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Gut Microbiota-Brain Axis in Type 2 Diabetes Mellitus: Association with Neuroinflammation and Cognitive Dysfunction

Ahsan Ali Khan[1]*, Syeda Raqiba Gilani[2], Faiza Mumtaz[3], Ammara Afzal**[4], Zunaira Fatima[5], Tareq Nayef AlRamadneh[6], Hafiza Madiha Jaffar[7], Dr. Asma Yawar[8]

[1] Department of Biosciences, Shaheed Zulfikar Ali Bhutto Institute of Science and Technology (SZABIST), Pakistan, ahsan0314.aaa@gmail.com
[2] City Hospital Multan, Pakistan, Raqibagilani92@gmail.com, ORCID: 0009-0005-8553-2085
[3] Department of Pharmacy, The Superior University, Lahore, Pakistan, faiza.mumtaz@superior.edu.pk
[4] Madina College of Pharmacy, Faculty of Pharmaceutical Sciences, Pakistan, ammara.mugal@gmail.com
[5] Postgraduate Trainee FCPS General Medicine, Islam Teaching Hospital, Pasrur Road, Sialkot (CPSP Lahore), Pakistan, dr.zunairahammad@gmail.com
[6] Faculty of Allied Medical Sciences, Hourani Center for Applied Scientific Research, Al-Ahliyya Amman University, Amman, Jordan, t.alramadneh@ammanu.edu.jo, ORCID: 0000-0001-6543-552
[7] University Institute of Diet and Nutritional Sciences, Allied Health Sciences, The University of Lahore, Pakistan, madihajaffar06@gmail.com
[8] Department of Nutritional Sciences, Faculty of Science and Technology, Government College Women University Faisalabad, Pakistan, Asmabukhari758@gmail.com
*Corresponding Author: Ahsan Ali Khan, ahsan0314.aaa@gmail.com
**Corresponding Author: Ammara Afzal, ammara.mugal@gmail.com

ABSTRACT: Diabetes mellitus Type 2 (T2DM) is a metabolic disorder that is now emerging as a risk factor for cognitive dysfunction, neuroinflammation, and gut microbiota changes. The gut microbiota – brain axis is a bidirectional communication system with links between intestinal microorganisms and the central nervous system via immune, endocrine, neural, and metabolic systems. Dysbiosis could be responsible for intestinal barrier dysfunction, systemic inflammation, changed production of microbial metabolites, and compromised integrity of the blood–brain barrier. The mechanisms may have implications for neuroinflammatory pathways and cognition in T2DM patients. The aim of this proposed study is to assess the correlation between gut microenvironment and inflammatory markers, metabolic control, and cognition in T2DM adults with healthy controls. A cross-sectional dataset is provided to illustrate the presentation of microbial diversity, inflammatory markers, cognitive scores, and correlation analyses. The proposed outcomes show the potential for decreased microbial diversity and the production of different short-chain fatty acids to coincide with increased inflammatory markers and decreased cognitive performance. However, these are simulated results and are not able to prove causation or provide observed clinical evidence. Knowing more about the gut microbiota–brain axis could lead to the discovery of novel biomarkers and targets for diabetes-related cognitive dysfunction. This work highlights the potential role of type 2

diabetes mellitus in the gut–brain axis and cognition, as well as gut microbiota, short-chain fatty acids, and intestinal permeability.

INTRODUCTION

Insulin resistance, impaired glucose regulation, and gradual deterioration in pancreatic beta-cells are the hallmarks of type 2 diabetes mellitus (T2DM) (Afzal et al., 2025; Ježek et al., 2019). Besides well-recognized effects on the cardiovascular, renal, and peripheral nervous systems, T2DM is now known to be associated with alterations in brain structure and function (Pruzin et al., 2018). T2DM can cause difficulties with attention, processing speed, executive function (prioritizing thoughts and actions), learning, and memory. These changes in cognition may lead to impairment in daily function and quality of life, especially in older people and those who have suffered from chronic metabolic dysfunction (Damanik & Yunir, 2021). While diabetes-induced cognitive dysfunction may be caused by vascular injury, oxidative stress, and insulin dysfunction, recent studies suggest that gut microbiota might play a role in these mechanisms. The human gastrointestinal ecosystem is a complex microbial community of bacteria, archaea, fungi, and viruses that interact to affect digestion, immune function, maintenance of intestinal barrier integrity, and metabolic homeostasis (Ruan et al., 2020).

Diet, medication, age, physical activity, and underlying disease are factors that affect the composition and functional activity of this community. Microbial dysbiosis has been correlated with changes in the diversity of bacteria, variations in gut bacterial abundance, and alterations in the production of biologically active metabolites in T2DM (Manor et al., 2020). These changes can have an impact on glucose metabolism and inflammatory signaling, but they can differ from one population to another and from one person to another. The gut microbiota–brain axis refers to the communication that occurs between the gut and the brain, via the interconnected neural, endocrine, immune, and metabolic pathways (Kasarello et al., 2023). Mechanisms involved in this communication include the vagus nerve, hypothalamic–pituitary–adrenal axis, circulating cytokines, microbial metabolites, and the function of the gut barrier (Haris et al., 2026; Longo et al., 2023).

Microbial fermentation of dietary substrates results in the generation of short-chain fatty acids (SCFAs), especially acetate, propionate, and butyrate. These metabolites may affect the barrier function of the intestinal epithelium, immune reactions, and host metabolism (Mann et al., 2024). Butyrate is particularly known to promote the barrier function of the epithelial lining and could have anti-inflammatory properties. Thus, alterations in microbial nutrient production of SCFA may impact metabolic regulatory pathways and brain health (Hays et al., 2024). Another possible connection between dysbiosis and systemic inflammation is through the dysfunction of the intestine's barrier. There may be greater intestinal permeability that allows passage of bacterial products such as lipopolysaccharide (LPS) into the bloodstream (Thevaranjan et al., 2017). This process may trigger innate immunity pathways and help to cause metabolic endotoxemia. Constant inflammatory signaling can influence the regulation of blood vessels, insulin sensitivity, and blood–brain barrier integrity. In the central nervous system, activation of microglia and astrocytes can lead to changes in the production of inflammatory mediators and may also be able to disrupt neuronal signaling (Ponce-Lopez, 2025). However, peripheral inflammatory markers alone cannot define neuroinflammation in the brain tissue, and the relationships between intestinal modifications and human cognitive function/acquisition are not fully understood.

Insulin resistance could also link metabolic dysfunction to cognitive decline. Insulin signaling has a role in neuronal plasticity, synaptic function, and memory. All these functions can be impaired together by chronic hyperglycemia, oxidative stress, microvascular injury, and altered inflammatory regulation. Some of these pathways may also be altered by gut microbial metabolites, while diet, obesity, physical inactivity, diabetes duration, and glucose-lowering medications can affect the microbes and cognition (Shahzadi et al., 2025; Spinelli et al., 2019) Thus, it is likely that cognitive impairment is linked to gut dysbiosis, and not to a single microbial species or metabolite. Although an increasing number of researchers are interested in microbiota-based interventions, there are missing pieces in key questions concerning what combination of microbes is most stable over time and most closely linked to cognitive dysfunction associated with T2DM (Van Hul & Cani, 2026). Combination studies of microbial diversity and concentrations of SCFAs with inflammatory markers, glycemic control, and standardized cognitive assessments may offer a more holistic view of these relationships (Abildinova et al., 2024).

Thus, the aim of this study is to explore the relationship between gut microbial characteristics and cognitive functioning in T2DM adults, in particular the level of systemic inflammation and microbial metabolite profiles. The main aims are to examine the differences in microbial diversity and cognitive scores between people with T2DM and healthy controls; to analyse

differences in inflammatory biomarker levels and SCFA concentrations; and to examine relationships between glycemic control, inflammatory markers, and cognitive scores. The purpose of this study is to consider these variables together as a set to develop a framework for exploring possible pathways between metabolic disease, intestinal microbial ecology, and brain health, but without assuming causality.

MATERIAL AND METHOD

Study design and population

The proposed cross-sectional observational study would include 90 adults: healthy controls (n=30), persons with T2DM who do not have cognitive impairment (n=30), and persons with T2DM and cognitive impairment (n=30). Patients would be sampled from the outpatient diabetes clinic or community settings. The sample size is only an example; the specific sample size should be calculated using a proper power calculation prior to the study.

Eligibility criteria

The diabetic groups would be suitable for adults 40–70 years of age who are clinically diagnosed with T2DM. Healthy controls would not have a known diagnosis of diabetes. Exclusion would be for having type 1 diabetes, diagnosed neurodegenerative disease, recent antibiotic use, recent gastrointestinal infection, or significant neurological disease that may affect cognitive function independently. Medication use, dietary factors, physical activity, body mass index, and diabetes duration would be collected as potential confounding factors.

Cognitive assessment

The Montreal Cognitive Assessment (MoCA) is a standardized screening tool with different cognitive domains including memory, attention, executive function, language, visuospatial ability, and orientation that would be used to assess cognitive function. An assessment would be given by trained personnel. Language, education, and validated population-specific adjustments would be used appropriately. Prespecified validated criteria would be used to classify cognitive impairment, not a numeric score (Julayanont et al., 2012).

Gut microbiota analysis

Fecal samples would then be collected according to standard procedure and would be stored in the laboratory under validated conditions until analysis. Characterization of the bacterial community composition would be done by 16S rRNA gene sequencing. Observed amplicon sequence variants (ASVs) and Shannon diversity index (SDI) would be used to assess alpha diversity. The distance measure will be used to measure beta diversity along with a permutational multivariate analysis of variance. Microbial abundance differences would be explored via methods that take compositional data and multiple testing into consideration.

Biochemical and inflammatory measurements

Samples of blood would be drawn after the fast for glucose, glycated hemoglobin (HbA1c), high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). These short-chain fatty acids (SCFAs) in plasma, such as acetate, propionate, and butyrate, would be measured by a validated chromatographic technique, preferably gas chromatography–mass spectrometry (Lee et al., 2025). Exploratory considerations would include an intestinal permeability marker. Peripheral inflammatory biomarkers would not measure brain inflammation, but rather would be considered as indirect markers of inflammatory status.

Statistical analysis

Continuous variables would be reported as the mean ± standard deviation or median and interquartile range, depending on the distribution. ANOVAs or Kruskal–Wallis tests would be used for group differences, and appropriate post hoc comparisons would be performed. Chi-square or Fisher's exact tests would be used to analyze categorical variables. Spearman or Pearson correlation (depending on the type of data) would be used to explore associations between microbial diversity, SCFAs, inflammatory markers, HbA1c, and MoCA scores. Where applicable, age, sex, education, BMI, diabetes duration, diet, and medication use would be taken into account in a multivariable regression. For microbiome analyses, multiple comparisons were adjusted, and for all other analyses, $p < 0.05$ would be considered statistically significant.

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RESULTS

Characteristics and metabolic profile of the participants.

The data were a set of 90 subjects divided equally among three groups. The results generated in the simulation showed a progressive worsening from healthy controls to T2DM participants with cognitive impairments (Table 1).

Table 1. Demographic and metabolic characteristics

Parameter	Healthy controls (n = 30)	T2DM without cognitive impairment (n = 30)	T2DM with cognitive impairment (n = 30)
Age (years)	52.4 ± 6.8	55.1 ± 7.2	59.2 ± 6.5
BMI (kg/m²)	24.6 ± 3.1	28.3 ± 3.7	29.1 ± 4.0
HbA1c (%)	5.3 ± 0.3	7.4 ± 0.8	8.2 ± 1.0
Fasting glucose (mg/dL)	92 ± 9	145 ± 24	162 ± 31
Diabetes duration (years)	Not applicable	6.1 ± 3.2	8.4 ± 4.1

Gut microbial diversity

The simulated values showed possible patterns of reduced alpha diversity in the group of participants with T2DM and cognitive impairment (Table 2).

Table 2. Gut microbial diversity indices

Diversity parameter	Healthy controls	T2DM without cognitive impairment	T2DM with cognitive impairment
Shannon diversity index	3.80 ± 0.35	3.35 ± 0.40	2.95 ± 0.45
Observed ASVs	185 ± 28	158 ± 31	132 ± 29
Simpson diversity index	0.94 ± 0.02	0.91 ± 0.03	0.88 ± 0.04

ASVs = amplicon sequence variants. Values are means ± SD. Simpson index was presented on a 0–1 scale, where higher values indicate greater diversity under the convention used.

The concentration of short-chain fatty acids

The cognitive-impairment group had lower levels of the three major SCFAs compared to other groups in the dataset. Concentrations would vary according to specimen type and analytical method, diet, and sample handling (Table 3).

Table 3. Plasma short-chain fatty acid concentrations

Metabolite (µmol/L)	Healthy controls	T2DM without cognitive impairment	T2DM with cognitive impairment
Acetate	120 ± 22	101 ± 20	79 ± 18
Propionate	25 ± 6	20 ± 5	15 ± 4
Butyrate	12 ± 3	9.5 ± 2.5	6.8 ± 2.1

Values are means ± SD.

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Inflammatory biomarkers

The simulated values represent the potential for increased systemic inflammatory activity for cognition-impaired participants with T2DM (Table 4).

Table 4. Circulating inflammatory biomarkers

Biomarker	Healthy controls	T2DM without cognitive impairment	T2DM with cognitive impairment
hs-CRP (mg/L)	1.4 ± 0.7	3.2 ± 1.4	4.6 ± 1.8
IL-6 (pg/mL)	2.1 ± 0.8	3.8 ± 1.3	5.2 ± 1.7
TNF-α (pg/mL)	4.5 ± 1.2	6.8 ± 1.8	8.4 ± 2.1

Values are means ± SD.

Cognitive performance

The cognitive scores reflect the cognitive differences between the groups, with a lower mean MoCA score in the group with T2DM and cognitive impairment (Table 5).

Table 5. Cognitive assessment results

Cognitive measure	Healthy controls	T2DM without cognitive impairment	T2DM with cognitive impairment
MoCA total score (30 points)	27.2 ± 1.6	25.4 ± 2.0	21.8 ± 2.5
Memory domain (illustrative normalized score, 0–100)	82 ± 8	74 ± 10	61 ± 12
Attention domain (illustrative normalized score, 0–100)	85 ± 7	77 ± 9	65 ± 11
Executive function (illustrative normalized score, 0–100)	81 ± 9	72 ± 10	60 ± 12

Values are means ± SD.

The association between microbiota, inflammation and cognition

Table 6. Correlations with cognitive performance

Variable	MoCA score, Spearman's ϱ	Illustrative direction
Shannon diversity index	+0.42	Positive
Plasma butyrate	+0.46	Positive
Plasma acetate	+0.35	Positive
HbA1c	−0.48	Negative
hs-CRP	−0.44	Negative
IL-6	−0.39	Negative
TNF-α	−0.43	Negative

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DISCUSSION

The study of the gut microbiota–brain axis may be a useful model to explore in investigating the link between metabolic dysfunction, systemic inflammation, and cognitive decline in individuals with T2DM. This study's results model the potential for reduced microbial diversity, decreased levels of SCFA, elevated inflammatory biomarkers, and impaired cognitive functioning to all happen together. The numerical values are simulated, however, and as such can't be interpreted as evidence of a real relationship. The following discussion describes the biological basis for proposed relationships and discusses their relationship to existing research.

The simulated decrease in Shannon diversity and observed decrease in microbial diversity among the three groups indicate a hypothesis that the microbial community structure could be different based on metabolic status and cognitive function (Tardy et al., 2014). There are some previous clinical studies that have found that gut microbial diversity/composition is different between people with T2DM with cognitive impairment and without cognitive impairment (Kolobaric, 2023). A study involving 154 T2DM older adults examined the diversity of the participants with and without cognitive impairment; this is relevant to this research question. However, decreased diversity is not exclusive to metabolic/neurological conditions. The results may differ based on geographic location, dietary habits, drug exposure, order of administration, and analysis. Furthermore, the alpha diversity cannot be used to determine the nature of the microbial community; i.e., it cannot determine if the microbial community is beneficial or detrimental (Hugerth & Andersson, 2017). Therefore, beta diversity, the individual microbial taxa, functional pathways, and microbial metabolites should be assessed in addition to diversity indices in future analyses.

The speculative decrease in acetate, propionate, and butyrate suggests the possible role of microbial metabolites in gut–brain communication. These compounds are mainly produced by microbial fermentation of dietary substrates and may be responsible for regulating epithelial barrier integrity and host metabolism, as well as regulating immune responses (Fu et al., 2023). Butyrate is especially relevant due to its ability to promote intestinal epithelial cell proliferation and control inflammation. These metabolites could be investigated in diabetes-associated cognitive impairment based on the clinical evidence (Yu et al., 2026). In a cross-sectional study, 30 patients with diabetic cognitive impairment were compared to 30 patients with T2DM without mild cognitive impairment, where lower levels of several SCFAs were shown to be associated with higher levels of TNF-α and IL-8 in patients with cognitive impairment. These findings suggest that SCFAs may serve as a link between the function of the intestinal flora and systemic inflammatory status, and warrant further investigation. But the levels of circulating SCFAs do not always correlate with production, as absorption, tissue utilization, hepatic metabolism, and clearance can also have an effect on SCFA levels. These measurements should therefore be interpreted in view of the amount of dietary fiber consumed and the use of medication (Zhao et al., 2025).

The elevations in hs-CRP, IL-6, and TNF-α suggest a possible link between diabetes-associated inflammation and cognitive performance. Persistent inflammatory signaling can be caused by chronic hyperglycemia, insulin resistance, adipose tissue inflammation, and vascular dysfunction. Microbial products that enter the circulation after disruption of the intestinal barrier could be a further source of innate immune stimulation. The central nervous system can be indirectly involved through circulating inflammatory mediators, endothelial dysfunction, and blood–brain barrier (BBB) integrity. In the normal healthy brain, microglia maintain neural homeostasis, but under pathological conditions, they can take on an inflammatory state. Recent reviews outline possible interactions between microbial metabolites and microglial activation and inflammatory pathways, including those involving TLR4/NF-κB signaling and the inflammasome involving NLRP3. Much of the mechanistic evidence is based on experimental models, and not on definitive human causal studies (Ma et al., 2026). One should note, however, that high peripheral cytokines do not necessarily indicate neuroinflammation. In future clinical trials, a combination of systemic biomarkers and validated neurological assessments and, if appropriate, further measures of central nervous system function should be used.

Notice the possible link between poor glycemic control and cognitive performance in the lower scores on the MoCA in the cognitive-impairment group and the higher HbA1c. These are all possible consequences of chronic hyperglycemia, such as oxidative stress, formation of advanced glycation end products, microvascular damage, and impairment of insulin signaling in neurons. These processes can impact attention, memory, and executive functioning. Other factors, such as age, education, diabetes duration, hypertension, sleep quality, depression, and vascular disease, however, can affect cognition independently of gut microbiota. These factors need to be included in the statistical models to assess whether microbial and inflammatory factors offer further explanatory information beyond the traditional metabolic risk factors. The gut microbiota could eventually help

identify biomarkers or the development of additional strategies for diabetes-associated cognitive dysfunction. Some areas of potential research involve dietary fiber interventions, specific probiotics, prebiotics, and other interventions on the microbiome.

CONCLUSION

The gut–brain axis is a viable biological pathway to explore the relationship between T2DM, inflammation, and cognitive dysfunction. Pathways between metabolic disease and brain function are possible to connect through microbial dysbiosis, changes in SCFA profile, intestinal barrier disruption, and inflammatory signaling. But the proposed connections between the two have to be substantiated by actual data at the individual level, the use of the appropriate statistical methods, and the correction for possible confounding factors. To elucidate the role of microbiota-related changes in cognitive decline, longitudinal and interventional studies will be required, with the former distinguishing between direct causal and primary association pathways. A major drawback of this draft is the fact that the results are not measured, but rather simulated numerically. Therefore, none of the tables can be claimed to have statistical significance, clinical association, or causal relationship. The proposed cross-sectional design would also restrict inferences of temporality and causation. Some potential challenges for a future study may involve variations in diet, medication use, and control for diabetes, residual confounding, geographic differences in microbiome composition, and the inability to assess central neuroinflammation from peripheral inflammatory markers alone.

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