate is the most abundant SCFA and an essential metabolite for the growth of other bacteria. For instance, *Faecalibacterium prausnitzii* will not grow in pure culture in the absence of acetate [43]. After production, acetate is transported to the peripheral tissues, where it is used in cholesterol metabolism and lipogenesis and may play a role in central appetite regulation [44]. Some enzymes produced by gut microbes are involved in bile acid metabolism and generating secondary bile acids that act as signaling molecules and metabolic regulators to modulate important host pathways [45].

2.2. Immuno-Protective Role of Gut Microbiota

The intestinal epithelium is the main interface between the immune system and the external environment. The development of a host's immune system is affected by continuous and dynamic interactions with the intestinal microbiota and its metabolites. Gut bacteria are integral to the early development of the gut mucosal immune system, both in terms of its physical components and function 46. The intestinal microbiota co-evolved with the host in a symbiotic relationship. In addition to some metabolic benefits, the symbiotic gut microbiota provides the host with several functions that promote immune homeostasis, immune responses, and protection against pathogen attachment and colonization. The ability of symbiotic gut bacteria to inhibit pathogen invasion and colonization is accomplished through several mechanisms. Many intestinal bacteria produce antimicrobial compounds such as bacteriocins which can inhibit the growth of pathogenic bacteria [47,48]. Many studies have reported that some probiotic Lactobacillus species were able to protect the host from foodborne infections and this was dependent on the Lactobacillus bacteriocin [49,50]. Gut bacteria compete for nutrients and sites of attachment in the gut lining and outcompete pathogenic bacteria, thereby preventing their attachment and colonization. The gut microbiota influences the immune system, modulating effector and regulatory T cells' number and function. To achieve this aim, mutual regulation between the immune system and microbiota is achieved through several mechanisms, including the engagement of toll-like receptors (TLRs) [51,52]. TLRs are transmembrane receptors mostly present on the membrane of immune cells and epithelial cells, including intestinal epithelial cells. TLRs are capable of recognizing conserved molecular motives, generally divided into microbe-associated molecular patterns (MAMPS) expressed by resident commensal microbiota and pathogens-associated molecular patterns (PAMPS) produced or expressed by microbial invaders. TLRs' engagement with PAMPS induces several intracellular signalling cascades resulting in the activation of immune cells and the production of cytokines and chemokines while maintaining tolerance towards most members of commensal gut microbiota expressing MAMPS [53].

Gut intestinal bacteria are also implicated in the prevention of allergy. Allergic infants and young children have been found to have a different composition of intestinal bacteria than those who do not develop allergies [54,55]. It is suggested that the intestinal microbiota stimulates the immune system and trains it to respond proportionately to all antigens. The altered composition of intestinal microbiota in early life can lead to an inadequately trained immune system that can often overreact to antigens [56,57]. There is growing evidence to show that short-chain fatty acids (SCFAs) play an important role in the modulation of host's immunity and the development of several diseases [58]. Short-chain fatty acids SCFAs are end products of the microbial digestion of dietary fibre that humans cannot digest because the human genome does not encode the genes needed to produce the enzymes required for digestion of dietary fibre. These missing enzymes are provided by the gut microbiota. The luminal concentration of intestinal SCFAs is affected by the amount of fibre in the diet. In addition to acting as an energy source for the host, SCFAs also regulate host immune responses. SCFAs exert their signalling and immune-modulatory effects by binding to receptors such as G-protein-coupled receptors (GPCRs), FFA2, FFA3, and Olfr78 which are sensed by specific membrane-bound receptors on the surface of gut epithelial cells [59]. Studies have revealed that the binding of SCFAc to these receptors can lead to the regulation of metabolism, inflammation and other immune-modulatory effects. SCFAc such as butyrate exhibit anti-inflammatory effects by regulating the release of cytokines, and can modify the cytokine production profile of T-helper cells[60] and promote intestinal epithelial barrier integrity by serving as an energy source for gut epithelial cells [44], which in turn can help to limit the exposure of the mucosal immune system to luminal microbes and prevent aberrant inflammatory responses. Several studies have also highlighted the role of SCFAs as a major player in maintaining gut and immune homeostasis [46].

A study showed the important role of acetate production in preventing infection with the enteropathogenic *Escherichia coli* (0157:H7) and this effect was attributed to acetate's ability to maintain the gut epithelial barrier function [61, 62] investigated the therapeutic effect of administering butyrate orally to patients with Crohn's disease CD. The result showed that the administration of 4g of butyrate daily for 8 weeks induced clinical

improvement and remission in 53% of patients where butyrate successfully downregulated mucosal levels of NF-kB and IL-1b. [63] and [64] compared the production of SCFAs in allergic and non-allergic children and found that allergic children had lower levels of propionate, acetate, and butyrate in their faeces compared to non-allergic children. Mouse studies have also shown that SCFAs were beneficial in colitis as mice treated with butyrate had reduced inflammation in their colonic mucosa with reduced neutrophil infiltration [65] and treatment with acetate had similar beneficial effects. [66] carried out a metagenomic analysis of the gut microbiota of type one diabetic subjects, and the 16S rRNA sequencing revealed a larger relative abundance of bacterial species capable of producing butyrate in controls than individual

3.0. Effect of Diet on Gut Microbiome

Dietary intake and eating habits directly influence the composition and diversity of the gut microbiome. The intestinal microbiota rapidly responds to diet variations. Some gut bacteria can metabolize particular dietary components for energy, leading to their overgrowth in the gut when diets containing these particular dietary components are consumed. Furthermore, microbial metabolites such as short-chain fatty acids (SCFAs) produced from the consumption of complex carbohydrates promote the acidic environment in the gut, which in turn shapes the microbial composition [67]. Transient changes to the gut microbiota composition with a change in diets have been noted in several studies. Studies in mice have demonstrated that a high-fat diet decreases the number of bacterial species in the gut microbiome, and the gut microbiome of mice given a high-fat diet and those given a regular unpurified diet showed variations in microbial diversity [68,69]. Wu *et al*., 2011 investigated controlled feeding in 10 subjects and found that the microbiome composition changed within 24hrs of initiating a high-fat and low-fibre or low-fat and high-fibre diet and remained stable during the 10 days study [70]. Many dietary patterns such as the Western diet and Mediterranean diet have been shown to affect the gut microbiota's diversity, which may affect host metabolism [71] (**Fig 2**). The Western diet consists of a high intake of saturated fats, refined grains, sugar and a low fibre intake. It is highly associated with obesity and other metabolic diseases. The Western has also been shown to promotes inflammation, decrease the total gut bacteria diversity and the beneficial species in the gut [72]. A Mediterranean diet is rich in dietary fibre, vegetables, grains and low in fat, contrasting with the Western diet. Abundant fibre in Mediterranean diets promotes stable gut microbiome profile and increases the presence of beneficial bacteria such as lactic acid bacteria [73], [74] reported that African children, who consume a low-fat and high-fibre diet, have less potentially pathogenic bacteria and exhibited a greater degree of diversity and microbial richness than European children consuming a high-fat diet (Western diet).

Figure 2: Many dietary patterns such as the Western diet and Mediterranean diet have been shown to affect the gut microbiota's diversity, which may affect host metabolism

4. Pathophysiology of Diabetes Mellitus

Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both [75]. Prolonged hyperglycemia leads to disturbances in carbohydrate, fat, and protein metabolism, and long-term damage to blood vessels, nerves, kidneys, eyes, and other organs [76]. Insulin is a protein hormone secreted by the β cells of the islets of Langerhans of the pancreas under the influence of the blood glucose level. After secretion, insulin is deposited into the intracellular space from where it then passes into the bloodstream and proceeds through the circulation to the body's cells. At the cellular level, insulin interacts with a protein on the cell surface called an insulin receptor. This interaction stimulates a cascade of intracellular reactions, each catalyzed by a different enzyme, which ultimately results in another protein's production, called a glucose transporter (GLUT4) in cells. GLUT4 passes or migrates to the cell surface, facilitating the entrance of glucose. There are several forms or types of diabetes mellitus, but the vast majority of cases fall into two main categories: type 1 diabetes (TIDM) and type 2 diabetes (T2DM) [77]. TIDM is an autoimmune disorder usually due to immune-mediated destruction of insulin-producing pancreatic islet β -cells by immune cells. Consequently, the body produces little or no insulin. Without insulin, glucose cannot enter the body's cells to be used for energy. This leads to glucose building up in the bloodstream (hyperglycemia).

Type 2 diabetes is a more complex condition that develops over time. It is primarily characterized by two interconnected problems: insulin resistance and a progressive decline in beta-cell function. In the early stages, the body's cells (especially muscle, fat, and liver

cells) become less responsive to insulin. This means that even though the pancreas is producing insulin, the cells aren't effectively taking up glucose from the blood. The pancreas initially compensates by producing more insulin to overcome this resistance, which keeps blood glucose levels in a normal range for a while. Over time, the pancreas's beta cells become exhausted from the hypersecretion of insulin and lose their ability to produce enough insulin to meet the body's demands. This progressive failure leads to insufficient insulin and persistent hyperglycemia. This process is often gradual and can take many years, which is why Type 2 diabetes is often diagnosed in adults. Factors such as genetics, obesity, a sedentary lifestyle, and poor diet significantly contribute to the development of insulin resistance and the eventual onset of Type 2 diabetes [78,79]. Resistance to insulin at the cellular level can be due to many causes. There can be genetic abnormalities of the insulin receptor so that insulin produced by the pancreatic islet β -cells cannot fit into the receptor and thus not activate the needed reactions within the cells. There can also be genetic abnormalities that result in a defect in any of the enzymes involved in any insulin actions within the cells.

5. Relationship Between Gut Microbiome and Diabetes

Various factors are involved in the development of diabetes, including genetic factors, diet, lifestyle and intestinal microbiota [80]. Several studies have shown that the gut microbial composition differs between healthy hosts and hosts with diabetes or at risk of diabetes, suggesting that the gut microbiota play a role in the aetiology of diabetes [81–83]. Several alterations of gut microbiota composition were described both in animal model and in humans. The decrease of *Firmicutes/ Bacteroides* ratio was the most frequent pattern described, in particular, in human studies. At the phyla level, it was well documented that the relative abundance of Firmicutes phyla decreased in T1DM patients compared to the healthy individual group, while Bacteroidetes abundance increased successively [53,54].

In a case-control study that included sixteen (16) children with type1 diabetes and sixteen (16) healthy children, their gut microbial composition showed significant differences between the healthy children and the children with type1 diabetes. At the phyla level, the abundance of Firmicutes and Actinobacteria and the ratio of Firmicutes to Bacteroidetes were all significantly lower in the children with type1 diabetes than the healthy children [83]. At the genus level, the healthy children had greater numbers of *Bifidobacterium, Lactobacillus, Blautia coccoides/Eubacterium rectale* group, and *Prevotella* specie in the gut whereas children with type1 diabetes contained greater numbers of *Veillonella, Clostridium* and *Bacteroides*, [83]. also reported that compared with healthy control subjects, type 1 diabetes was associated with a significantly lower microbiota diversity, a significantly higher relative abundance of *Veillonella, Ruminococcus, Blautia, Bacteroides* and *Streptococcus* genera, and a lower relative abundance *of Faecalibacterium, Bifidobacterium, Roseburia and Lachnospira*. From the above discussion, it can be deduced that diabetic patients' gut microbiota is characterized by an increased level of opportunistic pathogens and decreased level of probiotics. Furthermore, Firmicutes are negatively associated with diabetes, while Bacteroidetes is a positive factor at the phyla level. However, conflicting results have been reported on the difference between healthy individuals and diabetic patients at the genus level.

The mechanisms by which altered gut microbiota and diabetes interact are complex and are likely to proceed through a cascade of events primarily driven by how microbial activity impacts host metabolism and inflammation. The Gut microbiota contribute to the regulation of host glucose homeostasis through multiple mechanisms, including the synthesis of metabolites during fermentation of dietary fibre and their resulting secondary effects [86]. Butyrate, acetate, and propionate represent the principal short-chain fatty acids (SCFAs) produced through the microbial fermentation of dietary fibers in the intestine [87,88]. Among these, acetate and propionate are mainly synthesized by members of the phylum *Bacteroidetes*, whereas butyrate is predominantly produced by *Firmicutes* [58]. SCFAs may be directly utilized by intestinal epithelial cells as an energy substrate or absorbed into the systemic circulation, where they constitute an important energy source for the host and act as bioactive signaling molecules [90,91]. Even though SCFAs share some similarities in their mechanisms, they can exert different physiological effects due to variations in their chemical structure, receptor binding, cellular targets, and the specific metabolic pathways and tissues they influence. Butyrate has been reported to be the most effective short-chain fatty acid (SCFA) for improving diabetic conditions, with evidence showing it significantly reduces fasting blood glucose levels and improves insulin sensitivity in animal models [92,93].

When the gut microbiota is in dysbiosis, as in the cases of diabetes, it is directly related to alteration of SCFA production [94,95]. According to [96], the main characteristics of the microbiota of diabetic patients are reduced butyrate-producing bacteria population, dysbiosis, proinflammatory environment, increased intestinal permeability and increased serum lipopolysaccharide (LPS) concentration [66,67]. Butyrate improves insulin sensitivity and secretion by stimulating the secretion of glucagon-like peptide1 (GLP-1) from enteroendocrine L-cells [98–100]. GLP-1 intensifies glucose-induced insulin release from β-cells, suppresses glucagon secretion, protects β-cells from apoptosis, promotes β-cell proliferation [70,71]. Butyrate also serves as a primary energy source for colonic epithelial cells, essential for maintaining their integrity. Insufficient levels of butyrate have been linked to increased gut permeability due to weakened colonic epithelial cells [103–105].

Thus, the decrease in relative abundance of Firmicutes and the corresponding increase in the level of Bacteroidetes in diabetic individuals could be linked with metabolic endotoxemia [106,107]. Bacteroidetes are gram-negative bacteria containing lipopolysaccharides in their outer membranes. Dysbiosis can compromise the

integrity of the intestinal barrier, leading to a condition known as "leaky gut." This increased permeability allows bacterial components, such as lipopolysaccharides (LPS) from the cell walls of Gram-negative bacteria, to leak into the bloodstream. This translocation of LPS triggers a state of chronic, low-grade systemic inflammation, which is a major contributor to insulin resistance [108]. This inflammation disrupts insulin signaling pathways, making the body's cells less responsive to insulin and impairing glucose uptake [109]. Chronic Inflammation may lead to the activation of T-cells that mistakenly target and destroy the insulin-producing beta cells in the pancreas especially in genetically predisposed individuals.

Despite extensive research on the relationship between diabetes and the gut microbiome, the precise role of the intestinal microbiome in the pathogenesis of both Type 1 and Type 2 diabetes remains to be fully elucidated. The existing evidence is insufficient to definitively establish a causal relationship, leaving an open question as to whether dysbiosis is a cause or merely a consequence of the metabolic changes associated with the disease.

Conclusion

Dietary modulation of the gut microbiome represents a compelling and evidence-based strategy for the prevention and management of diabetes. Accumulating research demonstrates that nutrient composition, food diversity, and bioactive dietary components can reshape gut microbial communities, enhance short-chain fatty acid production, and reduce systemic inflammation, thereby improving insulin sensitivity and glycemic control. While promising, the clinical application of these findings requires further large-scale, long-term studies to identify optimal dietary patterns, individualize interventions, and elucidate host–microbe interactions that drive therapeutic benefits. Integrating dietary strategies with existing pharmacological and lifestyle approaches offers a holistic framework for diabetes management, paving the way for personalized nutrition and microbiome-targeted therapies.

Conflict of Interest: The authors declare no conflict of interest concerning the contents, authorship, and/or publication of this article.

References

1. Abonahas HH, Darwish AMG, El-Kareem HFA, Abonahas YH, Mansour SA, Korra YH, et al. Trust Your Gut: The Human Gut Microbiome in Health and Disease. Microbiome-Gut-Brain Axis Implic Heal. 2022 Jan 1;53–96.
2. De Vos WM, Tilg H, Van Hul M, Cani PD. Gut microbiome and health: mechanistic insights. Gut. 2022 May 1 [cited 2023 Oct 17];71(5):1020–32. Available from: https://gut.bmj.com/content/71/5/1020
3. Hou K, Wu ZX, Chen XY, Wang JQ, Zhang D, Xiao C, et al. Microbiota in health and diseases. Signal Transduct Target Ther 2022 71. 2022 Apr 23 [cited 2025 Sep 4];7(1):1–28. Available from: https://www.nature.com/articles/s41392-022-00974-4
4. Sasidharan Pillai S, Gagnon CA, Foster C, Ashraf AP. Exploring the Gut Microbiota: Key Insights Into Its Role in Obesity, Metabolic Syndrome, and Type 2 Diabetes. J Clin Endocrinol Metab. 2024;109(11):2709–19. https://dx.doi.org/10.1210/clinem/dgae499
5. Madhogaria B, Bhowmik P, Kundu A. Correlation between human gut microbiome and diseases. Infect Med [Internet]. 2022;1(3):180–91. Available from: https://www.sciencedirect.com/science/article/pii/S2772431X22000375
6. Afzaal M, Saeed F, Shah YA, Hussain M, Rabail R, Socol CT, et al. Human gut microbiota in health and disease: Unveiling the relationship. Front Microbiol. 2022 Sep 26;13:999001.
7. Neri-Rosario D, Martínez-López YE, Esquivel-Hernández DA, Sánchez-Castañeda JP, Padron-Manrique C, Vázquez-Jiménez A, et al. Dysbiosis signatures of gut microbiota and the progression of type 2 diabetes: a machine learning approach in a Mexican cohort. Front Endocrinol (Lausanne). 2023;14:1170459.
8. Del Chierico F, Rapini N, Deodati A, Matteoli MC, Cianfarani S, Putignani L. Pathophysiology of Type 1 Diabetes and Gut Microbiota Role. Int J Mol Sci. 2022;23(23):14650. Available from: https://www.mdpi.com/1422-0067/23/23/14650/htm
9. Sadagopan A, Mahmoud A, Begg M, Tarhuni M, Fotso M, Gonzalez NA, et al. Understanding the Role of the Gut Microbiome in Diabetes and Therapeutics Targeting Leaky Gut: A Systematic Review. Cureus. 2023;15(7):e41559. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10405753/
10. Tunuguntla MN. Understanding Gut Health: Probiotics, Dysbiosis, and their Connection to Type 2 Diabetes - A Review. Int J Clin Case Reports Rev. 2024 Jun 24;18(1):01–7.
11. Chong S, Lin M, Chong D, Jensen S, Lau NS. A systematic review on gut microbiota in type 2 diabetes mellitus. Front Endocrinol (Lausanne). 2024 Jan 17;15:1486793.
12. Alshareef AA, Alrawaili MS, Almutairi SA, Ayyad MM, Alshora W. Association of Hematological Parameters and Diabetic Neuropathy: A Retrospective Study. Diabetes, Metab Syndr Obes. 2024;17:779–93. https://www.dovepress.com/association-of-hematological-parameters-and-diabetic-neuropathy-a-retr-peer-reviewed-fulltext-article-DMSO
13. Rafaqat S, Rafaqat S. Role of hematological parameters in pathogenesis of diabetes mellitus: A review of the literature. http://www.wjgnet.com/. 2023 Mar 24;10(3):25–41. Available from: https://www.wjgnet.com/2218-6204/full/v10/i3/25.htm
14. Islam S, Rahman S, Haque T, Sumon AH, Ahmed AM, Ali N. Prevalence of elevated liver enzymes and its association with type 2 diabetes: A cross-sectional study in Bangladeshi adults. Endocrinol Diabetes Metab. 2020 Apr 1;3(2):e00116. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7170449/
15. Alam S, Raghav A, Reyaz A, Jain V, et al. Prevalence of elevated liver enzymes and its relationship with type 2 diabetes mellitus in North Indian adults. Metab. 2021;12:100130. https://www.sciencedirect.com/science/article/pii/S2589936821000542
16. Zheng D, Zhang X, You L, Li F, Lin D, Sun K, et al. The association of liver enzymes with diabetes mellitus risk in different obesity subgroups: A population-based study. Front Endocrinol (Lausanne). 2022 Oct 13;13:961762.
17. Bi Y, Yang Y, Yuan X, Wang J, Wang T, Liu Z, et al. Association between liver enzymes and type 2 diabetes: a real-world study. Front Endocrinol (Lausanne). 2024;15:1340604. https://pmc.ncbi.nlm.nih.gov/articles/PMC10913017/
18. Rinninella E, Tohumcu E, Raoul P, Fiorani M, Cintoni M, Mele MC, et al. The role of diet in shaping human gut microbiota. Best Pract Res Clin Gastroenterol. 2023;62–63:101828. Available from: https://www.sciencedirect.com/science/article/pii/S1521691823000069
19. Cruz MC, Azinheiro S, Pereira SG. Modulation of gut microbiota by diet and probiotics: potential approaches to prevent gestational diabetes mellitus. Gut Microbiome [Internet]. 2023;4:e17. https://www.cambridge.org/core/journals/gut-microbiome/article/modulation-of-gut-microbiota-by-diet-and-probiotics-potential-approaches-to-prevent-gestational-diabetes-mellitus/CC0C855C73C28EA1A65CE0F13094F260
20. Menafra D, Proganò M, Tecce N, Pivonello R, Colao A. Diet and gut microbiome: Impact of each factor and mutual interactions on

prevention and treatment of type 1, type 2, and gestational diabetes mellitus. Hum Nutr Metab. 2024;38:200286. Available from: https://www.sciencedirect.com/science/article/pii/S2666149724000483

21. Luvhengo T, Mabasa S, Molepo E, Taunyane I, Palweni ST. Paneth Cell, Gut Microbiota Dysbiosis and Diabetes Mellitus. Appl Sci 2023, Vol 13, Page 6605 [Internet]. 2023 May 29;13(11):6605. Available from: https://www.mdpi.com/2076-3417/13/11/6605/htm

22. Ye J, Wu Z, Zhao Y, Zhang S, Liu W, Su Y. Role of gut microbiota in the pathogenesis and treatment of diabetes mullites: Advanced research-based review. Front Microbiol. 2022 Oct 19;13:1029890.

23. Tsai YC, Tai WC, Liang CM, Wu CK, Tsai MC, Hu WH, et al. Alternations of the gut microbiota and the Firmicutes/Bacteroidetes ratio after biologic treatment in inflammatory bowel disease. J Microbiol Immunol Infect. 2025;58(1):62–9. Available from: https://www.sciencedirect.com/science/article/pii/S1684118224001841

24. Wang M, Zhang Z, Liu Y, Jian E, Ye P, Jiang H, et al. Research trends between childhood obesity and gut microbiota: a bibliometric analysis (2002–2023). Front Microbiol. 2024 Sep 27;15:1461306. Available from: https://bibliometric.com/

25. Baars DP, Fondevila MF, Meijnikman AS, Nieuwdorp M. The central role of the gut microbiota in the pathophysiology and management of type 2 diabetes. Cell Host Microbe .202;32(8):1280–300. https://www.sciencedirect.com/science/article/pii/S1931312824002762

26. Młynarska E, Wasiak J, Gajewska A, Steć G, Jasińska J, Rysz J, et al. Exploring the Significance of Gut Microbiota in Diabetes Pathogenesis and Management—A Narrative Review. Nutr. 2024;16(12):1938. Available from: https://www.mdpi.com/2072-6643/16/12/1938/htm

27. Bielka W, Przezak A, Pawlik A. The Role of the Gut Microbiota in the Pathogenesis of Diabetes. Int J Mol Sci. 2022;23(1):480. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC8745411/

28. Wang HJ, Battousse O, Ramadas A. Modulation of gut microbiota by dietary macronutrients in type 2 diabetes: a review. Prog Microbes Mol Biol. 2021;4(1). Available from: https://journals.hh-publisher.com/index.php/pmmb/article/view/362

29. Sharma BR, Jaiswal S, Ravindra P V. Modulation of gut microbiota by bioactive compounds for prevention and management of type 2 diabetes. Biomed Pharmacother 2022;152:113148. Available from: https://www.sciencedirect.com/science/article/pii/S0753332222005376

30. Farahbod K, Slouha E, Gerts A, Rezazadah A, Clunes LA, Kollias TF. The Effects of Diet Intervention on the Gut Microbiota in Type 2 Diabetes Mellitus: A Systematic Review. Cureus. 2024;16(3):e56737. https://pmc.ncbi.nlm.nih.gov/articles/PMC11033091/

31. Donati Zeppa S, Gervasi M, Bartolacci A, Ferrini F, Patti A, Sestili P, et al. Targeting the Gut Microbiota for Prevention and Management of Type 2 Diabetes. Nutr. 2024;16(22):3951. Available from: https://www.mdpi.com/2072-6643/16/22/3951/htm

32. Kuziel GA, Rakoff-Nahoum S. The gut microbiome. Curr Biol. 2025;32(6):R257–64. Available from: https://www.cell.com/action/showFullText?pii=S0960982222002457

33. Bidell MR, Hobbs ALV, Lodise TP. Gut microbiome health and dysbiosis: A clinical primer. Pharmacother J Hum Pharmacol Drug Ther. 2022;42(11):849–57. Available from: /doi/pdf/10.1002/phar.2731

34. Fontaine SS, Kohl KD. Optimal integration between host physiology and functions of the gut microbiome. Philos Trans R Soc B. 2020;375(1808).

Available from: /doi/pdf/10.1098/rstb.2019.0594

35. Gomaa EZ. Human gut microbiota/microbiome in health and diseases: a review. Antonie van Leeuwenhoek, Int J Gen Mol Microbiol. 2020;113(12):2019–40. Available from: https://link.springer.com/article/10.1007/s10482-020-01474-7

36. Khalil M, Di Ciaula A, Mahdi L, Jaber N, Di Palo DM, Graziani A, et al. Unraveling the Role of the Human Gut Microbiome in Health and Diseases. Microorg. 2024;12(11):2333. Available from: https://www.mdpi.com/2076-2607/12/11/2333/htm

37. Fan Y, Pedersen O. Gut microbiota in human metabolic health and disease. Nat Rev Microbiol. 2021;19(1):55–71. Available from: https://www.nature.com/articles/s41579-020-0433-9

38. Cani P, Delzenne N. The Role of the Gut Microbiota in Energy Metabolism and Metabolic Disease. Curr Pharm Des.2009;15(13):1546–58. Available from: https://www.benthamdirect.com/content/journals/cpd/10.2174/138161209788168164

39. Sun J, Chen S, Zang D, Sun H, Sun Y, Chen J. Butyrate as a promising therapeutic target in cancer: From pathogenesis to clinic (Review). Int J Oncol. 2024;64(4):44. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10919761/

40. Hodgkinson K, El Abbar F, Dobranowski P, Manoogian J, Butcher J, Figeys D, et al. Butyrate's role in human health and the current progress towards its clinical application to treat gastrointestinal disease. Clin Nutr. 2023;42(2):61–75. Available from: https://www.sciencedirect.com/science/article/pii/S0261561422003843

41. Oncel S, Safratowich BD, Lindlauf JE, Liu Z, Palmer DG, Briske-Anderson M, et al. Efficacy of Butyrate to Inhibit Colonic Cancer Cell Growth Is Cell Type-Specific and Apoptosis-Dependent. Nutrients. 2024;16(4):529. Available from: https://www.mdpi.com/2072-6643/16/4/529/htm

42. Wang GY, Qin SL, Zheng YN, Geng HJ, Chen L, Yao JH, et al. Propionate promotes gluconeogenesis by regulating mechanistic target of rapamycin (mTOR) pathway in calf hepatocytes. Anim Nutr. 2023;15:88.

Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10568569/

43. Rowland I, Gibson G, Heinken A, Scott K, Swann J, Thiele I, et al. Gut microbiota functions: metabolism of nutrients and other food components. Eur J Nutr. 2017;57(1):1. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC5847071/

44. Portincasa P, Bonfrate L, Vacca M, De Angelis M, Farella I, Lanza E, et al. Gut Microbiota and Short Chain Fatty Acids: Implications in Glucose Homeostasis. Int J Mol Sci. 2022;23(3):1105. Available from: https://www.mdpi.com/1422-0067/23/3/1105/htm

45. Guzior D V., Quinn RA. Review: microbial transformations of human bile acids. Microbiome. 2021;9(1):1–13. Available from: https://microbiomejournal.biomedcentral.com/articles/10.1186/s40168-021-01101-1

46. Marco R Di, Cortés M, Olate P, Rodriguez R, Diaz R, Martínez A, et al. Human Microbiome as an Immunoregulatory Axis: Mechanisms, Dysbiosis, and Therapeutic Modulation. Microorg. 2025;13(9):2147. Available from: https://www.mdpi.com/2076-2607/13/9/2147/htm

47. Anjana, Tiwari SK. Bacteriocin-Producing Probiotic Lactic Acid Bacteria in Controlling Dysbiosis of the Gut Microbiota. Front Cell Infect Microbiol. 2022;12:851140. Available from: www.frontiersin.org

48. Dogan S, Koc TY, Karadayi M. Effect of Bacteriocins on the Intestinal Microbiota. Eurasian J Med. 2023;55:S165. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11074934/

49. Hossain MI, Mizan MFR, Toushik SH, Ashrafudoulla M, et al. Listeria monocytogenes biofilm inhibition on food contact surfaces by application of postbiotics from Lactobacillus curvatus B.67 and Lactobacillus plantarum M.2. Food Res Int. 2021;148:110595. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0963996921004944

50. Liang Q, Zhou W, Peng S, Liang Z, Liu Z, Zhu C, et al. Current status and potential of bacteriocin-producing lactic acid bacteria applied in the food industry. Curr Res Food Sci. 2025;10:100997.

https://www.sciencedirect.com/science/article/pii/S2665927125000280

51. Wiertsema SP, van Bergenhenegouwen J, Garssen J, Knippels LMJ. The Interplay between the Gut Microbiome and the Immune System in the Context of Infectious Diseases throughout Life and the Role of Nutrition in Optimizing Treatment Strategies. Nutr. 2021;13(3):886. Available from: https://www.mdpi.com/2072-6643/13/3/886/htm

52. Kaur H, Ali SA. Probiotics and gut microbiota: mechanistic insights into gut immune homeostasis through TLR pathway regulation. Food Funct. 2022;13(14):7423–47. Available from: https://pubs.rsc.org/en/content/articlehtml/2022/fo/d2fo00911k

53. Ghasemi H. Microbial metabolites and immune modulation. J Prev Complement Med. 2024;3(4):209–11. Available from: https://www.jpcmed.com/article_212403.html

54. Pantazi AC, Mihai CM, Balasa AL, Chisnoiu T, Lupu A, Frecus CE, et al. Relationship between Gut Microbiota and Allergies in Children: A Literature Review. Nutrients. 2023;15(11):2529. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10255222/

55. Ke H, Yao H, Wei P. Advances in research on gut microbiota and allergic diseases in children. Curr Res Microb Sci. 2025;8:100362. Available from: https://www.sciencedirect.com/science/article/pii/S2666517425000240

56. Wu Y, Zhang G, Wang Y, Wei X, Liu H, Zhang L, et al. A Review on Maternal and Infant Microbiota and Their Implications for the Prevention and Treatment of Allergic Diseases. Nutrients. 2023;15(11):2483. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10255369/

57. Rey-Mariño A, Francino MP. Nutrition, Gut Microbiota, and Allergy Development in Infants. Nutr. 2022;14(20):4316. Available from: https://www.mdpi.com/2072-6643/14/20/4316/htm

58. Liu M, Lu Y, Xue G, Han L, Jia H, Wang Z, et al. Role of short-chain fatty acids in host physiology. Anim Model Exp Med. 2024;7(5):641. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11528394/

59. Akhtar M, Chen Y, Ma Z, Zhang X, Shi D, Khan JA, et al. Gut microbiota-derived short chain fatty acids are potential mediators in gut inflammation. Anim Nutr. 2022;8:350–60. Available from: https://www.sciencedirect.com/science/article/pii/S2405654521002201

60. Fusco W, Lorenzo MB, Cintoni M, Porcari S, Rinninella E, Kaitsas F, et al. Short-Chain Fatty-Acid-Producing Bacteria: Key Components of the Human Gut Microbiota. Nutr. 2023;15(9):2211. Available from: https://www.mdpi.com/2072-6643/15/9/2211/htm

61. Fukuda S, Toh H, Taylor TD, Ohno H, Hattori M. Acetate-producing bifidobacteria protect the host from enteropathogenic infection via carbohydrate transporters. Gut Microbes. 2012;3(5):449–54. Available from: https://pubmed.ncbi.nlm.nih.gov/22825494/

62. Sabatino ADI, Era RMOR, Cioppo RC, Cazzola P, Tinozzi FP, Tinozzi S, et al. Oral butyrate for mildly to moderately active Crohn ' s disease. 2005;789–94.

63. Alarifi SN, Alyamani EJ, Alarawi M, Alquait AA, Alolayan MA, Aldossary AM, et al. Integrative Metagenomic Analyses Reveal Gut Microbiota-Derived Multiple Hits Connected to Development of Diabetes Mellitus. Metabolites. 2024;14(12). Available from: https://pubmed.ncbi.nlm.nih.gov/39728500/

64. Brown CT, Davis-Richardson AG, Giongo A, Gano KA, Crabb DB, Mukherjee N, et al. Gut Microbiome Metagenomics Analysis Suggests a Functional Model for the Development of Autoimmunity for Type 1 Diabetes. PLoS One. 2011;6(10):e25792. Available from: https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0025792

65. Fachi JL, Felipe J de S, Pral LP, da Silva BK, Corrêa RO, de Andrade MCP, et al. Butyrate Protects Mice from Clostridium difficile-Induced Colitis through an HIF-1-Dependent Mechanism. Cell Rep. 2019;27(3):750-761.e7. Available from: https://www.sciencedirect.com/science/article/pii/S2211124719303845

66. Peng W, Huang J, Yang J, Zhang Z, Yu R, Fayyaz S, et al. Integrated 16S rRNA Sequencing, Metagenomics, and Metabolomics to Characterize Gut Microbial Composition, Function, and Fecal Metabolic Phenotype in Non-obese Type 2 Diabetic Goto-Kakizaki Rats. Front Microbiol. 2020;10:504623. Available from: www.majorbio.com

67. Overby HB, Ferguson JF. Gut microbiota-derived short chain fatty acids facilitate microbiota:host crosstalk and modulate obesity and hypertension. Curr Hypertens Rep. 2021;23(2):8. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7992370/

68. Zhang P. Influence of Foods and Nutrition on the Gut Microbiome and Implications for Intestinal Health. Int J Mol Sci. 2022;23(17):9588. Available from: https://www.mdpi.com/1422-0067/23/17/9588/htm

69. Qi Y, Wang X. The Role of Gut Microbiota in High-Fat-Diet-Induced Diabetes: Lessons from Animal Models and Humans. Nutr. 2023;15(4):922. Available from: https://www.mdpi.com/2072-6643/15/4/922/htm

70. Wu GD, Chen J, Hoffmann C, Bittinger K, Chen YY, Keilbaugh SA, et al. Linking long-term dietary patterns with gut microbial enterotypes. Science. 2011;334(6052):105–8. Available from: https://pubmed.ncbi.nlm.nih.gov/21885731/

71. Soldán M, Argalášová Ľ, Hadvinová L, Galileo B, Babjaková J. The Effect of Dietary Types on Gut Microbiota Composition and Development of Non-Communicable Diseases: A Narrative Review. Nutr. 2024;16(18):3134. Available from: https://www.mdpi.com/2072-6643/16/18/3134/htm

72. Beam A, Clinger E, Hao L. Effect of Diet and Dietary Components on the Composition of the Gut Microbiota. Nutr. 2021;13(8):2795. Available from: https://www.mdpi.com/2072-6643/13/8/2795/htm

73. Perrone P, D'Angelo S. Gut Microbiota Modulation Through Mediterranean Diet Foods: Implications for Human Health. Nutr. 2025;17(6):948. Available from: https://www.mdpi.com/2072-6643/17/6/948/htm

74. De Filippo C, Cavalieri D, Di Paola M, Ramazzotti M, Poullet JB, Massart S, et al. Impact of diet in shaping gut microbiota revealed by a comparative study in children from Europe and rural Africa. Proc Natl Acad Sci U S A. 2010;107(33):14691–6. Available from: https://pubmed.ncbi.nlm.nih.gov/20679230/

75. Banday MZ, Sameer AS, Nissar S. Pathophysiology of diabetes: An overview. Avicenna J Med. 2020;10(4):174. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC7791288/

76. Abdelhalim Yameny A, Yameny AA. Diabetes Mellitus Overview 2024. J Biosci Appl Res. 2024;10(3):641–5. Available from: https://jbaar.journals.ekb.eg/article_382794.html

77. J N, M.K V, S S. A Brief Review on Diabetes Mellitus: Short Communication. J Pharma Insights Res. 2024;2(1):117–21. Available from: http://jopir.in/index.php/journals/article/view/89

78. Ojo OA, Ibrahim HS, Rotimi DE, Ogunlakin AD, Ojo AB. Diabetes mellitus: From molecular mechanism to pathophysiology and pharmacology. Med Nov Technol Devices. 2023;19:100247. Available from: https://www.sciencedirect.com/science/article/pii/S2590093523000425

79. Lu X, Xie Q, Pan X, Zhang R, Zhang X, Peng G, et al. Type 2 diabetes mellitus in adults: pathogenesis, prevention and therapy. Signal Transduct Target Ther. 2024;9(1):1–25. Available from: https://www.nature.com/articles/s41392-024-01951-9

80. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, et al. Pathophysiology of Type 2 Diabetes Mellitus. Int J Mol Sci. 2020;21(17):6275. Available from: https://www.mdpi.com/1422-0067/21/17/6275/htm

81. Mostafavi Abdolmaleky H, Zhou JR. Gut Microbiota Dysbiosis, Oxidative Stress, Inflammation, and Epigenetic Alterations in Metabolic Diseases. Antioxidants. 2024;13(8):985. Available from:

https://www.mdpi.com/2076-3921/13/8/985/htm

82. Mart B, Castellano-castillo D, Moreno-indias I, Urda-cardona A, Tinahones FJ. Gut Microbiota Differs in Composition and Functionality Between Children With Type 1 Diabetes and MODY2 and Healthy Control Subjects : A Case-Control Study. Diabetes Care. 2018;41(11):2385–95.

83. Murri M, Leiva I, Gomez-zumaquero JM, Tinahones FJ, Cardona F, Soriguer F, et al. Gut microbiota in children with type 1 diabetes differs from that in healthy children : a case-control study. BMS Medicine. 2013;11(1)1–12.

84. Crudele L, Gadaleta RM, Cariello M, Moschetta A. Gut microbiota in the pathogenesis and therapeutic approaches of diabetes. eBioMedicine. 2023;97:104821. Available from: https://www.thelancet.com/action/showFullText?pii=S2352396423003870

85. Wu Z, Tian E, Chen Y, Dong Z, Peng Q. Gut microbiota and its roles in the pathogenesis and therapy of endocrine system diseases. Microbiol Res.2023;268:127291. Available from: https://www.sciencedirect.com/science/article/pii/S0944501322003317

86. Cunningham AL, Stephens JW, Harris DA. Gut microbiota influence in type 2 diabetes mellitus (T2DM). Gut Pathog. 2021;13(1):1–13. Available from: https://gutpathogens.biomedcentral.com/articles/10.1186/s13099-021-00446-0

87. Pham NHT, Joglekar M V., Wong WKM, Nassif NT, Simpson AM, Hardikar AA. Short-chain fatty acids and insulin sensitivity: a systematic review and meta-analysis. Nutr Rev. 2023;82(2):193. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC10777678/

88. Zheng J, An Y, Du Y, Song Y, Zhao Q, Lu Y. Effects of short-chain fatty acids on blood glucose and lipid levels in mouse models of diabetes mellitus: A systematic review and network meta-analysis. Pharmacol Res. 2024;199:107041. Available from: https://www.sciencedirect.com/science/article/pii/S1043661823003973

89. Xie C, Qi C, Zhang J, Wang W, Meng X, Aikepaer A, et al. When short-chain fatty acids meet type 2 diabetes mellitus: Revealing mechanisms, envisioning therapies. Biochem Pharmacol. 2025;233:116791. Available from: https://www.sciencedirect.com/science/article/pii/S000629522500053X

90. Huda MN, Kim M, Bennett BJ. Modulating the Microbiota as a Therapeutic Intervention for Type 2 Diabetes. Front Endocrinol. 2021;12:632335. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC8060771/

91. Tariq HMA, Khan NY, Manzoor H, Kayani MUR. Exploring the impact of type 2 diabetes and glucose-lowering drugs on gut microbiome dynamics. Discov Med. 2025;2(1):1–16. Available from: https://link.springer.com/article/10.1007/s44337-025-00241-9

92. Pedersen SS, Prause M, Sørensen C, Størling J, Moritz T, Mariño E, et al. Targeted Delivery of Butyrate Improves Glucose Homeostasis, Reduces Hepatic Lipid Accumulation and Inflammation in db/db Mice. Int J Mol Sci. 2025;24(5):4533. Available from: https://www.mdpi.com/1422-0067/24/5/4533/htm

93. Arora T, Tremaroli V. Therapeutic Potential of Butyrate for Treatment of Type 2 Diabetes. Front Endocrinol. 2021;12:761834. Available from: www.frontiersin.org

94. Salgaço MK, Oliveira LGS, Costa GN, Bianchi F, Sivieri K. Relationship between gut microbiota, probiotics, and type 2 diabetes mellitus. Appl Microbiol Biotechnol. 2019;103(23–24):9229–38. Available from: https://link.springer.com/article/10.1007/s00253-019-10156-y

95. Nireeksha, Maniangat Luke A, Kumari N S, Hegde MN, Hegde NN. Metabolic interplay of SCFA's in the gut and oral microbiome: a link to health and disease. Front Oral Heal .2025;6:1646382. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC12414996/

96. Sabatino A, Regolisti G, Cosola C, Gesualdo L. Intestinal Microbiota in Type 2 Diabetes and Chronic Kidney Disease. Intestinal Microbiota in Type 2 Diabetes and Chronic Kidney Disease. Current Diabetes. 2017;17(3)

97. Syromyatnikov M, Nesterova E, Gladkikh M, Smirnova Y, Gryaznova M, Popov V. Characteristics of the Gut Bacterial Composition in People of Different Nationalities and Religions. Microorg. 2022;10(9):1866. Available from: https://www.mdpi.com/2076-2607/10/9/1866/htm

98. Iatcu CO, Steen A, Covasa M. Gut Microbiota and Complications of Type-2 Diabetes. Nutr. 2022;14(1):166. Available from: https://www.mdpi.com/2072-6643/14/1/166/htm

99. Ji H, Su S, Chen M, Liu S, Liu S, Guo J. The role of gut microbiota in insulin resistance: recent progress. Front Microbiol. 2025 Jul 25;16:1633029.

100. Siptroth J, Moskalenko O, Krumbiegel C, Ackermann J, Koch I, Pospisil H. Variation of butyrate production in the gut microbiome in type 2 diabetes patients. Int Microbiol. 2023;26(3):601–10. Available from: https://link.springer.com/article/10.1007/s10123-023-00324-6

101. Abdalqadir N, Adeli K. GLP-1 and GLP-2 Orchestrate Intestine Integrity, Gut Microbiota, and Immune System Crosstalk. Microorg. 2022;10(10):2061. Available from: https://www.mdpi.com/2076-2607/10/10/2061/htm

102. Wang QY, Cai JP. Dietary Modulation of Glucagon-like Peptide 1 Secretion: insights and innovations. Food Heal. 2024;6(2).

103. Winiarska-Mieczan A, Tomaszewska E, Donaldson J, Jachimowicz K. The Role of Nutritional Factors in the Modulation of the Composition of the Gut Microbiota in People with Autoimmune Diabetes. Nutr. 2022;14(12):2498. Available from: https://www.mdpi.com/2072-6643/14/12/2498/htm

104. Salamone D, Rivellese AA, Vetrani C. The relationship between gut microbiota, short-chain fatty acids and type 2 diabetes mellitus: the possible role of dietary fibre. Acta Diabetol. 2021;58(9):1131–8. Available from: https://link.springer.com/article/10.1007/s00592-021-01727-5

105. Tang R, Li L. Modulation of Short-Chain Fatty Acids as Potential Therapy Method for Type 2 Diabetes Mellitus. Can J Infect Dis Med Microbiol. 2021;2021(1):6632266. Available from: /doi/pdf/10.1155/2021/6632266

106. Gomes JMG, Costa J de A, Alfenas R de CG. Metabolic endotoxemia and diabetes mellitus: A systematic review. Metabolism. 2017;68:133–44. https://www.sciencedirect.com/science/article/abs/pii/S0026049516301871

107. Yuan X, Wang R, Han B, Sun CJ, Chen R, Wei H, et al. Functional and metabolic alterations of gut microbiota in children with new-onset type 1 diabetes. Nat Commun. 2022;13(1):1–16. Available from: https://www.nature.com/articles/s41467-022-33656-4

108. Murugesan R, Kumar J, Leela KV, Meenakshi S, Srivijayan A, Thiruselvam S, et al. The role of gut microbiota and bacterial translocation in the pathogenesis and management of type 2 diabetes mellitus: Mechanisms, impacts, and dietary therapeutic strategies. Physiol Behav. 2025;293:114838. Available from: https://www.sciencedirect.com/science/article/abs/pii/S0031938425000040X

109. Di Vincenzo F, Del Gaudio A, Petito V, Lopetuso LR, Scaldaferri F. Gut microbiota, intestinal permeability, and systemic inflammation: a narrative review. Intern Emerg Med. 2024;19(2):275–93. Available from: https://link.springer.com/article/10.1007/s11739-023-03374-w