

Association of Gutmicrobiota-Derived Lipopolysaccharide (LPS) and Inflammatory Biomarkers with Glycemic Control and Type 2 Diabetes Patients

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Abstract:

Background: Type 2 diabetes mellitus (T2DM) is characterized not only by chronic hyperglycemia but also by insulin resistance, dyslipidemia, oxidative stress, and low-grade systemic inflammation. Altered gut microbial composition and impaired intestinal-barrier function may allow translocation of gut-derived lipopolysaccharide (LPS), a component of the outer membrane of Gram-negative bacteria, into systemic circulation. Recent literature supports an association between metabolic endotoxemia, inflammation, insulin resistance, and T2DM; however, direct clinical correlations between circulating LPS, C-reactive protein (CRP), glycemic markers, and lipid parameters remain insufficiently studied, particularly in Indian patients with T2DM. In our result we have observed that, serum LPS has negative correlation with glycemic marker and lipid profile, which statistically not significant. And CRP has positive correlation with glycemic marker and lipid profile (except HDL-Cholesterol), which was found statistically significant.

Keywords: Type two diabetes mellitus, lipopolysaccharides, Glycemic marker and Lipid profile.

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INTRODUCTION

Type-2 diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia resulting from insulin resistance, progressive pancreatic beta-cell dysfunction, or both^[1]. It is frequently accompanied by obesity, dyslipidemia, hypertension, endothelial dysfunction, and increased cardiovascular risk. Glycemic status in patients with T2DM is commonly assessed by fasting blood sugar, postprandial blood sugar, and HbA1c^[2].

Lipopolysaccharide (LPS) is a glycolipid that resides within the outer membrane of Gram-negative bacteria. Upon introduction into the body, LPS interacts with Toll-like receptors (TLR4) in vivo even at exceedingly minute concentrations, thereby giving rise to pro-inflammatory cytokines, and subsequently inciting a pronounced systemic inflammatory reaction^[3]. This state is termed a cytokine storm condition, and it can elicit shock-related symptoms such as fever and diarrhea and can lead to mortality^[4]. Since its discovery, LPS has been termed an endotoxin and has served as an inflammatory-inducing agent in the activation of immune cells in vitro and inflammation models in animals (in vivo) through injection. Moreover, bacterial translocation has been detected in persistent inflammatory lesions in the intestinal

tract and periodontal tissues, and the continual invasion of bacteria and LPS in organisms provokes systemic inflammation^[5]. In this regard, LPS is considered to be a contributing factor to chronic inflammatory conditions, that include lifestyle-related diseases^[5]. Specifically, numerous investigations have reported that LPS is a causative agent for type 2 diabetes mellitus, which is one of the major lifestyle diseases associated with modern living habits and behaviors.

CRP is an acute-phase protein synthesized by the liver in response to pro-inflammatory cytokines such as IL-6 and TNF- α , which are over expressed by adipose tissue in obesity. Healthy individuals exhibit low levels of CRP while those with T2DM have persistently high concentrations (often in the high-Sensitivity CRP range of 3–10 mg/L, and higher still with acute illness). This reflects subclinical inflammation due to insulin resistance and visceral obesity^[6] (Julijana Stanimirovic).

CRP production may be initiated by several metabolic and inflammatory factors associated with the progression of T2DM such as increased blood glucose, adipokines, and free fatty acids. In addition, increased CRP levels may be used as a reliable predictor of vascular complications and the

progression of cardiovascular disease in diabetic patients^[7,8].

Lipid profile assessment plays a crucial role in the management of diabetes mellitus due to the close relationship between dyslipidemia and both the pathophysiology of the disease and its cardiovascular complications^[9]. In type 2 diabetes, insulin resistance and relative insulin deficiency lead to increased lipolysis in adipose tissue, elevated free fatty acid flux to the liver, and enhanced hepatic triglyceride and very-low-density lipoprotein (VLDL) production, along with reduced lipoprotein lipase activity and impaired triglyceride clearance^[10]. This results in the classic “diabetic dyslipidemia” profile of elevated triglycerides, reduced high-density lipoprotein (HDL) cholesterol, and a preponderance of small, dense low-density lipoprotein (LDL) particles – which are more atherogenic than larger LDL particles – contributing to accelerated endothelial dysfunction and atherosclerosis^[10]. Lipid abnormalities in diabetes are further associated with glycemic control (e.g., HbA1c levels) and play a role in lipotoxicity-driven β -cell dysfunction and systemic insulin resistance, thus participating in a vicious cycle that exacerbates metabolic derangements. As such, lipid profile assessment in diabetes is essential for risk stratification, monitoring, and guiding dyslipidemia treatment to mitigate the considerable atherosclerotic cardiovascular disease burden in this population^[1].

Type 2 diabetes mellitus (T2DM) is characterized by chronic hyperglycemia, dyslipidemia, inflammation, and heightened risk of cardiovascular complications^[11]. Routine biochemical tracking of T2DM primarily focuses on glucose and lipid biomarkers while the role of metabolically derived inflammatory mediators originating from the gastrointestinal tract is poorly recognized in the clinical research setting.

The current study investigated the potential association of serum lipopolysaccharide (LPS), an indicator of metabolic endotoxemia with the condition of glycemic control, and C-reactive protein (CRP), a systemic inflammatory marker, with dyslipidemia in patients with T2DM. Identification of such associations could contribute to the enhanced understanding of the gut-inflammation-metabolism axis and facilitate the recognition of individuals at increased cardiometabolic risk.

Aim and Objectives:

Aim:

To assess the association of serum lipopolysaccharide and CRP with glycemic markers and lipid profile among patients with T2DM and non-diabetic.

Objectives:

To evaluate the level of plasma FBG, PPBG, serum LPS, serum CRP and lipid profile among diabetic and non-diabetic subjects.

To find correlation and association of serum LPS and serum CRP level in type two diabetic subjects.

Methodology:

This hospital-based cross-sectional analytical study was conducted on 150 adult patients diagnosed with T2DM and 150 non-diabetic subjects attending the Department of Biochemistry, Kanti Devi Medical College Hospital and Research Center Mathura U.P., India. Both the gender was included in this study, who were fulfill the inclusion and exclusion criteria.

Patient's aged 30 years or older, Patients diagnosed with T2DM, and patients able to provide fasting and postprandial blood samples were included in this study.

Patients with type 1 diabetes mellitus, gestational diabetes mellitus, or secondary diabetes, Acute febrile illness, sepsis, acute infection, or recent hospitalization within the previous 4 weeks, Autoimmune diseases, inflammatory bowel disease, chronic liver disease, malignancy, or chronic infectious disease, Severe renal impairment or end-stage renal disease, recent major surgery, trauma, myocardial infarction, or stroke, current use of systemic corticosteroids, immunosuppressive drugs, or long-term antibiotics within the previous 4 weeks, probiotic, prebiotic, and synbiotic, or faecal microbiota-based treatment within the previous 4 weeks were excluded from the study.

After the inclusion and exclusion criteria following overnight fasting, 5 ml of venous blood samples were collected for estimation of FBS, HbA1c, lipid profile, CRP, and serum LPS. PPBS was measured after 2 hours of meal.

Serum lipopolysaccharide (LPS) was estimated using the Human LPS ELISA (sandwich ELISA) Kit (ELK Biotechnology Co., Ltd., Wuhan, China) according to the manufacturer's instructions^[12].

CRP levels were estimated using the GENLISA® Human C-Reactive Protein (CRP) ELISA Kit Manufactured by Krishgen Biosystems, Mumbai, India^[13].

Plasma glucose levels were determined using an enzymatic UV method based on the hexokinase principle. Roche Diagnostics – Glucose HK Gen.3 Reagent Kit (for use on Cobas analyzers) was used and the assay was performed strictly according to the manufacturer's instructions^[14].

Serum total cholesterol was estimated using the Cholesterol Gen.2 reagent on the Cobas Pure 303 clinical biochemistry analyzer (Roche Diagnostics, Mannheim, Germany). The assay is based on an enzymatic colorimetric method employing cholesterol esterase, cholesterol oxidase, and peroxidase (CHOD-PAP method) and was

performed according to the manufacturer's instructions^[15].

Serum triglyceride concentrations were estimated using the Triglycerides GPO-PAP reagent on the Cobas C clinical biochemistry analyzer (Roche Diagnostics, Mannheim, Germany). The assay is based on an enzymatic colorimetric method employing glycerol phosphate oxidase–peroxidase (GPO-PAP^[16]).

Serum HDL-cholesterol was estimated using the HDL-Cholesterol Plus 4th Generation (HDL-C Plus Gen.4) reagent on the Cobas C clinical biochemistry analyzer (Roche Diagnostics, Mannheim, Germany). The assay is based on a homogeneous enzymatic colorimetric method that enables direct measurement of HDL cholesterol without prior precipitation or centrifugation^[17].

Serum VLDL-cholesterol and LDL-cholesterol were not measured directly but calculated by Friedewald's equation using the serum TG, HDL and cholesterol concentration.

VLDL and LDL cholesterol was calculated using the following formula:

VLDL-C (mg/dL) = Triglycerides (mg/dL)/ 5

LDL-C (mg/dL) = Total Cholesterol – HDL-C – VLDL-cholesterol

After the estimation of all the biomarker, data was entered in MS Excel sheet. And SPSS software 29.0, was used to calculate mean. Standard deviation, unpaired student t-test and Pearson's correlation value. A p value of <0.05 was considered as statistically significant.

Result And Observation:

The present study was conducted on 300 subjects (150 diabetic and 150 non-diabetic) including both genders. In our result we have observed that, diabetic patients age (51.36 ± 13.31 years) was higher than non-diabetic subjects (38.25 ± 8.72 year).

Glycemic markers were significantly elevated among patients with T2DM. The mean value of fasting blood sugar was 151.85 ± 20.69 mg/dl in the T2DM group as compared with non-diabetic subject (89.46 ± 4.23 mg/dL). Similarly, the mean postprandial blood sugar was 245.20 ± 50.09 mg/dl in T2DM cases and 11.27 ± 9.64 mg/dL in non-diabetic subject, which was found statistically significant (p-value <0.001). The mean value of HbA1c was higher (8.99 ± 2.67 gm %) in T2DM subjects and within normal range (5.17 ± 0.26 gm %) in non-diabetic subjects, which was also found statistically significant (p-value <0.001) (Table 1).

The average of lipopolysaccharides (LPS) was higher (0.63 ± 0.1 ng/ml) in T2DM as compared with non-diabetic (0.31 ± 0.06 ng/ml), which was also found statistically significant (p-value <0.001). Serum CRP level was increased (11.29 ± 2.67 mg/dl) in T2DM subjects and 5.86 ± 2.68 mg/dl in non-diabetic subjects, which

was also found statistically significant (p-value <0.001) (Table 2).

In our result, patients diagnosed with T2DM also suffering from dyslipidemia and all the lipid parameters were found statistically significant (p-value <0.001). The mean value of total cholesterol was 208.03 ± 50.09 mg/dl in T2DM and 181.37 ± 25.06 mg/dl in non-diabetic subjects. Serum triglycerides level was 232.62 ± 99.75 mg/dl in T2DM and 118.37 ± 25.06 mg/dl in non-diabetic subjects. Serum HDL-cholesterol level was 41.08 ± 11.5 mg/dl in T2DM and 53.86 ± 9.98 mg/dl in non-diabetic subjects. Serum LDL-cholesterol level was 127.71 ± 34.5 mg/dl in T2DM and 99.92 ± 25 mg/dl in non-diabetic subjects. Serum VLDL-cholesterol level was 46.96 ± 20.23 mg/dl in T2DM and 24.48 ± 8.16 mg/dl in non-diabetic subjects (Table 3).

Table.1: Comparison of glycemic markers in T2DM cases and control group.

Glycemic Biomarker	Diabetic Group (T2DM)		Non-Diabetic Group		t-test	p-value
	mean	± SD	mean	± SD		
FBS (mg/dl)	151.85	20.69	89.46	4.23	33.67	<0.001
PPBS (mg/dl)	245.2	50.09	11.27	9.64	17.53	<0.001
HBA1C (gm %)	8.99	2.67	5.17	0.26	36.05	<0.001

Table.2: Comparison of biochemical markers (LPS and CRP) in T2DM cases and control group.

Biochemical Marker	Diabetic Group (T2DM)		Non-Diabetic Group		t-test	p-value
	mean	± SD	mean	± SD		
LPS (ng/ml)	0.63	0.1	0.31	0.06	32.04	<0.001
CRP (mg/dl)	11.29	2.67	5.86	2.68	17.39	<0.001

Table. 3: Comparison of lipid markers in T2DM cases and control group.

Lipid Profile	Diabetic Group (T2DM)		Non-Diabetic Group		t-test	p-value
	mean	± SD	mean	± SD		
T. Cholesterol (mg/dl)	208.03	50.09	181.37	25.06	6.64	<0.001
Triglycer	232.62	99.75	118.37	25.06	13	<0.001

ides(mg/dl)	62	5	.37	06	.26	001
HDL (mg/dl)	41.08	11.5	53.86	9.98	-10.24	<0.001
LDL (mg/dl)	127.71	34.5	99.92	25	7.96	<0.001
VLDL (mg/dl)	46.96	20.23	24.48	8.16	12.45	<0.001

In our result we have observed that, serum LPS has negative correlation with glycemic marker and lipid profile, which statistically not significant (table-4). And CRP has positive correlation with glycemic marker and lipid profile (except HDL-Cholesterol), which was found statistically significant (table-5).

Table-4: Correlation of LPS with biochemical markers in T2DM cases and control group.

Correlation in between		r-value	r-square value	p-value
LPS	CRP (mg/dl)	-0.141	0.02	0.043
	FBS (mg/dl)	-0.02	0.0004	0.41
	PPBS (mg/dl)	0.0075	0.157	0.46
	HbA1c (gm%)	-0.118	0.014	0.075
	T. cholesterol (mg/dl)	-0.121	0.0146	0.07
	Triglycerides (mg/dl)	-0.016	0.0002	0.42
	HDL (mg/dl)	-0.033	0.001	0.34
	LDL (mg/dl)	-0.102	0.01	0.12
	VLDL (mg/dl)	-0.034	0.0012	0.33

Chart-3: Comparison of lipid markers in T2DM cases and control group.

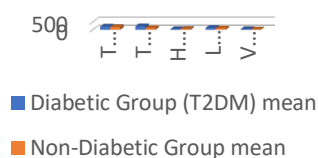


Table-5: Correlation of CRP with biochemical markers in T2DM cases and control group.

Correlation in between		r-value		r-square value	p-value
CRP	FBS (mg/dl)	0.359945	0.12		<0.01
	PPBS (mg/dl)	0.396584	0.15		<0.01
	HbA1c (gm%)	0.917817	0.84		<0.01
	T. cholesterol (mg/dl)	0.052968	0.02		0.27
	Triglycerides (mg/dl)	0.055058	0.03		0.29
	HDL (mg/dl)	-0.18281	0.033		0.34
	LDL (mg/dl)	0.030536	0.009		0.35
	VLDL (mg/dl)	0.046564	0.02		0.28

Discussion:

The present study demonstrated significant differences in glycemic, inflammatory, LPS, and lipid parameters between patients with T2DM and controls. Patients with T2DM had substantially higher FBS, PPBS, and HbA1c values, confirming poor glycemic control in the case group. The mean HbA1c of was above the commonly recommended treatment target of less than 7% for many nonpregnant adults, although therapeutic targets should be individualized according to age, comorbidities, treatment-related hypoglycemia, and life expectancy^[4].

The higher LPS concentration observed in the T2DM group suggests increased exposure to, or translocation of, bacterial endotoxin. LPS is the major component of the outer membrane of Gram-negative bacteria and can activate innate immune responses, primarily through the Toll-like receptor 4 pathway. This may promote cytokine production, systemic inflammation, insulin resistance, and metabolic dysfunction. Nevertheless, the present cross-sectional findings demonstrate an association only and cannot establish that increased LPS caused T2DM or poor glycemic control^[3].

The increased CRP concentration in the T2DM group is consistent with the concept that T2DM is accompanied by chronic low-grade inflammation. Recent clinical evidence has also reported higher inflammatory markers, including CRP, among people with T2DM and has linked inflammatory activity with poor glycemic control and lipid abnormalities. A recent study further reported that elevated CRP was associated with poor glycemic control, dyslipidemia, and diabetic complications in patients with T2DM^[11,18].

In the present study, CRP showed significant positive correlations with FBS and PPBS. This pattern indicates that greater short-term hyperglycemia was associated with increased systemic inflammation. The strongest association was observed between CRP and HbA1c, suggesting a relationship between chronic glycemic exposure and inflammation. A possible explanation is that persistent hyperglycemia promotes oxidative stress, advanced glycation end-product formation, endothelial dysfunction, and activation of inflammatory pathways. However, correlation does not demonstrate causality. In addition, the unusually high correlation coefficient between CRP and HbA1c should be validated by repeating the analysis and examining scatterplots, outliers, confidence intervals, and the underlying sample size.

Although LPS concentrations were significantly higher in T2DM cases, LPS did not show significant correlations with most glycemic or lipid variables. This may indicate that circulating LPS is influenced by several factors other than glycemic status, including diet, intestinal permeability, gut microbiota composition, obesity, medication use, renal function, recent infection, and preanalytical handling. Differences in LPS measurement methods are also important because endotoxin assays may be affected by contamination, sample matrix effects, and limited analytical sensitivity. Therefore, the absence of significant correlations should not be interpreted as evidence that LPS has no biological relevance in T2DM^[3,5,10,19].

The weak inverse correlation between LPS and CRP was statistically significant but biologically small. It explains only approximately 2% of the variation in CRP. This finding is contrary to the expected direction if both markers reflect the same inflammatory pathway. The LPS–CRP relationship should therefore be reassessed separately among T2DM cases and controls, with adjustment for age, sex, body mass index, diabetes duration, medications, renal function, and acute infection.

The lipid findings are characteristic of an atherogenic dyslipidemia pattern associated with T2DM: increased triglycerides, LDL cholesterol, VLDL cholesterol, and total cholesterol together with reduced HDL cholesterol. This pattern may increase cardiometabolic risk^[20].

Current diabetes guidance emphasizes assessment and management of lipid abnormalities using lifestyle measures and lipid-lowering therapy according to overall cardiovascular risk, rather than relying on total cholesterol alone^[1].

The significant age difference between the T2DM and control groups is an important limitation because age may independently affect glucose metabolism, inflammation, lipid concentrations, and LPS levels. Consequently, unadjusted comparisons may partly reflect age imbalance.

Future analyses should use age and sex-matched controls or multivariable regression models. The study should also report sample size, sex distribution, diabetes duration, medication use, body mass index, renal function, smoking status, and the method used for measuring LPS and CRP.

Conclusion:

Patients with T2DM showed significantly higher sugar level, LPS, CRP, total cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol, together with significantly lower HDL cholesterol, than controls. CRP was positively associated with FBS, PPBS, and HbA1c, particularly showing a strong association with HbA1c. Although LPS concentrations were significantly elevated in T2DM, LPS was not significantly correlated with most glycemic or lipid parameters.

These findings support the presence of metabolic and inflammatory abnormalities in T2DM and suggest that CRP may reflect poor glycemic control in this study population. However, the findings should be interpreted cautiously because of the cross-sectional design, age imbalance, unclear correlation population, possible inconsistencies in PPBS and CRP units, and discrepancies in the reported values. Confirmation using the original dataset and adjusted statistical analysis is recommended before final publication.

References:

1. Shrestha S. & Shrestha A. Dyslipidemia in Patients with Type 2 Diabetes Mellitus Attending Bir Hospital, NAMS, Nepal. *Journal of Medical Innovations*, 2025, 4(09):587-603, <https://doi.org/10.71017/djmi.4.9.d-0492>
2. American Diabetes Association. ADA 2026 Guidelines for Diabetes Management. 2026 Jul.
3. Qin X, Zou H. The role of lipopolysaccharides in diabetic retinopathy. *BMC Ophthalmology*. BioMed Central Ltd; 2022. doi:10.1186/s12886-022-02296-z PubMed PMID: 35193549.
4. Yamamoto K, Yamashita M, Oda M, Tjendana Tjhin V, Inagawa H, Soma GI. Oral Administration of Lipopolysaccharide Enhances Insulin Signaling-Related Factors in the KK/Ay Mouse Model of Type 2 Diabetes Mellitus. *Int J Mol Sci*. 2023;24(5). doi:10.3390/ijms24054619 PubMed PMID: 36902049.
5. Thiagarajan S, Bhaskar E, Gayathri V, Mohanalakshmi P, Silambanan S. Lipopolysaccharide as a biomarker of gut dysbiosis in diabetic Wistar rats – A pre-clinical experimental study. *Indian J Physiol Pharmacol*. 2025;69(4):374–9. doi:10.25259/IJPP_387_2024

6. Stanimirovic J, Radovanovic J, Banjac K, Obradovic M, Essack M, Zafirovic S, et al. Role of C-Reactive Protein in Diabetic Inflammation. *Mediators of Inflammation*. Hindawi Limited; 2022. doi:10.1155/2022/3706508 PubMed PMID: 35620114.
7. Esser N, Paquot N, Scheen AJ. Inflammatory markers and cardiometabolic diseases. *Acta Clinica Belgica: International Journal of Clinical and Laboratory Medicine*. 2015;70(3):193–9. doi:10.1179/2295333715Y.0000000004 PubMed PMID: 26103538.
8. Mugabo Y, Li L, Renier G. The Connection Between C-Reactive Protein (CRP) and Diabetic Vascuopathy. *Focus on Preclinical Findings. Curr Diabetes Rev*. 2010;6(1):27–34. doi:10.2174/157339910790442628 PubMed PMID: 20034371.
9. Wu J, Wang H, Fu M, Zhang M, Zhao G, Yu B. Lipid Metabolic Disorders: a Pivotal Driver in Type 2 Diabetes Mellitus Pathogenesis. *Current Obesity Reports*. Springer; 2026. doi:10.1007/s13679-026-00732-5
10. Abdul Wahab R, Wan Mahmood WA. Dyslipidemia in Diabetes: Navigating a Complex Landscape for Improved Cardiovascular Outcomes. *Clinical Medicine Insights: Endocrinology and Diabetes*. SAGE Publications Ltd; 2026. doi:10.1177/11795514261422310
11. Gogia T, Kaushik N, Jain G. A Cross-Sectional Analysis of Correlation Between Lipid Profile and HbA1c Among Non-Diabetics, Diabetics and Prediabetics. *National Journal of Medical Research*. 2025;15(04):244–9. doi:10.55489/njmr.150420251163
12. ELK Biotechnology. Human LPS(Lipopolysaccharide) ELISA Kit [Internet]. 2025. Available from: <https://www.elkbiotech.com>
13. Human High Sensitivity C Reactive Protein (CRP) GENLISATM ELISA [Internet]. Available from: www.krishgenbio.com
14. Cobas. Estimation of plasma glucose [Internet]. 2025 [cited 2026 Sep 5]. Available from: <https://www.cobas.com>
15. Roche Diagnostics. Quantitative determination of the serum total cholesterol concentration in human serum and plasma on Cobas Integra systems [Internet]. Available from: <https://www.roche.com>
16. Roche Diagnostics. Quantitative determination of the triglycerides concentration in human serum and plasma on Cobas Integra systems [Internet]. Available from: <https://www.roche.com>
17. Roche Diagnostics. Quantitative determination of the HDL-cholesterol [Internet]. Available from: <https://www.roche.com>
18. Nagalakshmi K. Association of C-reactive protein with glycemic control and diabetic complications in type 2 diabetes mellitus. *J Contemp Clin Pract*. 2025;11(6):783-90. doi:10.61336/jccp/25-06-112
19. Molugu SVR, Molugu CR, Molugu HR, Badvel SR. Lipid parameters and their association with liver function test variables in patients with type 2 diabetes mellitus. *Cureus*. 2025. doi:10.7759/cureus.92671
20. Krauss RM. Lipids and lipoproteins in patients with type 2 diabetes. *Diabetes Care*. 2004;27(6):1496-504. doi:10.2337/diacare.27.6.1496