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How Does the Gut Microbiome Contribute to Diabetes Development: A Literature Review

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Abstract

Introduction: Type 2 diabetes mellitus affects over 6% of the world population and is strongly linked to obesity. Traditional views have attributed obesity to sedentary lifestyles and high-fat diets but emerging evidence implicates gut microbiome dysbiosis as a pivotal factor in type 2 diabetes mellitus onset. High fat diets can impair microbiome health, leading to dysbiosis that triggers metabolic deregulation, insulin resistance, and inflammation. The purpose of this review is to elucidate how alterations in the gut microbiota contribute to type 2 diabetes mellitus development, with a focus on obesity, insulin resistance, and inflammatory pathways.

Methods: We conducted a comprehensive review of the literature, integrating findings from clinical trials, preclinical studies, and systematic reviews. Our analysis focused on interventions targeting the gut microbiota, including direct supplementation with short-chain fatty acids such as butyrate, probiotic treatments, and fecal microbiota transplantation. Additionally, we examined dietary strategies to boost butyrate production via increased fiber intake.

Results: Findings across studies consistently reveal a reduction in butyrate producing bacteria correlates with type 2 diabetes mellitus and metabolic dysregulation. Clinical trials have demonstrated that probiotic interventions, such as the daily consumption of probiotic yogurt, significantly lower fasting blood glucose while enhancing antioxidant defenses. Preclinical research in high fat diet induced obese mice revealed that butyrate supplementation preserves insulin sensitivity, promotes mitochondrial function, and enhances adaptive thermogenesis. Furthermore, specific microbial taxa such as *Akkermansia muciniphila* exert protective effects.

Discussion: These findings suggest that gut microbiota modulate host metabolism via key signaling pathways. The interactions between dietary factors, microbial composition, and host inflammatory responses highlights the potential for microbiome targeted therapies in type 2 diabetes mellitus management.

Conclusion: Gut targeted interventions including probiotics, prebiotics, and synbiotics, and fecal microbiota transplantation hold promise for improving glycemic control and mitigating insulin resistance. Future research should focus on optimizing these strategies to restore microbial balance, reducing obesity related metabolic derangements and improved long term outcomes in type 2 diabetes mellitus patients.

Keywords: diabetes; dysbiosis; microbiome; type 2 diabetes

Introduction

Obesity and type 2 diabetes (T2D) are interrelated global public health challenges with alarming prevalence rates. Nearly 39.8% of the U.S. population is classified as obese, with the epidemic steadily rising across all age groups [1]. This condition significantly contributes to the development of metabolic disorders, including T2D, which affects over 451 million individuals worldwide and is projected to rise to 693 million by 2045 [2]. Despite advances in prevention and treatment, current strategies remain inadequate in halting this trajectory, necessitating innovative approaches to address the pathophysiological underpinnings of these diseases. Emerging evidence highlights the gut microbiome's pivotal role in the development and progression of obesity and T2D. The gut microbiota, comprising trillions of microorganisms, influences host metabolism, energy regulation, and immune function [3]. Notably, alterations in the gut microbiome composition—such as a shift in the Firmicutes-to-Bacteroidetes ratio—have been associated with increased energy harvest and adiposity in obese individuals [1, 2]. Experimental studies have demonstrated that microbiota transplantation from obese to germ-free mice induces significant weight gain, underscoring the microbiome's role in metabolic regulation [1]. Additionally, disruptions in butyrate-producing bacteria and short-chain fatty acid metabolism have been linked to chronic inflammation, which is the elevated levels of pro-inflammatory cytokines

in circulation [4]. Such disruptions also have effects on insulin resistance and glucose dysregulation. Beyond observational studies, interventions targeting the gut microbiome—such as probiotics, prebiotics, and dietary modulation—have shown promise in improving metabolic outcomes. Certain probiotic strains, including *Lactobacillus acidophilus* and *Bifidobacterium lactis*, have demonstrated antidiabetic effects through mechanisms such as reactive oxygen species scavenging and the modulation of gut dysbiosis, which is an imbalance in the different microorganisms living together in a microbiome [5]. Similarly, supplementation with *Akkermansia muciniphila*, a mucin-degrading bacterium inversely associated with T2D, has been linked to improved metabolic parameters in clinical studies [6]. Despite these advances, critical gaps remain in understanding the mechanistic interactions between the microbiome and metabolic diseases. The objective of this study is to examine the influence of the gut microbiome on the development of diabetes. We expect to see individuals with poor gut microbiome and dysbiosis to be at greater risk of developing diabetes.

Methods

This literature review examines the intricate relationship between metabolic disorders, particularly T2D, and imbalances in gut microbiota, highlighting the potential mechanisms through which microbial composition influences metabolic health. Data were gathered through a systematic review of published articles, academic journals and other academic sources related to metabolic diseases, gut microbiota, and insulin homeostasis.

The data collection process involved a comprehensive search across multiple academic databases. Studies were selected based on the inclusion criteria, which covered the period from 2009 to 2024. Keywords used in the search included "metabolic disorders," "type 2 diabetes," "obesity," "gut microbiome," "insulin homeostasis," and "microbial diversity." Only studies that addressed the relationship between gut microbial composition and metabolic health, particularly in the context of T2D and obesity, were included. Both qualitative and quantitative studies were considered to provide a broad understanding of the mechanisms involved in metabolic disorders. Articles were included if they:

1. Investigated metabolic disorders, particularly T2D and obesity, and their relationship to gut microbiome composition.
2. Focused on the role of gut microbiota, including butyrate-producing bacteria and the gut mycobiome, in metabolic diseases.
3. Examined both qualitative and quantitative aspects of microbial diversity and insulin homeostasis.
4. Were published in English.

Studies were excluded if they did not address the connection between the microbiome and metabolic disorders or if they lacked relevant data on microbial composition and its impact on metabolic phenotypes. Additionally, studies were excluded if they were either purely speculative, did not include primary data, or failed to report clear methodologies. Review articles, editorials, and opinion pieces were excluded unless they provided substantial meta-analyses or systematic reviews with significant findings.

Studies that met the inclusion criteria based on titles and abstracts underwent a full-text review. Data from these selected articles were extracted and analyzed to identify recurring themes related to the relationship between gut microbiome composition, metabolic disorders, and insulin homeostasis, particularly focusing on T2D and obesity.

Results

Dysbiosis could be interrelated with the development of T2D because of a number of reasons that affect insulin sensitivity and glucose metabolic pathways. One of the important factors is related to the breaching of intestinal integrity, a factor commonly referred to as "leaky gut." Once the gut lining is compromised, there is easy translocation into the bloodstream by endotoxins such as lipopolysaccharides (LPS), which are molecules found in the outer membrane of gram-negative bacteria. This brings about systemic inflammation. Chronic, low-grade inflammation interferes with insulin signaling as a means of promoting insulin resistance and metabolic dysfunctions.

In the gut microbiota of individuals with either prediabetes or T2D, diversity is usually lowered, especially in butyrate-producing bacteria such as *Akkermansia muciniphila*. Butyrate is one of the most important SCFAs, reinforcing gut barrier integrity, reducing systemic inflammation, and enhancing insulin sensitivity. Besides, butyrate has been shown to improve mitochondrial function and energy expenditure, contributing to the prevention of diet-induced insulin resistance. Reduction of these beneficial SCFA-producing bacteria further increases gut permeability and inflammation, resulting in disease acceleration.

On the other hand, harmful metabolites accumulated in increased amounts, including branched-chain amino acids (BCAAs) and trimethylamine N-oxide (TMAO), promote metabolic dysfunction. *Prevotella copri*, a gut microbe linked to excess production of BCAA, has been implicated in systemic inflammation and glucose metabolism impairment. Similarly, bacterial metabolism of nutritional products, including choline and carnitine, leads to the generation of TMAO, which inhibits insulin signaling in the liver and enhances insulin resistance. The presence of genes such as *CutC* and *CutD* in TMAO-producing bacteria like *Pelobacter* further underlines the impact of gut microbiota composition on metabolic health.

Discussion

The Role of Beneficial Gut Bacteria in Managing Diabetes

Specific species of microbes in gut microbiota further play a role in metabolic health. In particular, *Akkermansia muciniphila* improves both glucose tolerance and insulin sensitivity through mechanisms such as the stimulation of Glucagon Like Peptide 1 (GLP-1) or activation of the Phosphoinositide 3-Kinase-Akt Pathway, both of which moderate lipid and glucose metabolism [6, 8]. Lactobacilli species have been well studied and shown to have a positive effect on the management of T2D. *Lactobacillus reuteri* intake as a probiotic has increased incretin release, which in turn improves insulin secretion [5]. The presence of GLP-2, endotoxins, and immune mediators were also increased. Ingestion of *Lactobacillus acidopilus* has also shown improvements in oxidative and glucose metabolism in people with T2D [8]. Lactobacilli species are also well known for their production of lactic acid. This product can be found in the glucose metabolism pathway and can reduce glucose absorption in the intestinal tract. In doing so, the presence of lactic acid in the gut can improve the management of blood glucose levels.

Gut microbiota play a crucial role in upkeeping metabolic health via their byproducts, such as Short Chain Fatty Acids (SCFAs). Beneficial bacteria, including *Faecalibacterium prausnitzii* and Roseburia, are known producers of butyrate, an important SCFA formed during the fermentation of indigestible carbohydrates. *F. prausnitzii* in particular is known for its anti-inflammatory properties and its role in maintaining gut health by producing metabolites such as butyrate and salicylic acid, both of which help to reduce proinflammatory cytokines, such as interleukin 6 (IL-6) and Tumor Necrosis Factor Alpha (TNF-α). Additionally, *F. prausnitzii* is associated with strengthening gut barrier and modulating immune responses, making it important in preventing gut permeability and systemic inflammation, both of which are often connected to metabolic disorders, including T2D [9]. Furthermore, SCFA's reinforced the gut barrier's integrity through a reduction in systemic inflammation and intestinal permeability which directly reinforces insulin activity and is a critical aspect of metabolic regulation [1, 4]. In the group of SCFAs, butyrate in particular plays a crucial role in mitigating T2D by not only reducing glycemia levels but additionally enhancing mitochondrial functioning and energy expenditure which directly addresses the concept of diet-induced insulin resistance. These benefits showcase its [butyrate] therapeutic potential in regulating metabolic disorders.

Propionate is a short-chain fatty acid that plays an important role in gut health and metabolic regulation, similar to butyrate. While butyrate is primarily associated with firmicutes, propionate is typically linked to bacteroidetes [1]. Propionate, like butyrate and acetate, is absorbed through the apical membrane of colonocytes via the sodium-coupled monocarboxylate transporter 1, although its transport is slower compared to butyrate [4, 10]. Studies on propionate show an improvement in the presence of plasma peptide YY and GLP-1, with both molecules helping to reduce energy intake and thus preventing adipose tissue development, weakening of insulin sensitivity, and reducing weight gain [11]. Additionally, physiologically relevant concentrations of propionate have demonstrated a suppression of gluconeogenesis (metabolic process of synthesizing glucose from sources like amino acids) in hepatocytes by downregulating glucose-6-phosphatase and phosphoenolpyruvate carboxykinase through the activation of AMP-activated protein kinase, both enzymes being crucial in the metabolic pathway [12].

Increased gluconeogenesis in the liver of patients with T2D is thought to be a significant contributor to elevated blood glucose levels in addition to organ damage [13]. Thus, by supporting the inhibition of this metabolic pathway, the presence of propionate and the bacterium that produce it can help support the management of diabetes.

Other gut microbes have also been identified to be linked to metabolic health, particularly in the context of T2D. Specifically, a common gut microbe *Prevotella copri* is known to produce BCAA's that play a complex role in metabolism [14]. Although BCAA's are important as essential nutrients for the body, in excess they are linked to insulin resistance and metabolic dysfunction. This is supported by findings showing an increased capacity for BCAA biosynthesis and a reduced potential for microbial uptake and degradation of BCAAs in the gut microbiota of insulin-resistant individuals [7]. BCAA impairs insulin signalling by activating the mechanistic target of rapamycin complex 1 (mTORC1) pathway. *P. copri* has been linked to an increased production of BCAA's which then causes systemic inflammation in addition to impairments to glucose metabolism.

Harmful Gut Microbes: Their Impact on Diabetes and Overall Health

Research has shown that although many butyrate-producing bacteria are generally associated with improved metabolic activity like an enhanced sensitivity to insulin and reduction of inflammation, other species can deviate from these findings. Specifically, the butyrate-producing bacteria *Flavonifractor* has been implicated to a decreased level of insulin sensitivity [15]. An explanation to this could be the differential metabolic effects of butyrate. Butyrate is an SCFA and is recognized for its benefits of gut health and systemic metabolism. However, in the case of *Flavonifractor,* its metabolic pathways and method of butyrate production differ in ways that counteract the typical benefits of butyrate. Additionally, *Flavonifractor* might also produce other metabolic byproducts in addition to butyrate which can cause the activation of pro-inflammatory pathways or interfere with the metabolism of glucose. Chronic low-grade inflammation, a telltale sign of

metabolic disorders, can also result from these interactions which can block the positive effects of butyrate and instead contribute to increasing insulin resistance. Furthermore, *Flavonifractor* also has been linked to patterns of dysbiosis, which is an imbalance in gut microbiota and as a result, can compromise the gut barrier which can lead to the translocation of the LPS into the bloodstream [15].

LPS contribute to metabolic endotoxemia. Specifically, endotoxemia caused by the LPS of *Escherichia coli* demonstrates deterioration of the gut barrier's function and weakens the glycemic management in mice [16]. Hence, when dysbiosis in the gut occurs, increased levels of the LPS can lead to low levels of inflammation, which promotes alterations in insulin and GLP-1 secretion.

Gut dysbiosis, which is characterized by a reduced microbial diversity, decreased level of butyrate production and elevated level of BCAA's, is closely tied to T2D. These changes cause systemic inflammation and disrupt insulin signalling which speeds up the progression of developing insulin resistance [1, 7]. This inflammation is driven by pro-inflammatory cytokines such as interleukin 1, IL-6, and TNF-α which further exacerbates the β-cell dysfunction and metabolic instability. These underline the intricate connection between gut health and metabolic diseases.

Another bacterial metabolite shown to develop insulin resistance is trimethylamine N-oxide (TMAO) [17]. TMAO is an oxidation product of trimethylamine, which is commonly found in nutrients like choline and carnitine phosphatidylcholine which can be seen in foods like red meat and eggs. There is evidence that TMAO also exacerbates insulin resistance and metabolic dysfunction. Specifically, TMAO interferes with the insulin signalling pathway in the liver by changing the expression of the genes that involve glycogen synthesis, gluconeogenesis, and glucose transport. Studies reveal the genes CutC and CutD to be involved in the metabolism of TMAO. Bacteria present with these genes include the *Pelobacter* genus [18].

Several studies attempt to show correlations to the abundance of an organism in relation to the onset of diabetes. However, confounding variables and a lack of information regarding the mechanism of action cause variations in studies and different results within a genus [19].

Therapeutic Strategies to Enhance Gut Health for Diabetes Management

Growing evidence suggests that gut microbiota, which plays a critical role in glucose metabolism and insulin sensitivity, may offer novel therapeutic targets for T2D management. Dysbiosis, marked by a reduction in beneficial bacteria and an increase in pathogenic bacteria, contributes to inflammation and impaired gut barrier function in T2D patients [20]. Probiotics, such as *Lactobacillus* and *Bifidobacterium* species, have been shown to improve glycemic control and insulin sensitivity through mechanisms like short-chain fatty acid production and reduced inflammation [19]. Additionally, dietary patterns high in fruits, vegetables, whole grains, and legumes can enhance the abundance of beneficial bacteria and support overall gut health [21].

Conclusions

The role of the gut microbiome in metabolic health highlights its potential as a therapeutic target for diseases such as T2D. Research underscores the significant influence of dietary patterns, probiotics, and microbial metabolites like butyrate in modulating gut health and metabolic pathways. While the probiotic *Akkermansia muciniphila* and dietary interventions, such as the microbiome diet and supplementation with butyrate, show promise in improving metabolic outcomes, challenges remain. Most studies rely heavily on animal models, necessitating robust, large-scale clinical trials to validate findings in human populations. Additionally, the complexity of T2D as a multifactorial disease demands a personalized approach that considers individual microbiota compositions, genetic predispositions, and dietary habits.

Although research has identified beneficial and harmful bacterial strains in metabolic regulation, the precise mechanisms by which gut microbiota influence insulin sensitivity, glucose metabolism, and inflammation require further investigation. Understanding how microbial metabolites such as short-chain fatty acids (SCFAs), branched-chain amino acids (BCAAs), and trimethylamine N-oxide (TMAO) interact with host metabolic pathways is crucial in refining therapeutic strategies. Future research should focus on the interplay between gut microbial diversity, diet, and metabolic function to develop targeted probiotic formulations and dietary interventions.

Advancements in microbiome research, coupled with precision nutrition and microbiota-based therapies, offer promising strategies for improving metabolic health. By continuing to explore the relationship between diet, the microbiome, and metabolic function, researchers and physicians can create innovative, personalized approaches to prevent and manage chronic diseases like T2D. Ultimately, a deeper understanding of gut microbiota could lead to novel therapeutics, including microbiome-based drugs, tailored probiotic treatments, and dietary recommendations that optimize metabolic outcomes and enhance overall health.

List of Abbreviations

BCAA: branched-chain amino acids
GLP-1: glucagon like peptide 1
GLP-2: glucagon like peptide 2
IL-6: interleukin 6
LPS: lipopolysaccharides
SCFA: short-chain fatty acid
TMAO: trimethylamine N-oxide
TNF-α: tumor necrosis factor alpha

Conflicts of Interest
The authors declare that they have no conflict of interests.

Ethics Approval and/or Participant Consent
This study did not require ethics approval or participant consent as it presents a literature review.

Authors' Contributions
RS: made substantial contributions to the design of the study, the collection of data as well as interpretation and analysis of the data, revised the manuscript critically, and gave final approval of the version to be published.
NW: contributed to study design and planning, assisted with the collection and analysis of data, and gave final approval of the version to be published.
SN: made contributions to the design of the study, collected and analysed data, drafted the manuscript, and gave final approval of the version to be published.

Acknowledgements
We would like to express our gratitude to Courtney Ostromecki, for providing guidance and support throughout this literature review.

Funding
This study was not funded.

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Article Information
Managing Editor: Jeremy Y. Ng
Peer Reviewers: Courtney Ostromecki, Mikayla Hall-Bruce
Article Dates: Received Feb 06 25; Accepted May 20 25; Published Jul 02 25

Citation
Please cite this article as follows:
Sundararajan R, Wang N, Niu S. How does the gut microbiome contribute to diabetes development: A literature review. URNCST Journal. 2025 Jul 02: 9(6). https://urncst.com/index.php/urncst/article/view/799
DOI Link: https://doi.org/10.26685/urncst.799