FUDMA Journal of Sciences (FJS)
ISSN online: 2616-1370
ISSN print: 2645 - 2944
Vol. 10 No. 11, June, 2026, pp 195 – 202
DOI: https://doi.org/10.33003/fjs-2026-1011-5143

Acetone Extract of Turmeric (*CURCUMA LONGA*) Attenuates Inflammation, Dyslipidemia, and Hepatorenal Dysfunction in Streptozotocin-Induced Type 2 Diabetic Rats

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Received: 22 Jan. 2026
Accepted: 12 Feb. 2026
Published: 14 May 2026

ABSTRACT

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder associated with hyperglycemia, inflammation, dyslipidemia, and organ dysfunction. This study investigated the anti-inflammatory, hypolipidemic, and hepatorenal protective effects of acetone extract of Turmeric in streptozotocin-induced type 2 diabetic rats. Thirty male Wistar rats were randomly assigned into five groups (n = 6): normal control, diabetic control, turmeric-treated groups (100 and 200 mg/kg body weight), and metformin-treated group (500 mg/kg). Type 2 diabetes was induced using 10% fructose administration for 14 days followed by a single intraperitoneal injection of streptozotocin (50 mg/kg). Biochemical analyses were performed to evaluate inflammatory markers (TNF-α and IL-6), glucose tolerance, lipid profile, and liver and kidney function parameters. Data obtained were analyzed using multivariate analysis of variance (MANOVA) and results were considered significant at $P < 0.05$. Diabetic control rats showed significant elevations in fasting blood glucose, TNF-α, IL-6, total cholesterol, triglycerides, LDL-C, ALT, AST, ALP, urea, and creatinine levels, with a concomitant reduction in HDL-C and glucose clearance ability compared to the normal control group. Treatment with turmeric extract significantly ameliorated these alterations. The 100 mg/kg dose produced greater improvement in lipid profile and reduction in atherogenic indices, while the 200 mg/kg dose demonstrated stronger hepatoprotective effects through greater reductions in liver enzyme activities. These findings suggest that acetone extract of Turmeric possesses anti-inflammatory, hypoglycemic, hypolipidemic, and hepatorenal protective properties, supporting its potential as a complementary therapeutic agent in the management of T2DM and its associated complications.

Keywords: Type 2 Diabetes Mellitus, Turmeric, Inflammation, Dyslipidemia, Hepatorenal Dysfunction, Insulin Resistance

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder characterized by chronic hyperglycemia, insulin resistance, and progressive pancreatic β-cell dysfunction (Galicia-Garcia et al., 2020). The persistent elevation in blood glucose levels activates multiple biochemical pathways, including the polyol pathway, hexosamine pathway, protein kinase C (PKC) activation, advanced glycation end product (AGE) formation, and endoplasmic reticulum (ER) stress. These interconnected pathways contribute significantly to oxidative stress and chronic inflammation, which are key drivers of insulin resistance and the development of diabetic complications (Caturano et al., 2025; González et al., 2023). In particular, the accumulation of reactive oxygen species (ROS) plays a pivotal role in the pathogenesis of diabetes-related complications by disrupting cellular homeostasis and triggering inflammatory responses (Bhatti et al., 2022; Caturano et al., 2023).

A central mechanism linking inflammation and insulin resistance in T2DM is the overproduction of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β)(Tsalamandris et al., 2019). These cytokines activate intracellular signaling pathways such as the Janus kinase-signal transducer and activator of transcription (JAK-STAT) pathway and the nuclear factor-kappa B (NF-κB) pathway, leading to the amplification of inflammatory signaling. This cascade not only sustains systemic inflammation but also impairs insulin signaling (Hu et al., 2021; Sarapultsev et al., 2023). At high concentrations, pro-inflammatory cytokines induce the expression of suppressor of cytokine signaling (SOCS) proteins, which interfere with insulin receptor signaling, exacerbating insulin resistance and glucose intolerance (Liu et al., 2008, Bako et al., 2019). Additionally, TNF-α-mediated activation of IκB kinase-β (IKK-β) leads to serine phosphorylation of insulin receptor substrate-1 (IRS-1), impairing its interaction with insulin receptors and further compromising glucose uptake by insulin-sensitive tissues such as skeletal muscle and adipose tissue(Rui et al., 2001; Yang et al., 2009).

Beyond glucose dysregulation, inflammation-induced metabolic disturbances contribute to the progression of lipid metabolism dysfunction, hepatic impairment, and renal dysfunction. Increased ROS production and inflammatory mediators disrupt lipid homeostasis, leading to dyslipidemia characterized by altered lipid profile parameters such as high levels of low-density lipoprotein (LDL), total cholesterol, and triglycerides and decreased levels of high-density lipoprotein (HDL)(Zhao et al., 2023). These lipid abnormalities contribute to the progression of artherogenic indices such as coronary risk index-I (CRI-I), coronary risk index-II (CRI-II), artherogenic coefficient (AC), and artherogenic index of plasma (AIP), which are major risk factors for cardiovascular diseases in diabetic individuals(Lumu et al., 2023). Moreover,

chronic inflammation and oxidative stress contribute to hepatic lipid accumulation, leading to non-alcoholic fatty liver disease (NAFLD), as evidenced by elevated levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP). Similarly, renal dysfunction in T2DM is linked to inflammatory damage to nephrons, leading to increased levels of urea and creatinine, key indicators of kidney impairment (Kathak et al., 2022; Xuan et al., 2024).

Given the critical role of inflammation in the pathogenesis of T2DM and its complications, there is growing interest in exploring natural anti-inflammatory agents that can modulate these pathways. Turmeric extract, particularly its active compound curcumin, has gained attention for its potent anti-inflammatory, antioxidant, and insulin-sensitizing properties. Turmeric has been widely studied for its pharmacological activities, particularly those attributed to curcumin, its major polyphenolic compound (Sharifi-Rad et al., 2020). Previous studies have demonstrated the anti-inflammatory, antioxidant, hypoglycemic, and lipid-lowering effects of curcumin in experimental diabetes models (El-Saadony et al., 2022). However, turmeric rhizome contains several other bioactive constituents, including turmerones and essential oils, which may contribute synergistically to its biological activities.

Despite numerous studies on isolated curcumin, limited information is available regarding the effects of whole turmeric extract on inflammation-associated metabolic complications in type 2 diabetes. In particular, its potential role in attenuating dyslipidemia and hepatorenal dysfunction associated with insulin resistance remains inadequately characterized. Therefore, this study investigated the effects of acetone extract of Turmeric on inflammatory markers, lipid profile, and liver and kidney function parameters in streptozotocin-induced type 2 diabetic rats. It was hypothesized that turmeric extract would ameliorate inflammation and metabolic complications associated with type 2 diabetes through its combined bioactive constituents.

MATERIALS AND METHODS

Chemicals and Reagents

The following chemicals and reagents were used during the study: streptozotocin (STZ), Tween 80, acetone, corn oil, fructose, citric acid monohydrate ($C_6H_8O_7 \cdot H_2O$), sodium citrate dihydrate ($Na_3C_6H_5O_7 \cdot 2H_2O$), and metformin. All chemicals were of analytical grade and obtained from Bridge Biotech Ltd., Ilorin, Kwara State, Nigeria. Commercial assay kits for lipid profile parameters (HDL-C, total cholesterol, triglycerides), liver function enzymes (AST, ALT, ALP), and kidney function markers (urea and creatinine) manufactured by Randox Laboratories were purchased from Royal Surgical Limited, Kaduna State, Nigeria. Enzyme-linked immunosorbent assay (ELISA) kits for pro-inflammatory cytokines (IL-6 and TNF-α) were obtained from Wuhan Fine Biotech Co., Ltd., China.

Collection and Preparation of Plant Material

Fresh rhizomes of Turmeric were purchased from Central Market, Kaduna State, Nigeria, and authenticated by a taxonomist in the Department of Biological Sciences, Kaduna State University. A voucher specimen (BSH/1242) was deposited in the departmental herbarium for future reference. The rhizomes were washed thoroughly with distilled water, sliced into smaller pieces, and air-dried at ambient temperature (25–28°C) for 10 days. The dried samples were pulverized using a laboratory grinder and sieved through a 60-mesh sieve to obtain a fine powder, which was stored in airtight containers at 4°C until extraction.

Extraction of Turmeric

The powdered turmeric sample was extracted using acetone in a Soxhlet apparatus at a solvent-to-sample ratio of 10:1 (v/w) for 8 h at 60°C. Acetone was selected because of its reported efficiency in extracting curcuminoids and other lipophilic phytoconstituents from turmeric rhizomes. The extract was concentrated under reduced pressure using a rotary evaporator (Stuart RE300) at 35°C and further dried under vacuum to obtain a semi-solid residue. The percentage yield of the extract was determined and the extract stored in amber-colored containers at −20°C until use.

Preliminary phytochemical screening of the extract was conducted using standard qualitative methods to identify the presence of major phytochemical constituents, including phenolics, flavonoids, alkaloids, saponins, tannins, and terpenoids.

Preparation of Extract for Administration

Due to the hydrophobic nature of the turmeric extract, Tween 80 was used as an emulsifying agent to improve solubility and ensure uniform dispersion. The extract was mixed with Tween 80 in a ratio of 1:3 (extract: Tween 80, w/w) and subsequently diluted in corn oil to obtain the required concentrations for administration. Corn oil was used as the vehicle because of its ability to improve the solubility and oral bioavailability of lipophilic compounds. The preparation was freshly reconstituted when required and stored in light-protected containers at 4°C.

Experimental Animals

Thirty male Wistar rats weighing 150–250 g were obtained from the Animal House Facility, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria. Animals were housed under standard laboratory conditions at room temperature (25–28°C) under a 12 h light/dark cycle and allowed free access to standard rat feed and water ad libitum. The study protocol was approved by the Animal Research Ethics Committee of Kaduna State University with ethical approval number: KASU/AREC/2025/017.

Experimental Design and Induction of Type 2 Diabetes

Following one week of acclimatization, rats were randomly assigned into five groups (n = 6 per group):

i. NC: Normal control
ii. DC: Diabetic control
iii. C100: Diabetic rats treated with 100 mg/kg turmeric extract
iv. C200: Diabetic rats treated with 200 mg/kg turmeric extract
v. DMET: Diabetic rats treated with 500 mg/kg metformin

The extract doses (100 and 200 mg/kg body weight) were selected based on previous experimental studies demonstrating the antidiabetic and anti-inflammatory effects of turmeric extract within this dose range without observable toxicity.

Insulin resistance was induced by administering 10% fructose solution ad libitum for 14 days to all groups except the normal control group. Thereafter, type 2 diabetes was induced using a single intraperitoneal injection of streptozotocin (50 mg/kg body weight) freshly prepared in citrate buffer (pH 4.5). One week after STZ administration, non-fasting blood glucose levels were measured using a glucometer. Rats with blood glucose levels greater than 300 mg/dL were considered diabetic and included in the study.

Treatment was administered orally once daily for 21 days using oral gavage. The normal control group received the

vehicle (corn oil) only. Blood glucose levels were monitored weekly throughout the experimental period.

Collection of Blood Samples

At the end of the treatment period, animals were sacrificed by cervical dislocation following overnight fasting. Blood samples were collected by cardiac puncture and centrifuged at 3000 rpm for 10 min to obtain serum, which was stored at −30°C pending biochemical analyses.

Analytical Methods

Serum concentrations of TNF-α and IL-6 were determined using rat-specific ELISA kits according to the manufacturer's instructions. Serum lipid profile parameters including total cholesterol, triglycerides, HDL-C, and LDL-C, as well as liver function enzymes (AST, ALT, ALP) and kidney function markers (urea and creatinine), were analyzed using standard commercial assay kits according to the manufacturers' protocols. Atherogenic indices were calculated using the following equations:

Atherogenic Index of Plasma (AIP) =Log (Triglycerides/HDL-C)

Castelli Risk Index I=Total Cholesterol/ HDL-C

Castelli Risk Index II=LDL-C/HDL-C

Atherogenic Coefficient (AC) =Total Cholesterol−HDL-C/HDL-C

Statistical Analysis

Data were expressed as mean ± standard error of mean (SEM). Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc multiple comparison test using SPSS version 23.0. Differences were considered statistically significant at $P < 0.05$. Each experimental group consisted of six biological replicates ($n = 6$).

RESULTS AND DISCUSSION

This study evaluated the effects of turmeric extract on key metabolic parameters associated with complications in type 2 diabetic rats. The levels of TNF-α and IL-6 (fig. 1) were significantly ($P < 0.05$) elevated in the diabetic control group compared to the normal control, indicating heightened systemic inflammation associated with diabetes. Treatment with turmeric extract and metformin led to a substantial ($P < 0.05$) reduction in these pro-inflammatory cytokines, with both the 100 mg/kg and 200 mg/kg doses of the extract showing a similar effect when compared to the diabetic control group. The first and second oral glucose tolerance tests (OGTT) (fig. 2), conducted on the 10th and 20th days of treatment, respectively, and revealed a marked impairment in glucose clearance in the diabetic control group, confirming insulin resistance. Treatment with turmeric extract significantly ($P < 0.05$) improved glucose tolerance, with a greater reduction in blood glucose observed at the 100 mg/kg dose compared to the 200 mg/kg dose during the second OGTT.

Furthermore, diabetes-induced dyslipidemia was evident in the diabetic control group, as demonstrated by a significant ($P < 0.05$) increase in total cholesterol, triglycerides, and LDL, along with a reduction in HDL (table 1). Turmeric extract administration at 100 mg/kg and 200 mg/kg resulted in a significant ($P < 0.05$) improvement in all lipid parameters. Moreover, the atherogenic indices (table 2), which serve as indicators of cardiovascular risk, were markedly elevated in the diabetic control group. Treatment with turmeric extract at both 100 mg/kg and 200 mg/kg led to a significant ($P < 0.05$) reduction in these indices, with the lower dose of 100 mg/kg showing greater efficacy. Although the reductions observed in turmeric-treated groups were slightly lower than those in the metformin group, the findings suggest that turmeric extract may contribute to cardiovascular risk reduction in diabetes. Liver function markers (ALT, AST, and ALP) (table 3) were significantly ($P < 0.05$) elevated in the diabetic control group, indicating hepatic injury. Turmeric extract administration at 100 mg/kg and 200 mg/kg reduced these enzyme levels, suggesting a hepatoprotective effect. The reduction was more pronounced at the higher dose. Similarly, kidney function markers (urea and creatinine) (table 4) were significantly ($P < 0.05$) elevated in diabetic rats, reflecting renal impairment. Turmeric extract treatment led to a significant reduction in these markers, indicating a protective effect on kidney function.

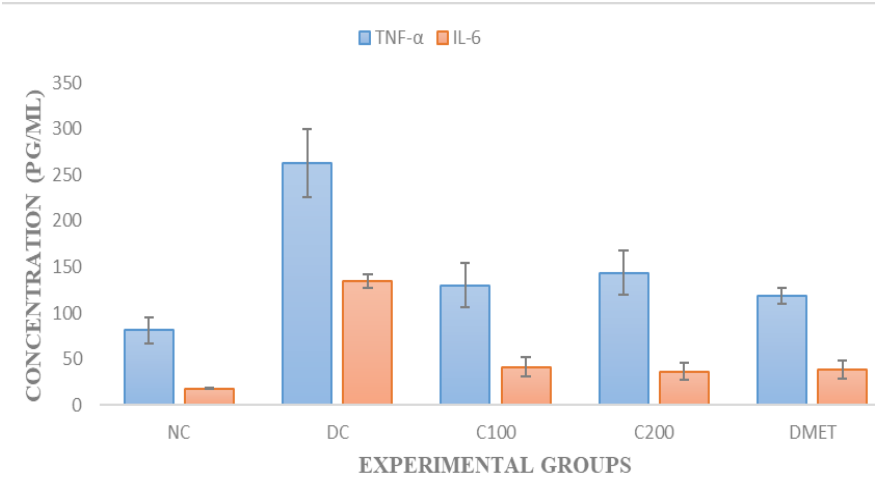

Figure 1: Effect of Turmeric Extract on the Level of TNF-α and IL-6 of Type 2 Diabetic Rats

The results are expressed as the Mean±SD. Different superscripts over the bars indicate significant difference (Tukey's-HSD multiple range post hoc test, $P<0.05$). NC=normal control, DC=diabetic control, C100=diabetic rats treated with 100mg/kg BW of concentration of turmeric extract, C200=diabetic rats treated with 200mg/kg BW of concentration of turmeric extract, DMET=diabetic rats treated with 500mg/kg BW of metformin

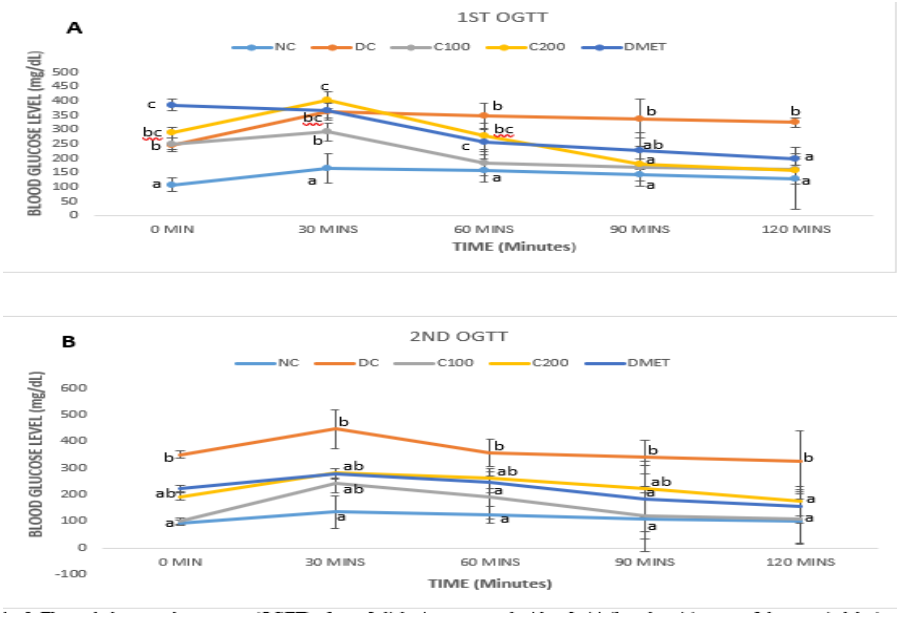

Figure 2: The Oral Glucose Tolerance Test (OGTT) of Type 2 Diabetic Rats Treated with Tofacitinib and Aspirin Over a 2-Hour Period During the 10th (A) and 20th (B) days of the Experiment

The results are expressed as the Mean±SD. Different superscripts over the bars indicate significant difference (Tukey's-HSD multiple range post hoc test, P<0.05). NC=normal control, DC=diabetic control, C100=diabetic rats treated with 100mg/kg BW of concentration of turmeric extract, C200=diabetic rats treated with 200mg/kg BW of concentration of turmeric extract, DMET=diabetic rats treated with 500mg/kg BW of metformin.

Table 1: Effect of Turmeric Extract on Lipid Profile of Type 2 Diabetic Rats

Parameters	CHO(mg/dl)	TRIG(mg/dl)	HDL(mg/dl)	LDL(mg/dl)
NC	22.60±5.32^{a}	24.18±4.29^{a}	14.25±2.87^{a}	9.68±1.18^{a}
DC	155.18±32.43^{b}	131.55±34.60^{b}	0.43±0.22^{b}	81.58±10.85^{b}
C100	43.60±6.47^{ac}	23.75±10.81^{a}	6.43±1.65^{c}	28.25±12.23^{ac}
C200	42.85±9.67^{ac}	44.35±18.06^{ac}	5.38±2.14^{c}	25.10±11.45^{ac}
DMET	76.43±16.40^{c}	86.13±3.69^{c}	5.25±1.26^{c}	50.73±4.82^{bc}

The results are expressed as the Mean±SD. Different superscripts across the rows indicate significant difference (Tukey's-HSD multiple range post hoc test, P<0.05). NC=normal control, DC=diabetic control, C100=diabetic rats treated with 100mg/kg BW of concentration of turmeric extract, C200=diabetic rats treated with 200mg/kg BW of concentration of turmeric extract, DMET=diabetic rats treated with 500mg/kg BW of metformin.

Table 2: Effect of Turmeric Extract on Artherogenic Indices of Type 2 Diabetic Rats

Parameters	AIP	CRI-I	CRI-II	AC
NC	0.24±0.11^{a}	1.75±0.45^{a}	0.71±0.17^{a}	0.41±0.23^{a}
DC	2.57±0.40^{b}	276.18±95.80^{b}	155.12±30.14^{b}	293.38±42.00^{b}
C100	0.54±0.13^{a}	3.57±0.91^{a}	4.54±2.44^{a}	6.13±2.00^{a}
C200	1.00±0.34^{ac}	7.95±3.60^{a}	4.30±1.58^{a}	8.45±3.89^{a}
DMET	1.20±0.26^{c}	18.01±10.55^{a}	9.22±7.68^{a}	11.91±8.61^{a}

The results are expressed as the Mean±SD. Different superscripts across the rows indicate significant difference (Tukey's-HSD multiple range post hoc test, P<0.05). NC=normal control, DC=diabetic control, C100=diabetic rats treated with 100mg/kg BW of concentration of turmeric extract, C200=diabetic rats treated with 200mg/kg BW of concentration of turmeric extract, DMET=diabetic rats treated with 500mg/kg BW of metformin.

Table 3: Effect of Turmeric Extract on Liver Function Parameters of Type 2 Diabetic Rats

Parameters	ALT (U/L)	AST(U/L)	ALP(U/L)
NC	12.75±3.20^{a}	14.50±3.32^{a}	15.25±1.42^{a}
DC	123.75±4.11^{b}	137.00±10.42^{b}	143.08±11.65^{b}
C100	23.00±5.47^{cd}	56.25±8.69^{c}	39.73±17.81^{c}
C200	19.00±2.94^{cd}	39.75±11.06^{ac}	33.23±10.18^{ac}

Parameters	ALT (U/L)	AST(U/L)	ALP(U/L)
DMET	27.75 ± 9.67^{d}	56.00 ± 31.59^{c}	39.20 ± 2.51^{c}

The results are expressed as the Mean±SD. Different superscripts across the rows indicate significant difference (Tukey's-HSD multiple range post hoc test, P<0.05). NC=normal control, DC=diabetic control, C100=diabetic rats treated with 100mg/kg BW of concentration of turmeric extract, C200=diabetic rats treated with 200mg/kg BW of concentration of turmeric extract, DMET=diabetic rats treated with 500mg/kg BW of metformin.

Table 4: Effect of Turmeric Extract on Kidney Function Parameters of Type 2 Diabetic Rats

Parameters	UREA(U/L)	CREATININE(U/L)
NC	15.00 ± 7.07^{a}	10.75 ± 0.96^{a}
DC	226.00 ± 12.68^{b}	183.50 ± 5.20^{b}
C100	71.50 ± 10.28^{c}	65.25 ± 7.41^{c}
C200	50.75 ± 18.59^{cd}	73.50 ± 9.15^{c}
DMET	41.00 ± 10.03^{ad}	25.50 ± 5.74^{d}

The results are expressed as the Mean±SD. Different superscripts across the rows indicate significant difference (Tukey's-HSD multiple range post hoc test, P<0.05). NC=normal control, DC=diabetic control, C100=diabetic rats treated with 100mg/kg BW of concentration of turmeric extract, C200=diabetic rats treated with 200mg/kg BW of concentration of turmeric extract, DMET=diabetic rats treated with 500mg/kg BW of metformin.

Discussion

This study investigated the potential of turmeric extract as an anti-inflammatory modulator in mitigating metabolic complications associated with type 2 diabetes. The findings demonstrated that turmeric extract effectively reduced systemic inflammation, improved glucose tolerance, and ameliorated dyslipidemia, liver dysfunction, and kidney impairment in diabetic rats. The significant elevation of TNF-α and IL-6 in the diabetic control group confirms the role of chronic inflammation in the progression of type 2 diabetes. These pro-inflammatory cytokines have been implicated in insulin resistance through their interference with insulin receptor signaling and activation of pro-inflammatory pathways such as the JAK-STAT and NF-κB cascades (Chen et al., 2015). The substantial reduction in TNF-α and IL-6 levels following turmeric extract administration underscores its anti-inflammatory properties. Curcumin and essential oils such as turmerones (ar-turmerone, α-turmerone, β-turmerone) are the primary bioactive compounds in turmeric, known to suppress NF-κB activation, thereby inhibiting cytokine production and reducing inflammatory stress(Orellana-Paucar, 2024; Sharifi-Rad et al., 2020). The results align with previous reports demonstrating curcumin's ability to attenuate systemic inflammation in metabolic disorders.

Importantly, the reduction in insulin resistance may have contributed to the improvements observed in glucose uptake and clearance, as seen in the oral glucose tolerance test results. These findings suggest that turmeric extract may enhance insulin sensitivity and promotes glucose uptake, possibly through activation of the AMPK pathway and suppression of inflammatory mediators that disrupt insulin signaling (Ghorbani et al., 2014). Previous studies have shown that curcumin enhances glucose homeostasis by modulating GLUT4 translocation to the cell surface for glucose transport into cells and reducing de novo hepatic glucose production, which could explain the observed improvements in dyslipidemia (Den Hartogh et al., 2020). High glucose production without appropriate utilization, commonly seen in insulin resistance or type 2 diabetes, leads to dyslipidemia through disrupted metabolic pathways. When glucose is overproduced by the liver and peripheral tissues cannot efficiently utilize it due to insulin resistance, the body compensates by increasing lipid metabolism for energy. This causes an elevated release of free fatty acids from adipose tissue, which are taken up by the liver and converted into triglycerides. The liver then packages these triglycerides into very low-density lipoproteins (VLDL) and releases them into the bloodstream. The result is a characteristic dyslipidemia marked by elevated triglyceride and VLDL levels, reduced high-density lipoprotein (HDL) cholesterol, and increased small, dense low-density lipoprotein (LDL) particles, all of which contribute to cardiovascular risk(Antar et al., 2023; Dilworth et al., 2021).

Dyslipidemia is a hallmark of type 2 diabetes, characterized by elevated cholesterol, triglycerides, and LDL, coupled with reduced HDL levels. The diabetic control group exhibited significant alterations in these lipid parameters, predisposing the animals to increased cardiovascular risk. Turmeric extract administration significantly improved lipid profiles, with both doses reducing cholesterol, LDL, and triglycerides while increasing HDL levels. This lipid-lowering effect may be attributed to curcumin's ability to enhance hepatic LDL receptor expression, suppress lipid synthesis via SREBP-1c inhibition, and increase reverse cholesterol transport (Kang & Chen, 2009; Xie et al., 2019). To further substantiate these findings, atherogenic indices which are reliable markers of cardiovascular risk were calculated. Treatment with turmeric extract resulted in significant reductions in the atherogenic index of plasma (AIP), Castelli risk index I (CRI-I), Castelli risk index II (CRI-II), and atherogenic coefficient (AC). Notably, the lower dose of turmeric extract demonstrated greater efficacy in reducing these indices, suggesting that moderate supplementation may offer optimal cardiovascular protection. These results are consistent with previous studies highlighting curcumin's role in modulating lipid metabolism and reducing atherosclerotic risk.

Furthermore, the observed reduction in pro-inflammatory cytokines (TNF-α and IL-6) in turmeric-treated groups underscores the contribution of chronic inflammation to dyslipidemia and cardiovascular complications in type 2 diabetes. In addition, the liver, being the central organ for metabolism, becomes overworked and burdened with excess free fatty acids, leading to their accumulation and subsequent destruction of hepatocytes. This hepatic lipid overload contributes to liver complications such as steatosis and inflammation (Ipsen et al., 2018; Liu et al., 2010). However, treatment with turmeric extract has been shown to reduce insulin resistance and improve lipid metabolism, thereby decreasing free fatty acid accumulation in the liver and mitigating hepatocellular damage. Liver dysfunction is a common complication in diabetes, often driven by oxidative

stress and chronic inflammation. The significantly elevated ALT, AST, and ALP levels in the diabetic control group indicate hepatic injury, likely due to increased lipid accumulation and mitochondrial dysfunction (Mobasheri et al., 2023; Mohamed et al., 2016). Turmeric extract treatment resulted in a dose-dependent reduction in these enzymes, suggesting hepatoprotective effects. The higher dose exhibited a more pronounced reduction in liver enzymes, indicating that turmeric extract mitigates hepatic damage by reducing oxidative stress, enhancing antioxidant defenses, and preventing lipid peroxidation. The hepatoprotective effects of turmeric extract are consistent with previous studies, which have reported its ability to reduce hepatic inflammation and fibrosis by modulating the Nrf2/HO-1 antioxidant pathway and suppressing pro-inflammatory cytokines (Rahban et al., 2020).

The improved glucose uptake observed as a result of reduced inflammation and insulin resistance has led to decreased glucose reabsorption by the renal tubules. Under diabetic conditions, persistent hyperglycemia causes excessive glucose to be filtered by the glomeruli, exceeding the reabsorptive capacity of the renal tubules. This high glucose load damages the tubular cells and contributes to the development of diabetic nephropathy (Vallon & Thomson, 2012). However, with improved glucose uptake by peripheral tissues, blood glucose levels are better regulated, reducing the burden on the kidneys and helping to protect renal cells from glucose-induced injury (Vallon & Thomson, 2012). Diabetes-induced nephropathy is associated with elevated urea and creatinine levels, reflecting impaired renal function due to inflammatory and oxidative damage to nephrons. The significant increase in these markers in the diabetic control group indicates renal dysfunction(Dabla, 2010). Turmeric extract treatment led to a substantial reduction in urea and creatinine levels, with both doses demonstrating nephroprotective effects. This suggests that turmeric extract protects against kidney injury, potentially by reducing inflammation, oxidative stress, and fibrosis. Previous research has highlighted curcumin's renoprotective effects through suppression of TGF-β signaling and inhibition of pro-inflammatory cytokine release, supporting the present findings (Ashrafizadeh et al., 2020).

Although the present findings demonstrate promising therapeutic effects of turmeric extract, this study has some limitations. Phytochemical standardization of the extract was limited to preliminary screening, and detailed characterization of the bioactive constituents was not performed. In addition, molecular mechanisms underlying the observed effects were not directly investigated. The relatively small sample size and short treatment duration may also limit broader interpretation of the findings. Therefore, further studies involving detailed phytochemical profiling, molecular analyses, toxicity evaluation, and long-term investigations are recommended.

The translational relevance of this study lies in the potential use of Turmeric as a complementary dietary or phytotherapeutic intervention for managing inflammation-associated metabolic complications in diabetes. Given its accessibility and traditional medicinal use, turmeric may provide a cost-effective adjunct approach, particularly in low-resource settings where the burden of diabetes is increasing. However, clinical studies are necessary to validate its efficacy and safety in humans.

CONCLUSION

Acetone extract of Turmeric demonstrated significant anti-inflammatory, hypoglycemic, hypolipidemic, hepatoprotective, and nephroprotective effects in streptozotocin-induced type 2 diabetic rats. The extract significantly reduced pro-inflammatory cytokines, improved lipid abnormalities, and attenuated liver and kidney dysfunction associated with diabetes. These findings suggest that turmeric extract may possess therapeutic potential as a complementary agent in the management of type 2 diabetes and its associated metabolic complications. However, the findings of this study are limited to evidence obtained from an experimental animal model, and further clinical investigations are required before extrapolation to human therapeutic applications.

Acknowledgement

Our sincere appreciation goes to Mr. Justus James, Dr. Tahir Mohammed, Dr. Abdulrahman Ahmad El-Fulaty, and Mr. Salim Sadiq Abubakar for their invaluable technical assistance. We also acknowledge Precision Biomedicals Laboratory, Kaduna State and the Department of Biochemistry and Pharmacy, Kaduna State University for providing access to the facilities used during this study.

Conflict of Interest

There is no conflict of interest

Author's Contribution

HYB and RA conceptualized the study, while the experimental design was developed by HYB and ZKM. BML and AOI conducted the experiments, and HYB performed the data analysis. HYB drafted the manuscript, which was reviewed by ZKM and RA.

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