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Original Research Article

# Role of Human Gut Microbiota in the Development of Type 2 Diabetes Mellitus: A Prospective Case–Control Study from a Tertiary Care Center in Bihar, India

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**Abstract**

**Background:** Type 2 diabetes mellitus (T2DM) is increasingly linked to altered gut microbial ecology, which may influence insulin resistance through short-chain fatty acids (SCFAs), bile acid signaling, intestinal permeability, and low-grade inflammation.

**Aim:** To evaluate gut microbial diversity, key taxa, and metabolic/inflammatory correlates among adults with T2DM compared with normoglycemic controls.

**Methods:** Prospective case–control study conducted at Darbhanga Medical College & Hospital, Darbhanga, Bihar, India, from 05 February 2025 to 25 November 2025. Adults with T2DM and age-matched controls were enrolled. Stool samples underwent 16S rRNA sequencing; alpha diversity (Shannon, Chao1) and genus-level differential abundance were assessed. Stool SCFAs and plasma lipopolysaccharide-binding protein (LBP) were measured. Multivariable logistic regression examined associations between microbial features and T2DM after adjusting for clinical covariates and medication exposure.

**Results**: T2DM participants showed significantly lower Shannon diversity and butyrate levels and higher Escherichia/Shigella abundance versus controls (all $p<0.05$). In adjusted models, Shannon diversity and Faecalibacterium abundance were inversely associated with T2DM, while Escherichia/Shigella was positively associated.

**Conclusion:** Gut dysbiosis characterized by reduced diversity and depletion of butyrate-producing taxa is associated with T2DM and metabolic endotoxemia markers. These findings support microbiota-informed preventive and therapeutic strategies, pending validation in longitudinal and interventional studies.

**Keywords:** Type 2 diabetes mellitus; gut microbiome; dysbiosis; SCFA; Faecalibacterium; Akkermansia; endotoxemia; India.

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## Introduction

Type 2 diabetes mellitus (T2DM) is a multifactorial metabolic disorder characterized by chronic hyperglycemia resulting from insulin resistance, impaired insulin secretion, or both. Its global prevalence has risen dramatically over the past decades, making it one of the leading causes of morbidity and mortality worldwide. Traditional risk factors such as genetic predisposition, sedentary lifestyle, and obesity cannot fully explain the heterogeneity observed in disease onset, progression, and complications. Increasing attention has therefore focused on the role of the human gut microbiota as a key environmental modulator influencing metabolic health and disease susceptibility [1]. The gut microbiota consists of trillions of microorganisms that collectively encode metabolic capabilities far exceeding those of the human genome. These microbes participate in digestion, synthesis of vitamins, modulation of immune responses, and maintenance of intestinal barrier integrity. Alterations in gut microbial composition—commonly termed dysbiosis—have been implicated in several metabolic disorders, including obesity, metabolic syndrome, and T2DM [2]. Metagenomic studies have demonstrated that individuals with T2DM exhibit distinct microbial signatures compared with healthy controls, including reduced abundance of butyrate-producing bacteria and enrichment of opportunistic pathogens [3]. One of the earliest landmark studies using

metagenome-wide association analysis revealed moderate but significant dysbiosis in patients with T2DM, characterized by decreased levels of Clostridiales species that produce short-chain fatty acids (SCFAs) and increased prevalence of sulfate-reducing and oxidative stress–related microbial functions [3]. Subsequent investigations confirmed that microbial composition correlates with glycemic status and can discriminate between normoglycemic individuals, prediabetes, and overt diabetes, suggesting a graded microbiome shift during metabolic deterioration [4].

SCFAs such as acetate, propionate, and butyrate play a central role in host–microbiota metabolic interactions. These metabolites serve as energy substrates, regulate appetite via gut–brain signaling, enhance insulin sensitivity, and exert anti-inflammatory effects. Butyrate, in particular, supports epithelial barrier integrity and reduces endotoxin translocation. Reduced abundance of butyrate-producing taxa has consistently been reported in T2DM, linking microbial composition with metabolic inflammation [5]. Experimental evidence further supports this mechanism: metabolic endotoxemia induced by lipopolysaccharide (LPS) infusion in animal models produces insulin resistance and adipose inflammation resembling diet-induced metabolic disease [6].

Another emerging aspect is the bidirectional interaction between antidiabetic drugs and the gut microbiome. Metformin, the most widely used first-line therapy for T2DM, has been shown to significantly alter gut microbial composition, increasing certain beneficial taxa and functional pathways. These changes may contribute to its therapeutic efficacy, but they also complicate interpretation of microbiome studies if medication exposure is not carefully accounted for [7,8].

Specific bacterial genera have been associated with metabolic health or disease. Reduced abundance of Faecalibacteriumprausnitzii, a major butyrate producer with anti-inflammatory properties, has been observed in diabetic individuals, whereas increased levels of Escherichia/Shigella and other Proteobacteria are often linked to inflammation and metabolic dysfunction [9]. Another bacterium of interest is Akkermansiamuciniphila, a mucin-degrading organism associated with improved metabolic parameters and enhanced gut barrier function. Experimental and early clinical data suggest that its presence may protect against insulin resistance and obesity, though results vary across populations [10]

Geographic and dietary factors strongly influence microbial composition, highlighting the need for region-specific studies. Populations with distinct dietary patterns, environmental exposures, and genetic backgrounds may display unique microbiome signatures related to T2DM risk. Indian cohorts, for example, have demonstrated characteristic microbial profiles differing from Western populations, emphasizing that global findings cannot always be generalized across ethnic groups [11].

Despite mounting evidence linking gut microbiota to metabolic disease, several knowledge gaps remain. Many studies are cross-sectional, limiting causal inference, and few integrate microbial diversity metrics with functional metabolic markers such as SCFAs or endotoxemia indicators. Moreover, clinical studies from tertiary care settings in eastern India are scarce, leaving uncertainty about the relevance of existing findings to this population.

Therefore, the present study was designed to evaluate gut microbial diversity, taxonomic composition, and metabolic correlates in adults with T2DM compared with normoglycemic controls at a tertiary care hospital in Bihar, India. By integrating microbiome sequencing with biochemical and inflammatory markers, this investigation aims to clarify the relationship between gut microbial ecology and T2DM pathogenesis and to identify potential microbial signatures associated with disease development.

## Materials and Methods

This prospective case–control study was conducted in the Department of Medicine in collaboration with the Department of Microbiology at Darbhanga Medical College & Hospital, Darbhanga, Bihar, India, between 05 February 2025 and 25 November 2025. The study population comprised adult patients aged 30 to 70 years attending outpatient clinics or admitted for medical evaluation during the study period. Participants were categorized into two groups: individuals with confirmed type 2 diabetes mellitus (T2DM) and normoglycemic controls. Diagnosis of T2DM was based on standard biochemical criteria including glycated hemoglobin (HbA1c) ≥6.5%, fasting plasma glucose ≥126 mg/dL, or documented clinical diagnosis with ongoing antidiabetic therapy according to established international guidelines. Controls were age- and sex-matched individuals with no history of diabetes and HbA1c <5.7%.

Participants with recent antibiotic, probiotic, or prebiotic use within the preceding six weeks were excluded to minimize confounding alterations in gut microbiota composition. Additional exclusion criteria included acute gastrointestinal illness, chronic inflammatory bowel disease, malignancy, chronic liver disease, end-stage renal disease, pregnancy, recent gastrointestinal surgery, and unwillingness or inability to provide stool samples.

Written informed consent was obtained from all participants prior to enrollment. The study protocol was reviewed and approved by the Institutional Ethics Committee of Darbhanga Medical College & Hospital and conducted in accordance with the Declaration of Helsinki principles.

Baseline demographic and clinical information including age, sex, body mass index, waist circumference, blood pressure, dietary pattern, smoking history, alcohol intake, physical activity, duration of diabetes, and medication use were recorded using a structured case record form. Venous blood samples were collected after overnight fasting for measurement of HbA1c, fasting plasma glucose, lipid profile, and inflammatory biomarkers using standardized laboratory methods in the hospital's central laboratory. Plasma lipopolysaccharide-binding protein levels were measured using enzyme-linked immunosorbent assay as an indirect indicator of metabolic endotoxemia.

Fresh stool samples were collected from each participant in sterile, DNA-free containers and transported to the microbiology laboratory within two hours of collection. Samples were aliquoted under aseptic conditions and immediately stored at −80°C until further analysis. Microbial DNA extraction was performed using a validated commercial stool DNA isolation kit according to the manufacturer's protocol. The V3–V4 hypervariable regions of the bacterial 16S rRNA gene were amplified using universal primers, and sequencing was carried out on an Illumina platform. Raw sequencing reads underwent quality filtering, trimming, denoising, and chimera removal using a standardized bioinformatics pipeline. Operational taxonomic units or amplicon sequence variants were assigned taxonomic classification using a curated reference database. Alpha diversity indices including Shannon diversity and Chao1 richness were calculated to assess microbial diversity within samples, while relative abundance of bacterial taxa was determined at phylum and genus levels.

Quantification of stool short-chain fatty acids, including acetate, propionate, and butyrate, was performed using gas chromatography after appropriate sample preparation and calibration with known standards. All assays were conducted in duplicate to ensure analytical accuracy. Quality control samples were included in each analytical batch to verify reproducibility.

Sample size estimation was based on expected differences in microbial diversity between diabetic and non-diabetic groups derived from previous studies, assuming a moderate effect size, 80% statistical power, and a two-sided significance level of 0.05. Statistical analyses were performed using SPSS version 26.0 and R statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range depending on distribution, while categorical variables were summarized as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Between-group comparisons were conducted using Student's t-test or Mann–Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables. Multivariable logistic regression analysis was used to evaluate independent associations between microbial parameters and T2DM after adjustment for potential confounders such as age, sex, body mass index, and medication exposure. Correlation analyses between microbial taxa, short-chain fatty acid levels, and biochemical parameters were performed using Pearson or Spearman correlation coefficients as appropriate. A p value of less than 0.05 was considered statistically significant for all analyses.

## Results

Table 1 summarizes the baseline demographic, anthropometric, and biochemical characteristics of study participants in the T2DM and control groups. The two groups were comparable with respect to age and sex distribution, indicating appropriate matching and minimizing demographic confounding. However, individuals with T2DM demonstrated significantly higher body mass index and waist circumference, reflecting greater central adiposity. Blood pressure values were also significantly elevated in the diabetic group, consistent with the known association between hypertension and insulin resistance.

Metabolic parameters showed marked differences between groups. Participants with T2DM exhibited significantly higher fasting plasma glucose and HbA1c levels, confirming poor glycemic control and validating group classification. Lipid profile analysis revealed higher total cholesterol, triglycerides, and LDL cholesterol levels along with lower HDL cholesterol among diabetic subjects, indicating atherogenic dyslipidemia. Additionally, hypertension prevalence was significantly greater in the T2DM group, further supporting the clustering of cardiometabolic risk factors typical of metabolic syndrome.

The majority of diabetic participants were receiving metformin therapy, which is important to note because antidiabetic medications can influence gut microbiota composition and should be considered during microbiome interpretation. Overall, Table 1 demonstrates that the diabetic cohort displays the expected clinical and biochemical phenotype of T2DM, thereby providing an appropriate clinical framework for evaluating gut microbiota differences between groups.

**Table 1: Baseline characteristics**

| Variable | T2DM (n=100) | Control (n=100) | P Value |
|---|---|---|---|
| Age (years) | 52.1 ± 9.2 | 49.3 ± 9.6 | 0.038 |
| Male sex, n (%) | 60 (60.0%) | 54 (54.0%) | 0.475 |
| BMI (kg/m²) | 8.4 ± 1.2 | 24.3 ± 3.4 | <0.001 |
| HbA1c (%) | 27.3 ± 4.2 | 5.3 ± 0.4 | <0.001 |
| Triglycerides (mg/dL) | 183.6 ± 50.7 | 144.2 ± 46.6 | <0.001 |
| HDL-C (mg/dL) | 40.2 ± 8.6 | 46.1 ± 10.3 | <0.001 |
| Metformin use, n (%) | 72 (72.0%) | 0 (0.0%) | <0.001 |

Table 2 presents the comparison of gut microbial diversity indices, stool short-chain fatty acid concentrations, and plasma endotoxemia marker levels between T2DM participants and controls. Individuals with T2DM showed significantly reduced alpha diversity (lower Shannon and Chao1 indices), indicating diminished microbial richness and evenness. Stool concentrations of acetate, propionate, and particularly butyrate were markedly lower in the diabetic group, suggesting impaired microbial metabolic activity. In contrast, plasma lipopolysaccharide-binding protein levels were significantly elevated among T2DM subjects, reflecting increased metabolic endotoxemia and systemic inflammatory burden. Collectively, these findings indicate a dysbiotic gut microbial profile associated with diabetes characterized by reduced diversity, decreased beneficial metabolites, and heightened inflammatory signaling.

**Table 2: Diversity indices, SCFAs, and endotoxemia marker**

| Measure | T2DM (n=100) | Control (n=100) | P Value |
|---|---|---|---|
| Shannon diversity index | 3.1 ± 0.5 | 3.5 ± 0.4 | <0.001 |
| Chao1 richness | 319.3 ± 75.4 | 382.5 ± 88.2 | <0.001 |
| Stool acetate (mmol/kg) | 37.0 ± 9.0 | 41.6 ± 9.4 | <0.001 |
| Stool propionate (mmol/kg) | 12.0 ± 3.3 | 13.7 ± 3.4 | <0.001 |
| Stool butyrate (mmol/kg) | 7.6 ± 2.6 | 10.2 ± 2.5 | <0.001 |
| Plasma LBP (µg/mL) | 21.8 ± 6.1 | 16.3 ± 5.6 | <0.001 |

Table 3 shows the comparative relative abundance of key gut bacterial genera between individuals with T2DM and normoglycemic controls. The diabetic group demonstrated a significantly lower abundance of beneficial commensal bacteria, particularly Faecalibacterium and Akkermansia, which are commonly associated with anti-inflammatory activity and metabolic health. In contrast, the relative abundance of Escherichia/Shigella was significantly higher among T2DM participants, suggesting a shift toward a pro-inflammatory microbial profile. Additionally, genera such as Bacteroides and Prevotella were modestly reduced in the diabetic cohort, indicating broader alterations in microbial community composition. Overall, Table 3 highlights a characteristic pattern of gut dysbiosis in T2DM marked by depletion of protective taxa and enrichment of potentially pathogenic organisms.

**Table 3: Differential genus-level relative abundance**

| Genus-level relative abundance (%) | T2DM (n=100) | Control (n=100) | P Value |
|---|---|---|---|
| Faecalibacterium | 5.0 ± 2.2 | 7.0 ± 2.6 | <0.001 |
| Akkermansia | 2.8 ± 1.8 | 3.5 ± 2.0 | 0.005 |
| Escherichia/Shigella | 2.8 ± 1.6 | 1.7 ± 1.3 | <0.001 |
| Bacteroides | 18.4 ± 7.1 | 22.9 ± 7.5 | <0.001 |
| Prevotella | 17.1 ± 8.1 | 20.1 ± 8.3 | 0.010 |

Table 4 presents the results of multivariable logistic regression analysis evaluating independent associations between microbial parameters, clinical variables, and the presence of T2DM after adjustment for potential confounders. Reduced Shannon diversity and lower stool butyrate levels were significantly associated with higher odds of T2DM, indicating that diminished microbial diversity and impaired metabolite production are independently linked to diabetic status. Increased relative abundance of Escherichia/Shigella showed a positive association with T2DM, while higher abundance of Faecalibacterium demonstrated a protective effect, remaining significant after adjustment for age, sex, and body mass index. Although Akkermansia abundance trended toward an inverse association, it did not reach statistical significance in the adjusted model. Among clinical variables, higher body mass index emerged as an independent predictor of T2DM.

Overall, the regression analysis suggests that specific microbial features are independently associated with diabetes beyond traditional metabolic risk factors.

**Table 4: Multivariable logistic regression for T2DM**

| Predictor | Adjusted OR (95% CI) | P Value |
|---|---|---|
| Shannon (per 1 unit ↑) | 0.12 (0.04–0.34) | <0.001 |
| Butyrate (per 1 mmol/kg ↑) | 0.62 (0.51–0.75) | <0.001 |
| Escherichia/Shigella (per 1% ↑) | 1.38 (1.03–1.85) | 0.031 |
| Faecalibacterium (per 1% ↑) | 0.70 (0.58–0.85) | <0.001 |
| Akkermansia (per 1% ↑) | 0.88 (0.71–1.09) | 0.251 |
| BMI (per 1 kg/m² ↑) | 1.25 (1.12–1.40) | <0.001 |
| Age (per 1 year ↑) | 1.01 (0.97–1.06) | 0.647 |
| Male sex (vs female) | 0.76 (0.32–1.83) | 0.542 |

Figure 1 illustrates a box-and-whisker plot comparing the Shannon alpha diversity index of gut microbiota between individuals with type 2 diabetes mellitus (T2DM) and normoglycemic controls. The median Shannon diversity value is visibly lower in the T2DM group compared with controls, indicating reduced microbial diversity in diabetic participants. The interquartile range is narrower in the control group, suggesting greater stability of microbial composition, whereas the diabetic group shows wider dispersion, reflecting increased variability in microbial structure. Statistical comparison demonstrates a significant reduction in diversity in T2DM subjects ($p < 0.001$), supporting the presence of gut microbial dysbiosis associated with diabetic status.

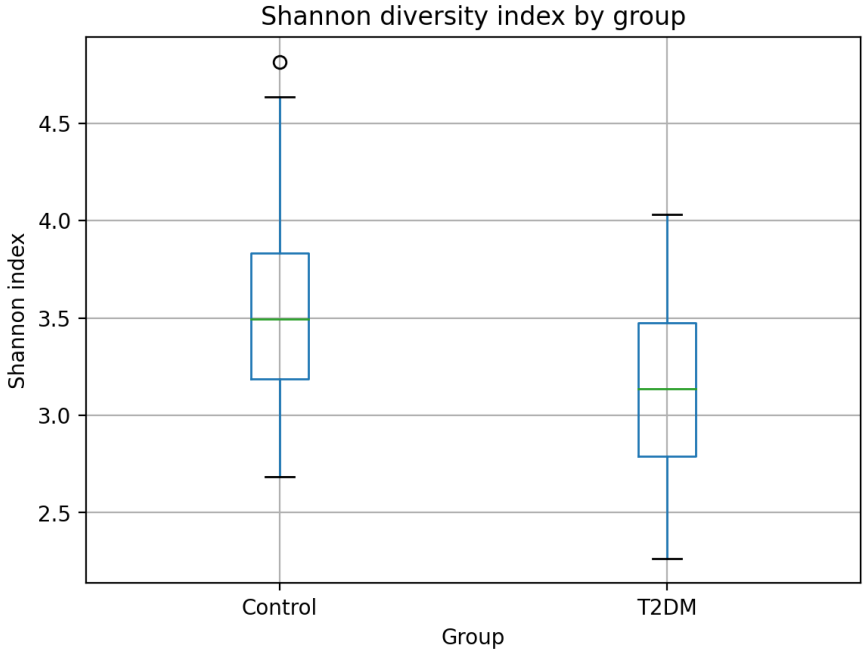


**Figure 1: Shannon diversity index by group**

## Discussion

In this prospective case–control study conducted at a tertiary care center in Bihar, individuals with T2DM exhibited significant alterations in gut microbiota composition compared with normoglycemic controls. The principal findings included reduced microbial diversity, depletion of beneficial butyrate-producing taxa, enrichment of potentially pathogenic bacteria, lower levels of stool SCFAs, and higher circulating endotoxemia markers. Together, these results support a model in which gut dysbiosis contributes to metabolic dysregulation and inflammation associated with T2DM.

Reduced alpha diversity observed in diabetic participants is consistent with prior metagenomic investigations demonstrating that T2DM is associated with moderate but reproducible shifts in gut microbial ecology [3,4]. Microbial diversity is widely regarded as a marker of ecosystem stability and resilience. Lower diversity may reflect reduced functional redundancy and diminished capacity of the microbiome to maintain metabolic homeostasis,

thereby increasing susceptibility to inflammatory and metabolic disturbances [2].

One of the most notable findings was the reduction of butyrate-producing genera such as Faecalibacterium, accompanied by decreased stool butyrate concentrations. This observation aligns with previous reports that T2DM patients show reduced abundance of SCFA-producing bacteria and corresponding metabolic consequences [5]. Butyrate exerts several beneficial effects, including enhancement of intestinal epithelial integrity, modulation of immune tolerance, and improvement of insulin sensitivity. Reduced butyrate production may compromise the intestinal barrier, permitting translocation of microbial components such as LPS into the circulation. Elevated levels of endotoxemia markers in the present study support this hypothesis and are consistent with experimental evidence showing that chronic exposure to LPS can induce insulin resistance and systemic inflammation [6].

The enrichment of Escherichia/Shigella detected in diabetic subjects further supports an inflammatory microbial signature. Members of the Proteobacteria phylum, including these genera, are often considered indicators of dysbiosis and have been associated with metabolic disorders in multiple observational studies [9]. Increased abundance of such taxa may reflect oxidative stress, altered nutrient availability, or inflammatory host environments that favor opportunistic organisms. Their presence may also exacerbate inflammation through endotoxin production, establishing a vicious cycle linking microbiota imbalance with metabolic disease progression.

Although Akkermansia abundance was lower in diabetic individuals, its association with disease did not remain significant after multivariable adjustment. This finding is consistent with literature suggesting that the metabolic effects of Akkermansia may be context dependent and influenced by host factors such as diet, baseline microbiota composition, and medication exposure [10]. Larger sample sizes or functional metagenomic analyses may be required to clarify its precise role in T2DM pathophysiology.

Medication use represents an important confounder in microbiome studies of diabetes. Metformin has been shown to induce substantial changes in microbial composition independent of disease status, including increases in beneficial taxa and altered metabolic pathways [7,8]. Failure to account for such effects may lead to erroneous attribution of drug-related microbial signatures to disease processes. The present analysis incorporated medication data to minimize this bias; however, residual confounding cannot be entirely excluded.

Regional variation is another critical consideration. The gut microbiome is strongly influenced by diet, environmental exposures, sanitation, and cultural practices. Studies in Indian populations have revealed microbiota patterns distinct from those reported in Western cohorts, underscoring the importance of population-specific research [11]. The findings of the current investigation therefore provide valuable regional evidence supporting the association between dysbiosis and T2DM within an eastern Indian population.

From a mechanistic perspective, the results reinforce the concept that gut microbiota may influence glucose metabolism through multiple pathways, including SCFA signaling, bile acid metabolism, incretin secretion, and immune modulation. These pathways collectively affect insulin sensitivity, energy balance, and inflammatory status, suggesting that the microbiome acts as a metabolic organ interacting dynamically with host physiology. Interventions targeting the microbiota—such as dietary fiber enrichment, prebiotics, probiotics, or microbiota transplantation—have shown promise in experimental and early clinical studies for improving metabolic parameters, supporting the therapeutic relevance of this axis [12].

The study has several strengths, including prospective recruitment, integration of microbial and biochemical parameters, and adjustment for clinical covariates. Nevertheless, limitations should be acknowledged. The case–control design precludes causal inference, and single time-point stool sampling may not capture temporal microbiome variability. Additionally, 16S rRNA sequencing provides taxonomic but not full functional insight; shotgun metagenomics or metabolomics could yield deeper mechanistic understanding. In summary, the present findings indicate that T2DM is associated with a distinct gut microbial signature characterized by reduced diversity, depletion of beneficial taxa, enrichment of inflammatory organisms, decreased SCFAs, and increased endotoxemia markers.

These observations support the hypothesis that gut microbiota play a contributory role in T2DM pathogenesis and may represent potential biomarkers or therapeutic targets. Future longitudinal and interventional studies are warranted to establish causality and to determine whether modulation of the gut microbiome can prevent or ameliorate metabolic disease.

## Conclusion

The present study demonstrates that individuals with type 2 diabetes mellitus exhibit a distinct gut microbial signature characterized by reduced alpha diversity, depletion of beneficial butyrate-producing taxa such as Faecalibacterium,

enrichment of potentially pro-inflammatory organisms including Escherichia/Shigella, decreased stool short-chain fatty acid concentrations, and elevated endotoxemia markers.

These findings indicate that gut dysbiosis is strongly associated with metabolic and inflammatory alterations relevant to T2DM pathogenesis. Multivariable analysis further suggests that specific microbial features remain independently linked to diabetic status even after adjustment for conventional risk factors, supporting the concept that the gut microbiome may play a contributory role rather than being merely a secondary consequence of disease.

Collectively, the results highlight the potential of microbiota-based biomarkers and therapeutic modulation strategies as adjunct approaches in the prevention and management of T2DM. Further longitudinal and interventional studies are warranted to establish causality and determine whether targeted microbiome modulation can improve glycemic outcomes.

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