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REVIEW ARTICLE

Gut Microbiome and Diabetes: Emerging Perspectives in Metabolic Regulation

Navreet Kaur,[1] Navjot Kaur,[1] Aanchal Verma,[1] Rahul Kumar Sharma[1]

Abstract

Diabetes mellitus type 2 (T2DM) is one of the fastest-growing metabolic disorders in the world. It is marked by insulin resistance, high blood sugar and high cholesterol levels. Recently, its prevalence has increased significantly, making it a major health issue globally. New evidence shows that people with T2DM often have changes in their gut microbiota composition and suffer from problems in multiple organ systems. This review focused on the changes in gut microbiota related to T2DM and examined how microbial byproducts affect the disease's development. The potential for identifying at-risk individuals based on microbial patterns and the creation of specific treatments was also discussed. Lastly, it was looked into gut health strategies that aim to change the gut microbiota to prevent or slow the advancement of T2DM.

Key words: Diabetes mellitus; Gastrointestinal microbiome; Hyperglycaemia; Hyperlipidaemias; Insulin resistance.

1. Department of Pharmacology, Amar Shaheed Baba Ajit Singh Jujhar Singh Memorial College of Pharmacy, Bela, Ropar, Punjab, India.

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Corresponding author:
RAHUL KUMAR SHARMA
E: buntyrahul177@gmail.com

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Introduction

The human gut is a large microbial ecosystem where the host has a symbiotic relationship with bacteria. Each person has their own unique gut ecology. Research shows that all humans share a "core" microbiota, which makes up about a third of the gut's species. The remaining two-thirds of species can differ among individuals.[1] Many factors contribute to this uniqueness. Intrinsic factors include antimicrobial proteins and mucus. Extrinsic factors involve nutrition and medication.[2-4] High blood sugar levels brought on by either insulin resistance or insulin insufficiency are the hallmark of diabetes mellitus, a chronic illness. Diabetes comes in two primary forms. Type 2 diabetes mellitus (T2DM) is brought on by genetic and environmental factors, whereas type 1 diabetes is brought on by an autoimmune reaction.[5, 6] The main cause of diabetes is beta cell death and loss of function, which causes the pancreas to produce hyperglycaemia. Activity in drug development for the management of this common disease, namely anti-diabetic medications, has also provided options for current and future treatment. However, it was discovered that blood sugar levels were effectively controlled by insulin signalling and that high blood glucose levels could be mildly remitted intermittently after learning about the interactions between lifestyle nutritional factors and the microbial changes in the gut brought on by the low-glycaemic, high-fibres diet.[7, 8] T2DM and cardiovascular disease (CVD) are included in the category of metabolic disorders, which are also defined by measuring specific biochemical, clinical and metabolic metrics.[9-11] According to a recent study, by reversing certain metabolic dysfunctions, such as increased fat mass, metabolic endotoxemia, tissue inflammation and insulin secretion and sensitivity, these bacterial species can also significantly lower the symptom scores in T2DM animal models.[12] Figure 1 conceptual

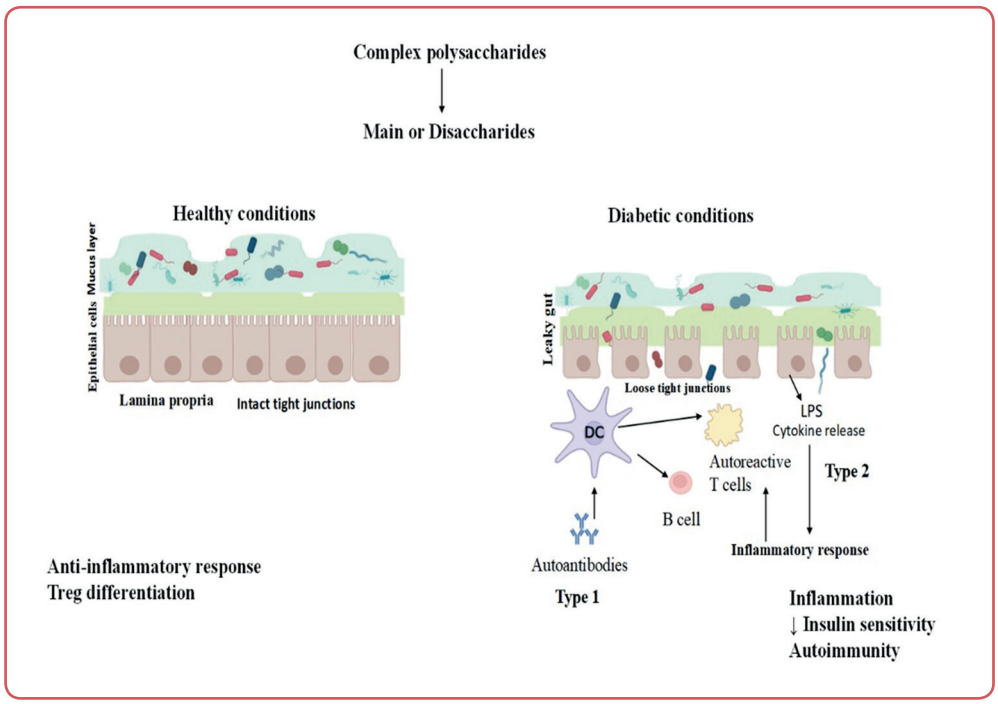

Figure 1: Link between gut microbiome, intestinal permeability and immune activation in diabetes

diagram created using *BioRender* to depict information synthesised from the studies discussed in this review.

Understanding gut microbiota and its vital functions

The gastrointestinal system has a direct and indirect impact on the body's ability to digest complex foods, produce immune cells, synthesis vitamins and convert indigestible carbohydrates into energy. The microbiota is transferred during pregnancy or delivery and stabilises at age two. The development of a stable stomach in neonates and young children is significantly facilitated by (a) the vaginal microbiota after a normal delivery, (b) the skin microbiota during a caesarean surgery, (c) antibiotics and (d) exclusive breastfeeding or formula feeding.[13] Microbiota transfer is often said to happen *in utero* or during delivery and to become stable by about two years old.

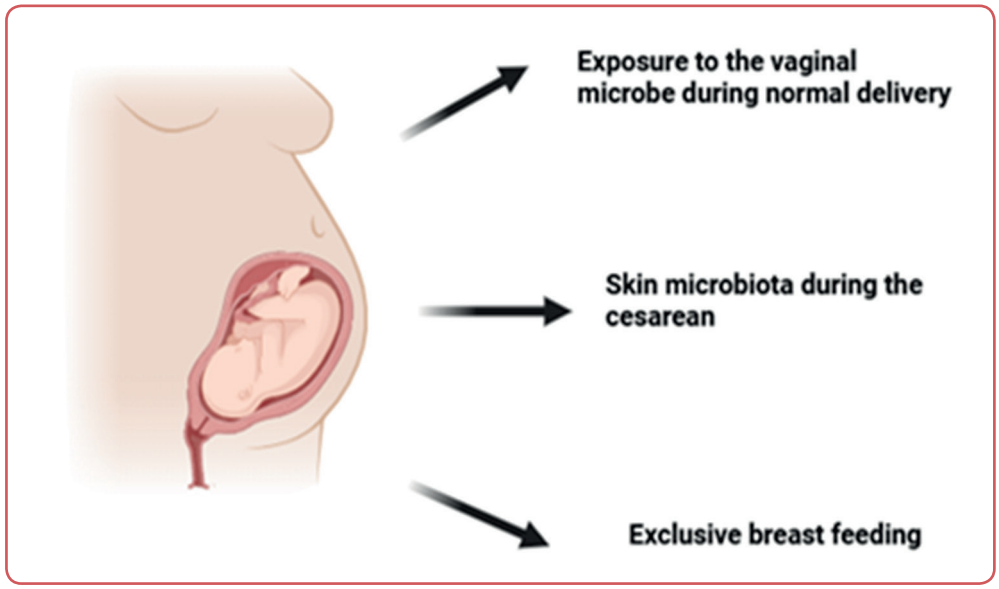

Figure 2: Microbiota transfer occurs in utero or during delivery and stabilises by ~ 2 years of age

However, recent studies show conflicting results. A 2025 study found no microbes in the amniotic fluid of healthy pregnancies from the second trimester to delivery. This suggests there is no ongoing colonisation of microbes in that environment during pregnancy. Meanwhile, work identifying bacterially-derived metabolites within foetal intestinal tissues (at mid-gestation) would suggest that there are microbiome associated products in utero, but not necessarily viable microbes per se. Infants followed longitudinally from preterm and term cohorts show that while the infant GI microbiota does indeed start from different places, it quickly converges to a state more adult-like, but appears to be even less stable than originally thought. Although microbiome stabilisation by age two is well supported, the key controversy is whether colonisation begins before birth or only at delivery, with current evidence favouring the latter. As shown in Figure 2 microbiota transfer occurs in utero or during delivery and stabilises by ~ 2 years of age. The figure was created using *BioRender* based on concepts and data discussed in this review.

Gut microbiota and carbohydrate metabolism

The ileum breaks down polysaccharides into monosaccharides, which are not broken down in the upper gastrointestinal tract, with the aid of bacterial enzymes like glycosidases.[14] After a carbohydrate-rich meal, blood glucose levels should increase; however, the hormones glucagon and insulin tightly control and keep them at a homeostatic level. In the upper digestive tract, specialised proteins called glucose transporters (GLUTs) on epithelial cells help break down and absorb carbohydrates into the bloodstream.[15] A basic explanation for how glucose is absorbed under normal circumstances is the transfer of glucose to the pancreatic β-cells by GLUT proteins. The oxidation of pancreatic glucose causes membrane depolarisation and potassium channel closure, which in turn stimulates insulin secretion. This leads to voltage-dependent calcium influx and insulin exocytosis.[16] The integrity of cells to cells is compromised by a decline in gut microbial diversity, which results in a leaky gut with increased permeability that triggers intestinal inflammation.[17] According to the host–environment axis of immune education, the gut is the area most exposed to different microorganisms and exogenous substances.[18] Some of the factors that contribute to T1DM include a person's genetic composition, host-related factors and external environmental factors. Therefore, the development of severe clinical illnesses like T1DM is significantly influenced by the gut microbiota and its role in the mucosal immune system.[19] Diabetes and immune system dysfunction are thought to be associated with decreased intestinal barrier function and oral tolerance. Complete insulin insufficiency is a hallmark of type 1 diabetes.[20] On the other hand, insulin insufficiency, low insulin production and hyperglycaemia in the fasting serum are characteristics of T2DM, a chronic metabolic disease.[21]

Intestinal flora and T2DM: a hidden connection

People are in a suboptimal state of metabolic health as a result of societal advancements that have led to a higher quantity intake of high-energy foods and less exercise.[22, 23] As prognostic biomarkers, diabetes indicators like venous glucose levels and glycated haemoglobin (HbA_1c) have limitations. We need to find more early predictors. Compared to the human genome, the gene pool in the microbiome is an order of magnitude larger.[24] The host-absorbed gut microbial metabolites affect receptors in the gut, liver, brown adipose tissue (BAT), white adipose tissue (WAT) and central nervous system (CNS) in addition to being involved in intestinal motility, mineral absorption, electrolyte absorption and micro-nutrient synthesis.[25, 26] Human diseases including T2DM and obesity are tightly linked via mutual metabolic and inflammatory pathways. Insulin resistance, chronic low-grade inflammation and oxidative stress are common among both diseases. In obesity, the aggravated mass of adipose tissue liberates pro-inflammatory cytokines (eg TNF-α, IL-6) and oxy fatty acids, which inhibit insulin signalling in liver, muscle and fat tissues. This deficiency in insulin functioning leads to hyperglycaemia and T2DM. Furthermore, gut dysbiosis (imbalance of intestinal microbiome) has been associated with the development of obesity and diabetes though its effects on energy metabolism, intestinal permeability and stimulation of endotoxin-related inflammation. Metabolic stress is exacerbated by mitochondrial dysfunc-

tion and increased reactive oxygen species (ROS) production. The synergy of these processes forms a destructive positive feedback loop of inflammatory, oxidative and metabolic dysregulation ultimately driving the onset and progression of obesity-related T2DM and associated comorbidities including fatty liver disease and cardiovascular disorders.[27-32] Since many researchers are concentrating in changing the intestinal microbiome for improving insulin sensitivity, gastrointestinal-targeted therapies are emerging therapeutic approaches. Modulating human metabolism, inflammation and glucose regulation through modifying the composition and function of intestinal microbes provides new avenues for controlling human diseases such as T2DM, obesity and other related metabolic diseases. Gut flora therapy has emerged as a novel therapeutic approach. Many researchers are interested in altering the intestinal microbiota's composition to improve insulin sensitivity and gut flora therapy has emerged as a cutting-edge therapeutic approach.

Modifications in intestinal microbiota in patients with T2DM and obesity

The primary cause of T2DM is obesity, which is also commonly used as an early warning sign for the condition. Overweight obesity is common in about 86 % of people with T2DM.[33] Studies have linked low gut flora abundance to insulin resistance, dyslipidaemia and excess weight.[34] *Lactobacillus, Prevotella, Bacteroides, Desulfovibrio* and *Oxalobacter spp* are becoming less prevalent in the intestinal microbiota, which may be a contributing factor to the development of metabolic disorders, according to studies on gut microbial diversity in patients with low gene count (LGC) and high gene count (HGC). Increased mucus degradation and a reduction in the potential for the production of hydrogen and methane are the outcomes of these modifications.[35] This metabolic disruption makes people with LGC more prone to metabolic diseases such as prediabetes and T2DM.[36] Additionally, studies on faecal transplantation have shown that obese people with metabolic syndrome have increased insulin sensitivity following the administration of gut microbiota from a slim donor.[37-39] These studies provide a theoretical basis for the connection between gut microbiota and whole-body energy conversion and highlight the vital role of intestinal flora in host metabolism.[40, 41] Recent studies have shown that the human intestinal microbiota of obese individuals differs from that of lean individuals.[42, 43] Research on 416 pairs of twins revealed that the intestinal flora of obese individuals had significantly lower levels of *Christensenellaceae, Dehalobacteriaceae* and SHA-98 than that of lighter-weight individuals.[44] Additionally, recipient mice that receive a transplant of modified *Christensenella minuta* have been demonstrated to have a tendency to lose weight. Furthermore, *Oscillospira* produces short-chain fatty acids (SCFAs) by breaking down host blood glucose. Current studies show that *Oscillospira* and the methanogenic archaeon *Methanobrevibacter smithii* are abundant in healthy weight subjects, which may aid in weight loss.[45-47] T2DM can also affect those who are lean.[48] According to a study on their intestinal flora, lean people had lower abundances of *Akkermansia muciniphila*, which is positively correlated with lower insulin production.[37] Research indicates that *Clostridium, Bifidobacterium, Bacteroides, Eubacterium, Listeria* and *Ruminococcus* are the primary intestinal bacteria that influence the synthesis of secondary bile.[49, 50] Bas has been shown to modify the host's metabolism and energy homeostasis by activating TGR5 and FXR receptors, thereby influencing the intestinal flora.[51] Furthermore, studies have shown that supplementing mice with *Prevotella copri* improves their metabolism of glucose.[52] It has been suggested that the higher richness of *Bacteroides vulgatus* and *Prevotella copri* in IR individuals compared to the normal group may result in an increased potential for BCAA synthesis, even though the abundance of *Butyrivibrio crossotus* and *Eubacterium siraeum* declines, resulting in decreased BCAA catabolism.[27]

The connection between T2DM and gut microbial metabolites

Bile acids (BAs)

Dietary lipids and fat-soluble vitamins are emulsified, absorbed and penetrated through the intestinal lumen with the help of BAs, which are produced in the liver from cholesterol. Intestinal bacteria, which are essential for controlling glucose, primarily metabolise BAs. Additionally, BAs function as signalling molecules that alter metabolic balance and control gut flora.

Targets of BAs

FXR, which is crucial for controlling insulin sensitivity and glucose homeostasis, is principally activated by BA CDCA.[53,54] FGF19 (or FGF15 in mice) transcription is triggered by FXR activation in ileal enterocytes.[55] Liver FXR activation not only inhibits CYP7A1 expression by inducing the expression of small heterodimer partners (SHPs), but it also adversely disrupts glycolysis.[56-58] When intestinal L cells are exposed to glucose, ChREBP and FXR work together to suppress the levels of proglucagon mRNA expression. By blocking the transcription of glycolytic enzymes, FXR's effects on ChREBP also reduce intracellular ATP levels by reducing the secretion of ATP-dependent glucagon-like peptide-1 (GLP-1).[59] It has been hypothesised that FXR activation in the pancreatic b cells raises cytosolic C^{2+} levels and inhibits the KATP current, ultimately leading to increased insulin production.[60,61] By reducing the amount and BSH activity of *Bacteroides fragilis* in type 2 diabetics, metformin raises intestinal concentrations of glycoursodeoxycholic acid (GUDCA). According to this study, the intestinal FXR axis between GUDCA and *Bacteroides fragilis* is how metformin corrects glucose metabolic inefficiency.[62] By activating FXR to mediate an increase in FGF19, Obet cholic acid (OCA, FXR agonist) decreases endogenous BA production in type 2 diabetic NAFLD patients, leading to weight loss and improved insulin sensitivity.[63] Although blood levels of BA and C4 vary greatly from person to person, type 2 diabetic patients have much higher C4 plasma concentrations.[64] By controlling BA and FXR signalling, intestinal microbiota contributes to the pathophysiology of IR and obesity, according to studies conducted on FXR-deficient mice given a high-fat diet. Conversely, by altering the gut microbiome's composition by decreasing *Firmicutes* and increasing *Bacteroidetes*, FXR may be the cause of the rise in adiposity.[65]

Short-chain fatty acids (SCFAs)

Among their many other roles in IR and T2DM, SCFAs, the most well-studied metabolites of gut flora, control immuno-modulatory processes, improve intestinal epithelium integrity, alter insulin release and control pancreatic β-cell proliferation.[66] Three SCFAs are primarily produced by the colon's microbial fermentation of unabsorbed

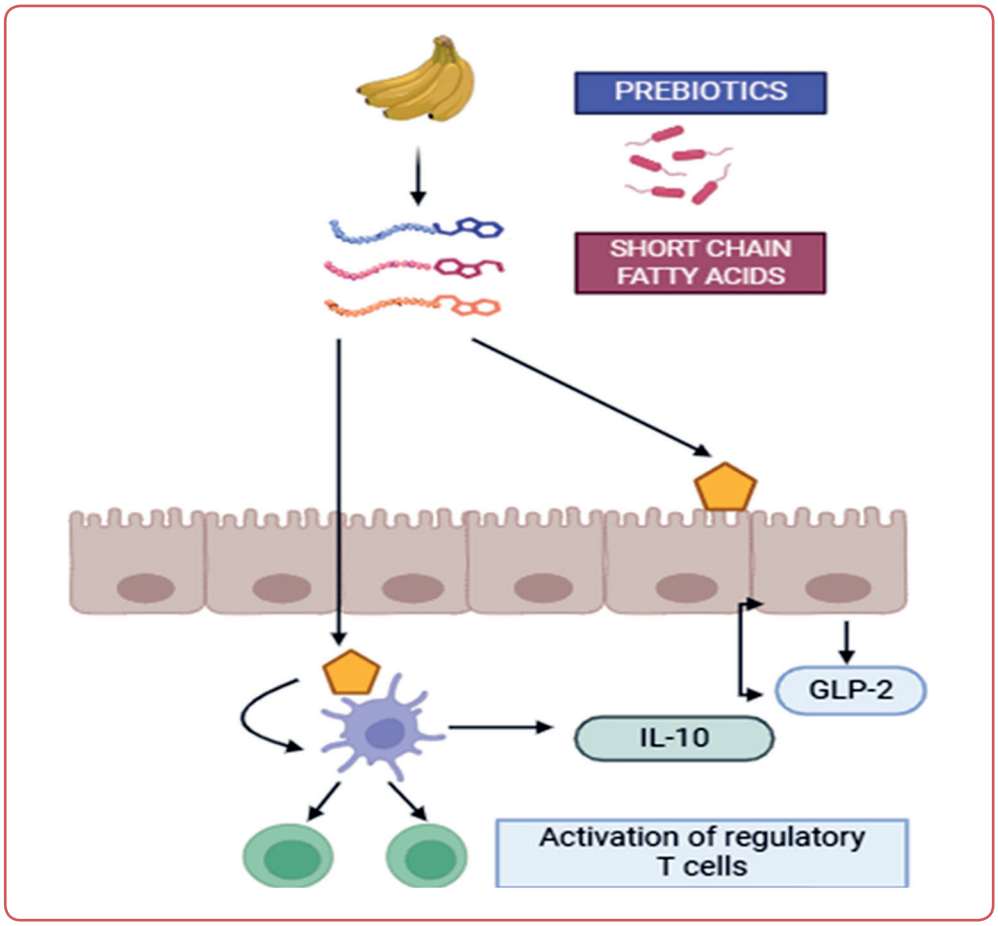

Figure 3: Prebiotics supports intestinal barrier function

dietary components, such as fibre and multiple pathways are involved.[67] The majority of enteric bacteria, including *Clostridium species, Blautia hydrogenotrophica* and *Akkermansia muciniphila,* have been found to produce acetate. On the other hand, it has been determined that *Eubacterium hallii, Eubacterium rectale* and *Roseburia inulinivorans* produce butyrate.[68, 69] As shown in Figure 3 prebiotics supports intestinal barrier function. The figure was created using *BioRender* based on concepts and data discussed in this review.

Amino acids (AAs)

Higher levels of AAs, particularly branched-chain amino acids (BCAAs), have been associated with increased IR and the risk of developing T2DM.[70] Additionally, BCAAs promote the development of IR associated with obesity when consumed as part of an HFD.[71] Because a low-isoleucine diet activates the FGF21-UCP1 axis, which restructures the liver and fat tissue metabolism to improve hepatic insulin sensitivity and energy expenditure, another previous study on BCAAs came to the conclusion that isoleucine plays a crucial role in metabolic well-being. In contrast to low leucine, dietary valine deficiency has comparable but less potent metabolic effects, indicating that dietary isoleucine deficiency is essential for both diabetes prevention and treatment.[72] It's interesting to note that IR can cause aminoacidaemia by compromising the oxidative metabolism of BCAA and causing the breakdown of protein that insulin normally inhibits. This suggests that rather than being the cause of decreased insulin action, elevated BCAA may be a symptom of it. Since their metabolisms differ before the change in glucose homeostasis occurs, aromatic amino acids, like BCAAs, also play a role in the pathophysiology of hyperglycaemia.[73] Both BCAAs and AAAs serve as markers of IR progression in young adults with normoglycaemia, suggesting that IR contributes to their correlation with the risk of diabetes.[74] *Clostridium sporogenes* metabolises all three AAAs in the human gut.[75] High monosodium glutamate (MSG) intake has been shown to be positively correlated with overweight in Chinese people, indicating a link between glutamate and obesity.[76] Bacteroides the taiotaomicron abundance declined in obese individuals and is inversely correlated with blood glutamate levels, according to Liu et al. Lower blood glutamate levels aid in the correction of insulin resistance and hyperglycaemia and bariatric surgery partially reverses these metabolic and microbiological alterations.[47]

Others

The gut microbiota converts dietary choline to trimethylamine. A high-fat diet in-creases the catabolism of choline by *Escherichia coli*, which raises blood levels of trimethylamine N-oxide (TMAO), by altering intestinal epithelial physiology.[77] Furthermore, the intestinal microbiota (primarily *Enterobacteriaceae*) converts TMAO to TMA, which is subsequently expelled from the host and carried to the liver, where hepatic enzymes further convert it to TMAO. However, TMAO affects the growth and metabolism of the gastro-intestinal microbiota in a taxonomic and source-dependent manner.[78] Furthermore, there is a connection between increased serum imidazole propionate concentrations and a decrease in intestinal flora density.[79] The gut flora's production of indole propionic acid, a possible biomarker for the development of T2DM, has been thought to be positively associated with insulin secretion and negatively associated with low-grade inflammation because it maintains the function of islet β-cells.[80]

Treatment options for gastro-intestinal in T2DM and obesity

Probiotics

Probiotics are "the living microbes" that improve the health of the host when given in adequate amounts.[81] It has been demonstrated that *Lactobacillus acidophilus* treatment enhances intestinal barrier function, reduces inflammation, controls intestinal flora and guards against infections.[82] Research on the molecular mechanisms of probiotic intervention in T2DM has shown that probiotics can lower insulin resistance and hyperglycaemia.[83] Probiotics can reduce insulin resistance and hyperglycaemia, according to research on the molecular mechanisms of their intervention in T2DM. Supplements containing *Akkermansia muciniphila* increase anti-inflammatory components such as beta sitosterol and alpha tocopherol and decrease chronic low-grade inflammation.[84] Lactobacillus mucilaginous supplementation increased the insulin sensitivity and glucose tolerance of diabetic mice. Additionally, it has been shown that supplementing with *Lactobacillus acidophilus* improves intestinal barrier function in animal models of diabetes.[85, 86] Additionally, fermented camel milk contained 14 probiotics that improved intestinal barrier

function, SCFA and GLP-1 secretion. All of these probiotics markedly raised blood glucose levels in mice. Furthermore, fermented camel milk contained 14 separate probiotic, multi strain probiotic preparations, all with similar probiotic effects on metabolic health and gut health. These probiotics improved intestinal barrier function, induced SCFAs and were involved in the production of GLP-1, essential components for glucose homeostasis. Fascinating, when tested in experimental studies using mice, it was found that administration of these probiotics greatly affected regulation of blood glucose, which suggests a promising role in the glycaemic modulation, inducing an improvement on metabolic function.[87] According to a randomised, double-blind, placebo-controlled study, probiotics help people with T2DM improve their glucose metabolism, but drinking fermented milk has other metabolic effects.[88] The FDA and EFSA have yet to issue probiotic approval statements, despite the fact that probiotics should be recognised for their role in T2DM (Figure 4).[89-91]

Herbal medicines

There is growing evidence that a number of herbs or their natural components may be able to treat T2DM by changing the intestinal flora. *Radix scutellariae* can eliminate heat and moisture, cure jaundice and quench thirst. A recent pharmacological study found that *scutellaria* produces hypoglycaemic and lipid-regulating effects by either increasing the excretion of BA in the faces or decreasing intrahepatic cholestasis.[92] Studies show that baicalin lowers diabetes by altering the way intestinal flora and BAs interact; FXR may be the mediating factor in this effect.[93] It has been discovered that liquorice extract reduces intestinal inflammation linked to endotoxemia. Additionally, glycyrrhiza extract alters the gut microbiome's composition by decreasing the *Lachnospiraceae* NK4A136 group and increasing *Bacteroides* and *Akkermansia.* These results imply that alterations in the gut microbiota in hyperglycaemic mice may be the main factor causing liquorice extracts hypoglycaemic action.[94] Experimental evidence supports the antidiabetic effects of the *Scutellaria coptis* herb pair (SC), a traditional herbal remedy for diabetes that has been shown to have hypoglycaemic and anti-inflammatory effects through the TLR4 signalling pathway, which are ascribed to gut microbiota modulation.[95] Rich-polyphenol extract from *Dendrobium loddigesii* (DJP) has been used to treat diabetic db/db mice. This could be because DJP helps mice with diabetes symptoms and complications by lowering oxidative stress and inflammation and enhancing the bal-

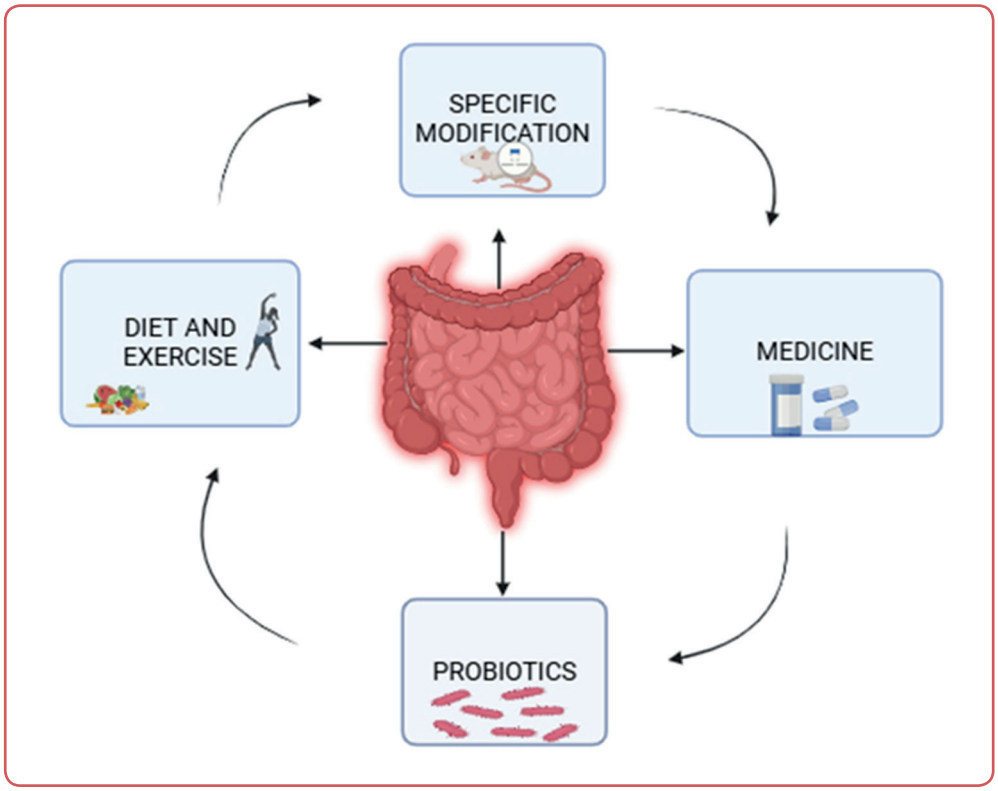

Figure 4: Therapeutic approaches for type 2 diabetes mellitus (T2DM)

ance of intestinal flora.[96] The main way that GQD for T2DM reduces intestinal inflammation and lowers blood glucose is by altering the makeup of the entire gut flora and boosting it with a variety of bacteria that produce butyrate-forming bacteria.[97] Berberine, a potentially significant active pharmaceutical component of GQD that acts as an active nitrogenous compound to produce pharmacological impact by controlling the gut bacteria, is also derived from *Rhisome coptidis*.[98] In a rat model of T2DM, berberine has been shown to protect insulin target organs, increase the number of beneficial bacteria and decrease potentially harmful bacteria by reducing the incursion of inflammatory biological units and preventing the onset of a widespread inflammatory response.[99] In a rat model of T2DM, ginsenosides have been shown to modify intestinal flora, reducing intestinal mucosal damage and a range of inflammatory responses.[100, 101] Purified citrus polymethoxyflavone-rich extract (PMFE) significantly increases *Bacteroides ovatus* abundance. Weight loss, decreased BCAA levels and MetS alleviation are associated with PMFE enrichment of *Bacteroides ovatus*.[102] Rb1 increases the number of *Akkermansia spp* to maintain glucose homeostasis, which may be related to alanine modification.[103] It is important to recognise the role that herbal remedies have played in enhancing the microbiota in the stomach. However, from the perspective of modern science, more investigation into the underlying mechanism is required. The effects of most herbal medicines on microbiota are associated with changes in gut flora structure, an increase in beneficial bacteria and butyrate levels in the gut and the inhibition of opportunistic pathogens. More evidence from human studies is needed because most of the results are based on animal testing and the underlying theories still need to be looked into.

Diet and exercise

According to research, 80 % of individuals with T2DM and obesity saw a remission of their diabetes mellitus after reducing their caloric intake by roughly 15 kg. Additionally, there is a correlation between this remission and the amount of weight loss.[104] Therefore, it is particularly important for people with T2DM to maximise their carbohydrate intake and boost their absorption of roughage supplements. It has long been known that fibre is important for regulating metabolism and preventing chronic gastrointestinal disorders.[105] The lower fibre intake in the Western diet, especially in developed countries, is closely associated with the higher prevalence of metabolic disease states. Thus, dietary fibre treatments that alter the microbiota can improve health. *Roseburia, Faecalibacterium prausnitzii* and *Eubacterium rectale* are among the taxa that are linked to an increase in total SCFAs when a diet is more plant-based.[106] SCFAs, which have the capacity to lower cholesterol and control blood sugar, are produced when bacteria in the intestine break down fibre.[107] Fiber has been shown to increase short chain fatty acids and *Bifidobacterium* levels while decreasing HbA1c.[108] According to a consistent study, almond supplements can lower body weight index and red blood pigment in individuals with T2DM while increasing microbes that produce short-chain fatty acids.[109] The plant-rich Mediterranean diet causes specific changes in intestinal flora and BCAA metabolism, which progressively raise phytochemicals and lead to increased insulin sensitivity. *Prevotella* abundance and branched chain amino acid catabolism have increased, while *Bifidobacterium* abundance and biological synthesis of branched chain amino acids have decreased.[110] Similarly, increasing physical activity and fitness is another important factor in lowering T2DM. Physical activity is necessary to lower blood sugar levels and boost the effectiveness of insulin.[111] A previous study found that exercise increases the diversity of *Akkermansia muciniphila* and gastric bacteria in athletes' intestinal flora.[112] Regular exercise has also been demonstrated to promote IR in skeletal muscle by altering the gastrointestinal microbiota's composition, boosting short-chain fatty acid synthesis, increasing blood's liquid matrix and raising SCFA concentrations.[113] Furthermore, a rise in microbes that produce short-chain fatty acids and improvements in intestinal barrier integrity brought on by exercise are significant factors in the management of diabetes.[114] It is suggested that SCFAs may contribute to improving locomotor function because studies have shown a positive association between taxa that produce SCFAs and changes in mass and movement.[115-122]

Conclusion

Diabetes is known to be induced by the interaction of several determinants, including genetic susceptibility, diet, exercise, smoking and stress. The relationship between food and gut bacteria determines the production and ab-

sorption of many metabolites. Based on patient characteristics and the risk of complications, people with T2DM can be divided into several batches. Individuals with prediabetes also displayed variations in the pathophysiology of various etiological groups. Different patient subgroups may experience quite different outcomes from the same treatment. Therefore, it is now feasible to predict how the disease will progress and how each patient will respond to the right treatment plan thanks to the early detection of patients with different T2DM characteristics. More people have recently become aware of the importance of the gut microbiota. Microbiology is one of the most active fields of biomedicine today because human-symbiotic bacteria are vital to both health and illness. The microbial "organ" of the intestinal flora plays a part in regulating human health and metabolism. Microbially directed interventions for diabetes fall into two categories: nontargeted therapy and targeted therapy. Nontargeted treatments that enhance the general makeup and function of the flora include FMT, probiotics, exercise and customised diet. Examples of targeted therapies include genetically modified microorganisms and drugs that target the metabolism of specific bacteria that function in relation to specific changes in the flora related to metabolism. Numerous animal tests and clinical trials have shown that different basal class categories respond quite differently to gastrointestinal-targeting medication strategies. This discrepancy may be directly related to the individual's gastrointestinal flora composition. Therefore, a critical prerequisite for the use of microbial therapeutics is knowledge of the process that causes illness as well as the typical bacterial magnitude of an individual. It also acts as the basis for the transplanted microorganisms successful and tailored colonisation. Overall, gut flora is a promising therapeutic target for T2DM. In light of the causal relation that has been shown to exist between gut microbiota and T2DM, we believe that the gut microbiome may serve as both a new therapeutic target and a position biomarker for diagnosing T2DM. Interventions tailored to the gut microbial composition of an individual can lead to more accurate and effective management strategies for T2DM patients.

Ethics

This study was a secondary analysis based on the currently existing data and did not directly involve with human participants or experimental animals. Therefore, the ethics approval was not required in this paper.

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Conflicts of interest

The authors declare that there is no conflict of interest.

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Data access

The data that support the findings of this study are available from the corresponding author upon reasonable individual request.

Author ORCID numbers

Navreet Kaur (NeK):
0009-0007-2741-5483
Navjot Kaur (NoK):
0000-0001-5230-3191
Aanchal Verma (AV):
0009-0000-3806-5991
Rahul Kumar Sharma (RKS):
0000-0002-9678-6882

Author contributions

Conceptualisation: NeK, NoK, AV, RKS
Validation: NeK, NoK, AV, RKS
Formal analysis: NeK
Investigation: NeK
Data curation: NeK
Writing - original draft: NeK
Writing - review and editing: NeK, NoK, AV, RKS
Visualisation: NeK
Supervision: NoK, AV, RKS.

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