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ARTICLE

Antidiabetic, Hypolipidaemic, and Antioxidant Potential of the Ethanolic Extract of Talinum cuneifolium Whole Plant in Streptozotocin-Induced Diabetic C57BL/6 Mice

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Abstract: Diabetes mellitus (DM) is a major non-communicable disease; current pharmacotherapy carries significant limitations, necessitating safer plant-based alternatives. To evaluate the antihyperglycaemic, hypolipidaemic, antioxidant, hepatoprotective, and insulin-modulatory activities of the ethanolic extract of *Talinum cuneifolium* whole plant (EETC) in streptozotocin (STZ)-induced diabetic C57BL/6 mice. Confirmed diabetic mice (n = 12/group) received EETC (200 or 400 mg/kg/day) or gliclazide (10 mg/kg) orally for 30 days. Fasting blood glucose, OGTT, lipid profile, hepatic antioxidant enzymes (SOD, CAT, GPx), MDA, liver markers (AST, ALT, ALP), serum insulin, and pancreatic histopathology were assessed. EETC (400 mg/kg) reduced fasting blood glucose to 108.4 ± 2.9 mg/dL versus 352.7 ± 10.8 mg/dL in diabetic controls ($p < 0.001$), restored lipid profile, antioxidant defences, and hepatic markers, and partially recovered serum insulin (51%) with near-normal islet architecture. EETC demonstrates broad-spectrum antidiabetic and metaboloprotective activity in STZ-induced C57BL/6 mice, validating its ethnomedicinal use and supporting phytopharmaceutical development.

Keywords: Talinum cuneifolium, diabetes mellitus, streptozotocin, hypolipidaemic, antioxidant, C57BL/6 mice, Antihyperglycaemic, Hepatoprotective, Phytochemistry.

1 Introduction

Diabetes mellitus (DM) is among the most burdensome non-communicable diseases of the modern era, characterised by chronic hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. An estimated 463 million adults lived with diabetes globally in 2019, a figure projected to reach 700 million by 2045 [1]. The disease drives a cascade of microvascular complications nephropathy, retinopathy, and neuropathy as well as macrovascular events including coronary artery disease and stroke, accounting for disproportionate global morbidity and mortality [2].

Contemporary management strategies encompass insulin, sulfonylureas, biguanides, thiazolidinediones, DPP-4 inhibitors, SGLT-2 inhibitors, and GLP-1 receptor agonists. Despite effective short-term glycaemic control, prolonged use is limited by hypoglycaemia risk, gastrointestinal intolerance, weight gain, cardiovascular contraindications, and prohibitive cost in low-to-middle-income countries [3]. These limitations have catalysed renewed interest in plant-derived phytomedicines as safer, more affordable antidiabetic strategies.

Medicinal plants offer a chemically diverse repertoire of flavonoids, alkaloids, tannins, saponins, and phenolic acids that modulate glycaemic homeostasis through complementary mechanisms: inhibition of intestinal α-glucosidase and α-amylase, stimulation of pancreatic insulin secretion, enhancement of peripheral glucose uptake via GLUT-4 translocation, suppression of hepatic gluconeogenesis via AMPK activation, and reduction of oxidative stress-mediated β-cell injury [4,5]. Traditional systems including Ayurveda and Traditional Chinese Medicine have long exploited such activity for diabetes management [6].

Streptozotocin (STZ)-induced diabetes is the most widely validated experimental model for evaluating antidiabetic compounds. STZ selectively alkylates DNA in pancreatic β-cells, triggering necrosis and insulin deficiency closely mirroring Type 1

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DM pathophysiology [7]. The C57BL/6 inbred strain is the gold-standard murine background for metabolic disease research, offering a defined genetic phenotype and extensive reference data for cross-study comparability.

Talinum cuneifolium Willd. (family Portulacaceae) is an ethnomedicinal succulent distributed across tropical Asia and Africa, traditionally used in South Asian medicine for diabetes and metabolic disorders. The related species T. triangulare has demonstrated significant antihyperglycaemic and hypolipidaemic activity in STZ-induced diabetic models [8]. Prior investigations employing leaf extracts of T. cuneifolium at 200–400 mg/kg confirmed blood glucose-lowering activity in alloxan-diabetic rodents [9,10]; however, whole-plant ethanolic extraction maximises recovery of the full phytochemical profile, and the impact of EETC on antioxidant status, hepatic function, insulin secretion, and pancreatic morphology has not been systematically investigated.

The present study therefore comprehensively evaluates the antihyperglycaemic, hypolipidaemic, antioxidant, hepatoprotective, and insulin-modulatory activities of the ethanolic extract of T. cuneifolium whole plant (EETC) in STZ-induced diabetic C57BL/6 mice over 30 days, with gliclazide as a clinically relevant positive control.

2 Materials and Methods

2.1 chemicals and Reagents

Streptozotocin was procured from Sisco Research Laboratories Pvt. Ltd. (Mumbai, India). Gliclazide was obtained from Konis Pharmaceutical Pvt. Ltd. (Solon, India). A mouse insulin ELISA kit was sourced from Mercodia AB (Uppsala, Sweden). Reagents for antioxidant enzyme assays (SOD, CAT, GPx) and the TBARS lipid peroxidation kit were from Sigma-Aldrich (St. Louis, MO, USA). All remaining biochemical reagents and diagnostic kits were of analytical grade.

2.2 Plant Collection, Authentication, and Extraction

Fresh Talinum cuneifolium whole plant was collected from Andhra Pradesh, India (March 2024) and authenticated by a qualified taxonomist (Voucher No. HB-TC-2024-011). Shade-dried material was powdered (sieve No. 22) and Soxhlet-extracted with 95% ethanol at 55–65°C for 72 hours. The filtrate was concentrated by rotary evaporation at 40°C to yield a dark-green semisolid residue (yield: 8.4% w/w). Preliminary phytochemical screening confirmed the presence of alkaloids, flavonoids, phenolic compounds, tannins, saponins, steroids, carbohydrates, and reducing sugars (Supplementary Table S1). For oral dosing, fresh suspensions were prepared daily in 1% Tween 80 (w/v).

2.3 Animals and Ethical Approval

Male C57BL/6 mice (20–25 g, 8–10 weeks) were procured from M/s Sri Raghavendra Enterprises (Bangalore, India) and housed in individually ventilated cages (22 ± 2°C, 50–60% humidity, 12 h light/dark cycle) with standard chow and water ad libitum. All procedures were approved by the Institutional Animal Ethics Committee (Approval No. IAEC/2024/012) and conducted in accordance with CPCSEA guidelines adhering to the 3Rs principles.

2.4 Acute Toxicity (OECD TG 423)

Acute oral toxicity was evaluated in female C57BL/6 mice (n = 3/dose) per OECD TG 423 at 300, 1000, and 2000 mg/kg. No mortality, behavioural abnormalities, or gross pathological changes were observed at any dose up to 2000 mg/kg; EETC was classified as Category 5 (LD50 > 2000 mg/kg). Therapeutic doses of 200 and 400 mg/kg (1/10 and 1/5 of the no-effect dose) were selected.

2.5 Diabetes Induction and Experimental Design

Diabetes was induced by a single i.p. injection of STZ (150 mg/kg) in 0.1 M citrate buffer (pH 4.5) following a 12 h fast. Animals with fasting blood glucose ≥ 250 mg/dL at 72 h post-injection were included. Sixty confirmed diabetic mice were randomised into five groups (n = 12/group) as shown in Table 1 and treated orally for 30 consecutive days.

2.6 Biochemical Assays

Fasting blood glucose was measured by the GOD-POD colorimetric method at baseline and on Days 1, 7, 14, 21, and 30. The OGTT was performed on Day 28 (oral glucose load 2 g/kg; blood sampled at 0, 30, 60, 90, 120 min). At study termination, serum was collected by cardiac puncture for lipid profile (GOD-POD, CHOD-POD, precipitation, Friedewald equation), total protein (Biuret), albumin (bromocresol green), uric acid (uricase-PAP), AST/ALT (IFCC kinetic UV), ALP (p-nitrophenyl phosphate), and insulin (ELISA, Mercodia). Hepatic antioxidant enzymes (SOD, CAT, GPx) and lipid peroxidation (MDA/TBARS) were assayed in post-mitochondrial supernatant using validated spectrophotometric methods. Pancreatic sections (5 µm, H&E) were evaluated by a blinded

Table 1. Experimental group design and treatment protocol (n = 12 per group)

Group	Description	Treatment	Dose	Route	Duration
I	Normal Control	Vehicle (1% Tween 80)	—	Oral gavage	30 days
II	Diabetic Control	Vehicle (1% Tween 80)	—	Oral gavage	30 days
III	EETC Low Dose	EETC	200 mg/kg	Oral gavage	30 days
IV	EETC High Dose	EETC	400 mg/kg	Oral gavage	30 days
V	Positive Control	Gliclazide	10 mg/kg	Oral gavage	30 days

STZ = Streptozotocin; EETC = Ethanolic Extract of Talinum cuneifolium; Vehicle = 1% Tween 80 in distilled water.

pathologist using a semiquantitative islet scoring system (0–3). Full assay details are provided in Supplementary Methods.

2.7 Statistical Analysis

Data are expressed as mean ± SEM (n = 12). One-way ANOVA followed by Tukey's HSD post hoc test was performed using GraphPad Prism v9.0. Significance thresholds: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ vs. normal control; † $p < 0.05$, †† $p < 0.01$, ††† $p < 0.001$ vs. diabetic control.

3 Results

3.1 Phytochemical Screening

Qualitative phytochemical analysis confirmed the presence of alkaloids, flavonoids, phenolic compounds, tannins, saponins, steroids, carbohydrates, and reducing sugars in the ethanolic extract. Proteins were not detected. The complete phytochemical profile is presented in Supplementary Table S1. The rich polyphenolic and flavonoid constitution provides a mechanistic basis for the observed antidiabetic, antioxidant, and lipid-modulating activities.

3.2 Effect of EETC on Fasting Blood Glucose

STZ injection produced rapid, sustained hyperglycaemia confirmed at 72 hours (>250 mg/dL). Diabetic control mice showed progressive glucose elevation from 318.4 ± 10.6 mg/dL (Day 1) to 352.7 ± 10.8 mg/dL (Day 30). EETC produced a significant, dose-dependent reduction throughout the treatment period (Table 2; Figure 1). The 400 mg/kg group reduced fasting blood glucose to 108.4 ± 2.9 mg/dL by Day 30 (-69% vs. diabetic control; $p < 0.001$), approaching the gliclazide group (94.8 ± 2.3 mg/dL; -73%). Normal controls remained stable at 88.7 ± 1.3 mg/dL throughout. In normoglycaemic mice, EETC did not produce significant hypoglycaemia (Supplementary Table S2).

3.3 Effect of EETC on Oral Glucose Tolerance (OGTT)

Diabetic control mice exhibited a severely blunted glucose disposal capacity, with blood glucose peaking at 437.6 ± 8.9 mg/dL at 60 minutes and remaining elevated at 374.5 ± 8.2 mg/dL at 120 minutes (Figure 2). EETC treatment dose-dependently and significantly attenuated this excursion. The 400 mg/kg group achieved a 120-minute blood glucose of 74.6 ± 1.6 mg/dL, statistically indistinguishable from gliclazide (73.2 ± 2.1 mg/dL; $p > 0.05$). These findings indicate that EETC effectively limits intestinal glucose absorption and/or enhances peripheral glucose disposal. Detailed OGTT values and AUC_{0-120} are presented in Supplementary Table S3.

3.4 Effect of EETC on Plasma Lipid Profile and Uric Acid

STZ-induced diabetic mice demonstrated pronounced dyslipidaemia: triglycerides elevated by 55%, total cholesterol by 52%, LDL-cholesterol by 1,872%, and VLDL-cholesterol by 63%, while HDL-cholesterol was depleted by 39% vs. normal controls. Serum uric acid was also significantly elevated (2.18 ± 0.03 vs. 1.22 ± 0.02 mg/dL; $p < 0.001$). EETC treatment dose-dependently corrected all parameters (Figure 3), with the 400 mg/kg group achieving near-normal values comparable to gliclazide. Full lipid and uric acid data are detailed in Supplementary Table S4.

3.5 Antioxidant, Hepatoprotective, Insulin, and Histopathological Outcomes

STZ induction caused profound hepatic oxidative stress (SOD -61%, CAT -60%, GPx -58% vs. normal; MDA elevated 4.3-fold) alongside significant hepatocellular injury (AST, ALT, ALP markedly elevated) and near-complete insulin depletion (1.74 ± 0.13 vs. 19.28 ± 0.82 µIU/mL; -91%). EETC 400 mg/kg dose-dependently restored all three antioxidant enzymes to approximately 85% of normal, reduced MDA by 61%, normalised hepatic

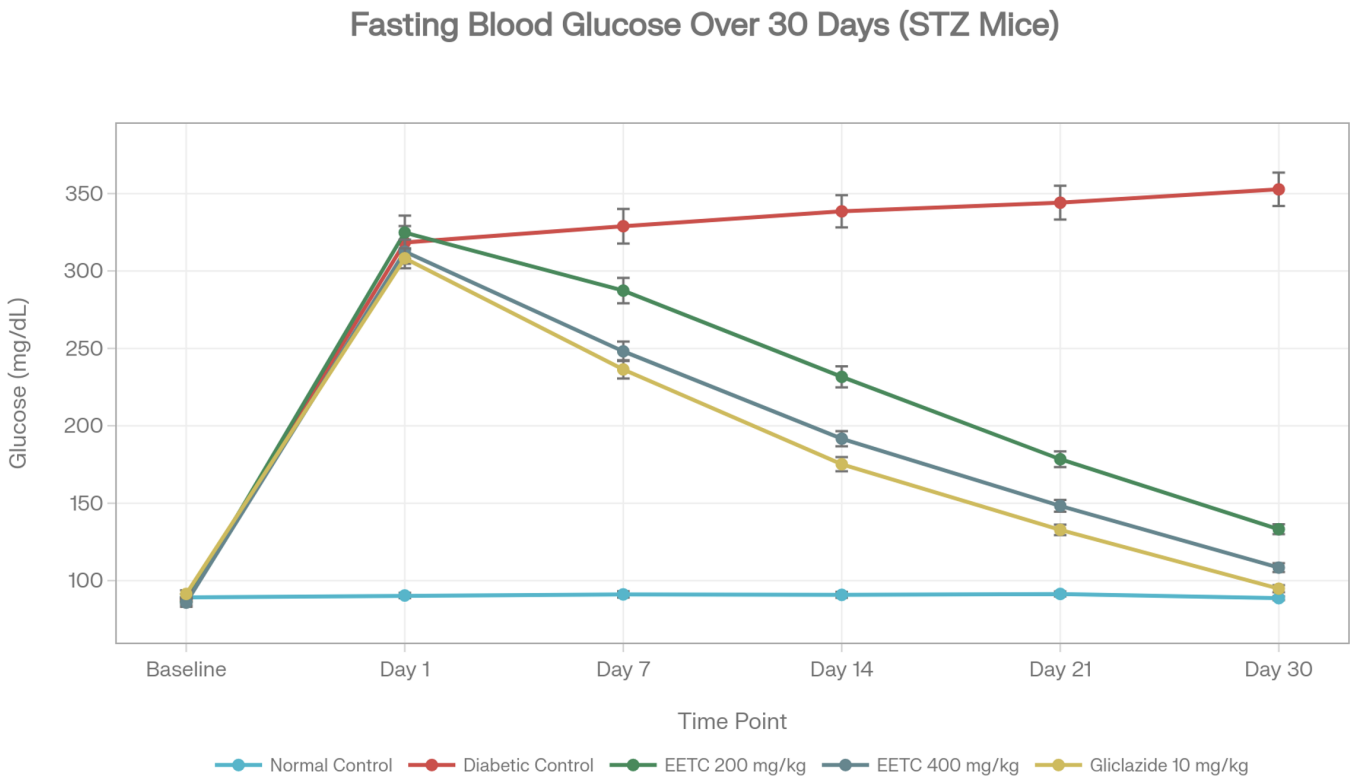

Figure 1. Fasting blood glucose levels in STZ-induced diabetic C57BL/6 mice over 30 days of EETC treatment. Values are Mean ± SEM (n = 12 per group). ***$p < 0.001$ vs. Normal Control (Group I); $^{\dagger\dagger\dagger}p < 0.001$ vs. Diabetic Control (Group II). One-way ANOVA, Tukey's HSD post hoc test.

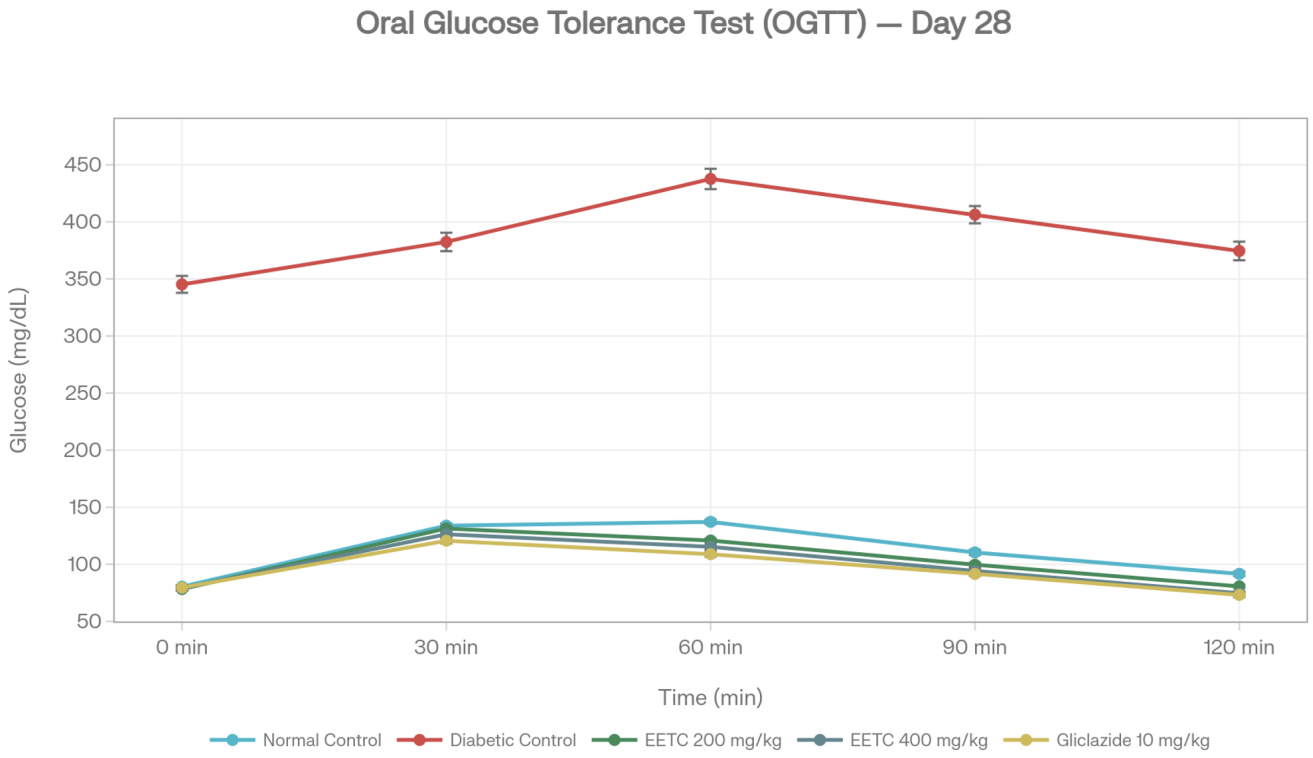

Figure 2. Oral Glucose Tolerance Test (OGTT) performed on Day 28 following an oral glucose load of 2 g/kg body weight. Values are Mean ± SEM (n = 12 per group). ***$p < 0.001$ vs. Normal Control; $^{\dagger\dagger\dagger}p < 0.001$ vs. Diabetic Control.

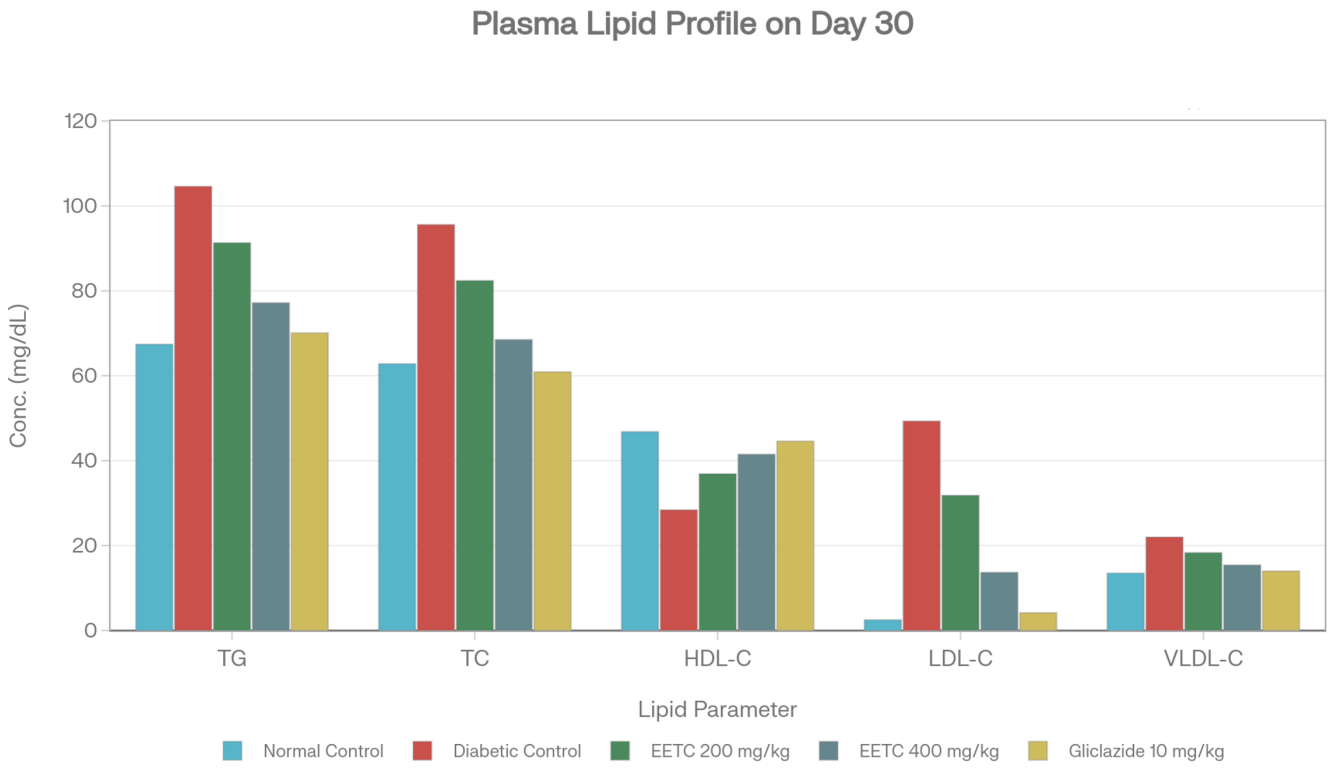

Figure 3. Effect of EETC on plasma lipid parameters (TG = Triglycerides; TC = Total Cholesterol; HDL-C = High-Density Lipoprotein Cholesterol; LDL-C = Low-Density Lipoprotein Cholesterol; VLDL-C = Very Low-Density Lipoprotein Cholesterol) on Day 30. Values are Mean ± SEM (n = 12). ***p < 0.001 vs. Normal Control; †††p < 0.001, ††p < 0.01 vs. Diabetic Control.

Table 2. Effect of EETC on fasting blood glucose in STZ-induced diabetic C57BL/6 mice (Mean ± SEM; n = 12)

Group	Baseline (mg/dL)	Day 1 (mg/dL)	Day 7 (mg/dL)	Day 14 (mg/dL)	Day 21 (mg/dL)	Day 30 (mg/dL)
Group I – Normal Control	89.2 ± 2.1	90.4 ± 1.7	91.1 ± 2.0	90.8 ± 1.9	91.4 ± 1.6	88.7 ± 1.3
Group II – Diabetic Control	87.6 ± 1.6	318.4 ± 10.6***	328.9 ± 11.2***	338.5 ± 10.4***	344.1 ± 10.9***	352.7 ± 10.8***
Group III – EETC 200 mg/kg	86.3 ± 2.0	324.8 ± 10.9***	287.3 ± 8.2†††	231.6 ± 6.8†††	178.4 ± 5.1†††	133.2 ± 3.2†††
Group IV – EETC 400 mg/kg	85.8 ± 2.7	312.5 ± 7.8***	248.1 ± 6.3†††	191.7 ± 4.9†††	148.3 ± 3.8†††	108.4 ± 2.9†††
Group V – Gliclazide 10 mg/kg	91.4 ± 2.4	308.2 ± 6.5***	236.4 ± 5.9†††	175.2 ± 4.6†††	132.8 ± 3.4†††	94.8 ± 2.3†††

*** p < 0.001 vs. Normal Control (Group I); ††† p < 0.001 vs. Diabetic Control (Group II). One-way ANOVA followed by Tukey's HSD post hoc test

marker enzymes, restored serum insulin to 51.2% of normal, and preserved near-normal pancreatic islet architecture on histopathology. Body weight loss was also dose-dependently attenuated, and serum protein fractions were restored toward normal values. Full data for all these endpoints are presented in Supplementary Tables S5–S7.

4 Discussion

The present study provides a comprehensive pharmacological characterisation of EETC in STZ-induced diabetic C57BL/6 mice. The adoption of inbred C57BL/6 mice over the outbred Wistar rats used in earlier studies represents a methodological advancement, offering a defined metabolic phenotype and superior cross-laboratory reproducibility [7]. The

STZ dose of 150 mg/kg yielded consistent diabetes (fasting glucose >250 mg/dL) in all confirmed animals within 72 hours.

The significant, dose-dependent reduction in fasting blood glucose by EETC- with 400 mg/kg achieving near-normal values (108.4 ± 2.9 mg/dL) by Day 30 (Figure 1, Table 2) aligns with published phytomedicinal evidence for flavonoid- and phenolic-mediated antihyperglycaemia [13,14]. These constituents modulate glycaemic homeostasis through α-glucosidase and α-amylase inhibition, pancreatic insulin secretion stimulation, GLUT-4 upregulation, and AMPK-mediated suppression of hepatic gluconeogenesis [15,16]. The absence of significant hypoglycaemia in normoglycaemic mice (Supplementary Table S2) confirms a glucose-dependent mechanism, a key safety advantage over sulfonylurea secretagogues [17].

The OGTT data (Figure 2) confirm markedly improved postprandial glucose disposal, with EETC 400 mg/kg achieving 120-min blood glucose statistically indistinguishable from gliclazide (74.6 ± 1.6 vs. 73.2 ± 2.1 mg/dL; $p > 0.05$). This effect likely reflects combined inhibition of intestinal brush-border carbohydrate-hydrolysing enzymes and enhanced peripheral glucose utilisation, well-documented for plant-derived flavonoids and phenolic acids [16].

The comprehensive correction of dyslipidaemia (Figure 3; Supplementary Table S4) is mechanistically attributable to HMG-CoA reductase inhibition, enhanced lipoprotein lipase activity, and reduced intestinal cholesterol absorption by EETC phytoconstituents [18,19]. Concomitant uric acid normalisation suggests anti-xanthine-oxidase activity clinically relevant given the association between hyperuricaemia, insulin resistance, and cardiovascular risk [20].

The antioxidant enzyme and MDA data (Supplementary Table S5) provide the most mechanistically informative findings: restoration of SOD, CAT, and GPx to ~85% of normal and 61% MDA reduction at 400 mg/kg. This antioxidant cytoprotection underlies the partial β-cell preservation confirmed histopathologically and the 51.2% insulin recovery (Supplementary Table S7). Flavonoids and tannins in EETC are well-established Nrf2/HO-1 pathway activators [22]. Hepatoprotection evidenced by normalisation of AST, ALT, and ALP (Supplementary Table S6) further supports restored anabolic metabolism, consistent with body weight preservation and serum protein fraction restoration [18,21].

Several limitations should be acknowledged. The investigation employed a crude extract rather than a standardised or fractionated preparation; active constituent(s) remain unidentified. Molecular signalling pathway analyses (IRS-1/PI3K/Akt, AMPK, Nrf2) were not performed. Future studies should employ bioactivity-guided fractionation, chronic toxicity evaluation, in vitro enzyme inhibition assays, and transcriptomic approaches to delineate precise molecular targets and facilitate clinical translation.

5 Conclusion

The ethanolic extract of Talinum cuneifolium whole plant (EETC) demonstrates broad-spectrum antidiabetic and metaboloprotective activity in STZ-induced diabetic C57BL/6 mice. At 400 mg/kg/day for 30 days, EETC significantly and dose-dependently reduced fasting blood glucose by 65%, restored oral glucose tolerance, corrected dyslipidaemia and hyperuricaemia, normalised serum proteins, restored hepatic antioxidant defences, reduced lipid peroxidation, protected hepatocellular integrity, partially restored insulin secretion (51.2%), and preserved near-normal pancreatic islet architecture effects largely comparable to gliclazide. These findings provide robust preclinical validation for the ethnomedicinal use of T. cuneifolium and strongly support its further development as a safe, multi-target phytotherapeutic antidiabetic candidate.

Conflicts of Interest

The authors declare no competing interests.

Ethical Approval

All animal experiments were approved by the Institutional Animal Care and Use Committee of Acharya Nagarjuna University.

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