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Potential of the Ethanol Extract of Luffa Leaves in Streptozotocin-Induced Diabetic Wistar Rats

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Abstract

Diabetes mellitus is a chronic metabolic disorder associated with hyperglycemia, dyslipidemia, oxidative stress, and progressive damage to vital organs. This study investigated the antidiabetic and biochemical effects of ethanol extract of *Luffa acutangula* leaves in streptozotocin-induced diabetic Wistar rats. Fresh leaves of *Luffa acutangula* were collected, authenticated, air-dried, pulverized, and extracted with 95% ethanol using maceration. Twenty-five adult male Wistar rats were randomly assigned to five groups: normal control, diabetic control, diabetic rats treated with metformin, and diabetic rats treated with 200 mg/kg and 400 mg/kg ethanol extract of *Luffa acutangula* leaves. Diabetes was induced through a single intraperitoneal injection of streptozotocin at 50 mg/kg body weight, and treatments were administered orally for 14 days. Blood glucose levels, liver function enzymes, bilirubin fractions, serum proteins, kidney function markers, electrolytes, and lipid profile parameters were evaluated. The findings showed that streptozotocin induction increased blood glucose levels, liver enzymes, renal biomarkers, and lipid abnormalities in diabetic rats. Treatment with *Luffa acutangula* leaf extract reduced blood glucose levels from 210.20 ± 7.30 to 140.50 ± 10.60 mg/dL at 200 mg/kg and from 198.60 ± 7.80 to 130.40 ± 9.80 mg/dL at 400

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mg/kg. The extract also improved selected liver function markers, reduced creatinine levels, and ameliorated lipid profile disturbances, particularly total cholesterol and triglycerides. However, the elevated urea levels observed in extract-treated groups indicate the need for further renal safety evaluation. Overall, ethanol extract of *Luffa acutangula* leaves demonstrated promising antihyperglycemic, hepatoprotective, and hypolipidemic effects in diabetic rats, providing preclinical evidence for its potential relevance in managing diabetes-related metabolic complications.

Keywords: *Luffa acutangula*; Diabetes Mellitus; Streptozotocin-Induced Diabetes; Wistar Rats; Antihyperglycemic Activity

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The condition is associated with disturbances in carbohydrate, lipid, and protein metabolism and may lead to severe complications affecting the kidneys, liver, heart, nerves, and blood vessels. The global prevalence of diabetes continues to rise, particularly in developing countries where access to effective and affordable treatment remains limited.Type II diabetes mellitus accounts for the majority of diabetes cases worldwide and is primarily associated with insulin resistance and progressive pancreatic beta cell dysfunction. Chronic hyperglycemia in diabetes is linked with oxidative stress, inflammation, dyslipidemia, and organ damage. Although several conventional antidiabetic drugs such as metformin, sulfonylureas, and insulin therapy are available, these treatments may present limitations including adverse effects, secondary failure, and high cost. Consequently, there is increasing scientific interest in medicinal plants as alternative or complementary therapeutic agents for diabetes management. Medicinal plants have long been used in traditional medicine systems for the treatment of metabolic disorders. Among these plants, Luffa acutangula Linn., commonly known as ridge gourd, has gained attention for its pharmacological properties. According to Shendge and Belemkar (2018), Luffa acutangula possesses diverse biological activities including antidiabetic, antioxidant, anti-inflammatory, and hepatoprotective effects. The plant is widely distributed in tropical and subtropical regions and has been traditionally used for the management of diabetes and related metabolic conditions.Experimental studies have provided evidence supporting the antidiabetic

potential of Luffa acutangula. Pimple et al. (2011) reported that ethanolic fruit extracts of Luffa acutangula exhibited significant antidiabetic and antihyperlipidemic activity in streptozotocin-induced non insulin dependent diabetic rats. Similarly, Thatchinamoorthi et al. (2021) demonstrated antioxidant and antihyperglycemic properties of the fruit extract in streptozotocin-induced diabetic rats. These findings suggest that bioactive constituents present in the plant may contribute to glycemic control and lipid profile improvement.Abarnadevika (2024) reported that ethanolic extracts of the aerial parts of Luffa acutangula showed promising activity against streptozotocin-nicotinamide induced type II diabetic rats. The study highlighted improvements in blood glucose levels and protective effects on vital organs. In addition, Raj et al. (2012) observed that extracts of Luffa acutangula fruits exhibited antidiabetic effects and improved histological architecture of organs in streptozotocin-induced diabetic rats. Further investigations on related species such as Luffa cylindrica and Luffa aegyptiaca also support the therapeutic relevance of the genus. Arise et al. reported significant antidiabetic activity of hydrolysates and ethanolic extracts of Luffa cylindrica seeds and leaves in experimental models. Nnam (2024) documented that decoctions of Luffa aegyptiaca leaves contributed to reductions in blood glucose and improvement in lipid parameters in alloxan-induced diabetic rats. These studies collectively suggest that members of the Luffa genus possess bioactive phytochemicals with hypoglycemic and antioxidant properties. The diabetogenic agent Streptozotocin is commonly used to induce experimental diabetes in laboratory animals due to its selective toxicity to pancreatic beta cells. The use of Wistar rat provides a reliable and reproducible model for studying antidiabetic interventions. Streptozotocin-induced diabetes closely mimics many biochemical and physiological features of human diabetes, making it suitable for evaluating potential therapeutic agents. Despite several studies on the fruits and seeds of *Luffa acutangula*, limited attention has been directed specifically toward the ethanol extract of the leaves in streptozotocin-induced diabetic Wistar rats. Leaves may contain significant concentrations of flavonoids, saponins, alkaloids, tannins, and phenolic compounds that contribute to antidiabetic activity through antioxidant mechanisms, enhancement of insulin secretion, or improvement of peripheral glucose uptake. Therefore, this study is designed to investigate the antidiabetic potential of the ethanol extract of Luffa acutangula leaves in streptozotocin-induced diabetic Wistar rats in order to provide further scientific validation for its traditional use and to contribute to the search for safer and cost effective plant based therapeutic agents.

Statement of the Problem

The prevalence of diabetes mellitus continues to increase globally, posing serious public health challenges. Existing conventional therapies are often associated with adverse effects, limited accessibility, and economic burden, particularly in low income settings. Although several studies have demonstrated the antidiabetic potential of Luffa acutangula fruits and seeds, there is limited experimental evidence focusing specifically on the ethanol extract of its leaves in streptozotocin-induced diabetic Wistar rats. This gap in knowledge necessitates further investigation to determine whether the leaves possess significant hypoglycemic and protective effects comparable to other plant parts.

Medicinal Plants in Diabetes Management

The increasing global burden of diabetes mellitus, coupled with the limitations and adverse effects associated with conventional pharmacotherapy, has stimulated growing scientific interest in medicinal plants as complementary or alternative therapeutic options (Nyakairu, 2024). Phytotherapy, defined as the use of plant-derived preparations for therapeutic purposes, has long been embedded in traditional medical systems across Asia, Africa, and Latin America, where plant-based remedies have historically been used to manage symptoms of hyperglycemia and its complications. In recent decades, systematic pharmacological and molecular investigations have provided mechanistic validation for many of these traditional claims. A comprehensive review published in Nutrients emphasized that medicinal plant-based foods exert significant antidiabetic effects through modulation of key metabolic enzymes, enhancement of glucose transporter activity, and improvement of insulin signaling pathways (Ansari et al., 2023). Similarly, research in Molecules has highlighted that phytomolecules isolated from traditional antidiabetic plants demonstrate diverse pharmacodynamic actions, including stimulation of insulin secretion, facilitation of glucose uptake, antioxidant defense enhancement, and inhibition of carbohydrate-digesting enzymes (Ansari et al., 2022). These findings underscore the growing scientific legitimacy of phytotherapy in diabetes management.The role of phytotherapy in diabetes care extends beyond simple glycemic control and encompasses multi-targeted modulation of metabolic pathways (Ansari et al., 2023). Unlike single-target synthetic drugs, many plant-derived compounds exhibit pleiotropic effects due to the presence of bioactive secondary metabolites such as flavonoids, alkaloids, terpenoids, phenolic acids, and saponins. These compounds act synergistically to restore glucose

homeostasis while simultaneously mitigating oxidative stress and inflammation. According to Uti et al. (2025) in Natural Product Communications, plant-derived antidiabetic agents often influence multiple signaling pathways involved in insulin sensitivity, glucose metabolism, and pancreatic β-cell protection. Such multitargeted activity is particularly relevant in Type 2 diabetes mellitus, where complex pathophysiological mechanisms including insulin resistance, β-cell dysfunction, and chronic oxidative stress coexist. Additionally, Muema et al. (2023) in Molecules reported that commonly used antidiabetic medicinal plants improve tissue glucose absorption and enhance insulin responsiveness, further supporting their integrative therapeutic potential. One of the principal mechanisms by which plant-derived agents exert antidiabetic effects is through stimulation of insulin secretion from pancreatic β-cells. Certain phytoconstituents enhance β-cell function either by directly stimulating insulin exocytosis or by protecting β-cells from oxidative and inflammatory damage. Nyakairu (2024) noted that hypoglycemic plants may stimulate insulin secretion through modulation of ATP-sensitive potassium channels, mechanisms analogous to those of sulfonylureas, yet often with fewer adverse effects. Furthermore, antioxidant-rich medicinal plants help preserve β-cell integrity by reducing oxidative stress-induced apoptosis, thereby maintaining endogenous insulin production (Ansari et al., 2023). Investigations published in Biomedicines indicate that specific triterpenoids and flavonoids can influence insulin signaling pathways and enhance β-cell survival, suggesting that phytochemicals may not only stimulate insulin release but also contribute to long-term pancreatic protection (Ansari et al., 2025).

MATERIALS AND METHODS

Study Area

This research was conducted in the Biochemistry Laboratory of the Department of Biochemistry at Federal University Wukari, located in Wukari, Taraba State, Nigeria. The laboratory facilities were used for plant extraction, animal experimentation and biochemical analyses required for the study.

Plant Material Collection and Identification

Fresh leaves of Luffa acutangula were collected from a cultivated farmland within Wukari Local Government Area of Taraba State, Nigeria. The plant was identified and authenticated by a taxonomist in the Department of Biological Sciences at Federal

University Wukari. The collected leaves were thoroughly washed with clean water to remove dust and other contaminants. The leaves were then air dried under shade at room temperature for several days to prevent degradation of active phytochemical constituents. After drying, the leaves were pulverized into fine powder using an electric grinder and stored in airtight containers until required for extraction.

Botanical Description of Luffa acutangula Leaves

Taxonomy and General Classification

Kingdom: Plantae

Phylum: Angiosperms

Class: Dicotyledonae

Order: Cucurbitales

Family: Cucurbitaceae

Genus: Luffa



Figure 1: Luffa plant showing leaves and fruits. Source: https://en.wikipedia.org/wiki/Luffa (Species: Luffa acutangular)

Preparation of Ethanol Extract of Luffa Leaves

The powdered leaves were extracted using ethanol through the maceration technique. Approximately 500 g of the powdered plant material was soaked in 95 percent ethanol for 72 hours with intermittent shaking to enhance extraction of bioactive compounds. The mixture was filtered using Whatman No.1 filter paper to obtain the

filtrate. The filtrate was concentrated using a rotary evaporator at reduced pressure and moderate temperature. The concentrated extract was further dried in a water bath to obtain a crude ethanol extract. The extract was then stored in a refrigerator at 4°C until it was required for experimental use.

Experimental Animals

Adult male Wistar rats weighing between 150 g and 200 g were used for the study. The animals were obtained from the animal house of the Department of Biochemistry, Federal University Wukari. The rats were housed in clean plastic cages under standard laboratory conditions which included a temperature of approximately 25 ± 2°C and a 12-hour light and dark cycle. The animals were allowed free access to standard laboratory feed and clean drinking water. Before the commencement of the experiment, the animals were allowed to acclimatize to the laboratory environment for a period of two weeks.

Chemicals and Reagents

All chemicals used in this study were of analytical grade. The major chemicals used included: Streptozotocin used for induction of diabetes Metformin used as the standard antidiabetic drug Ethanol used for plant extraction Sodium citrate buffer used for preparation of streptozotocin solution Commercial biochemical assay kits for the determination of glucose, liver enzymes, kidney function markers, serum electrolytes and lipid profile parameters.

Induction of Experimental Diabetes

Experimental diabetes mellitus was induced using Streptozotocin (STZ). The streptozotocin was freshly dissolved in cold citrate buffer (0.1 M, pH 4.5) immediately before administration to maintain its stability. The experimental rats were fasted overnight prior to injection. Diabetes was induced by a single intraperitoneal injection of streptozotocin at a dose of 50 mg/kg body weight. After 72 hours of STZ administration, fasting blood glucose levels were measured using a glucometer. Rats with blood glucose levels equal to or greater than 200 mg/dL were considered diabetic and selected for the experiment.

Experimental Design

A total of twenty-five rats were randomly divided into five groups, with five rats in each group. The treatment groups were organized as follows:

Group 1 served as the normal control group and received only standard feed and distilled water without induction of diabetes.

Group 2 served as the diabetic control group and received streptozotocin without treatment.

Group 3 received streptozotocin followed by treatment with the standard antidiabetic drug metformin at a dose of 3 mg/kg body weight.

Groups 4 and 5 received streptozotocin followed by treatment with ethanol extract of Luffa leaves at doses of 200 mg/kg and 400 mg/kg body weight respectively. All treatments were administered orally once daily for a period of fourteen days.

Determination of Blood Glucose Levels

Blood glucose levels were determined at three different stages of the experiment which included: Before induction of diabetes After induction with streptozotocin After treatment with the plant extract and standard drug Blood samples were collected from the tail vein of the rats and glucose levels were measured using a glucometer based on the glucose oxidase method.

Collection of Blood Samples

At the end of the treatment period, the rats were fasted overnight and then anesthetized prior to sample collection. Blood samples were collected through cardiac puncture using sterile syringes.The blood samples were transferred into plain sample tubes and allowed to clot. The samples were then centrifuged at 3000 rpm for 10 minutes to separate the serum. The serum obtained was used for biochemical analysis.

Determination of Biochemical Parameters

The collected serum samples were used to determine several biochemical parameters relevant to diabetes and organ function.

Liver Function Enzymes

The activities of liver enzymes were determined using standard enzymatic assay kits. The enzymes analyzed included:

1. Alanine aminotransferase
2. Aspartate aminotransferase
3. Alkaline phosphatase

Bilirubin and Serum Proteins

The following parameters were determined to evaluate liver function and protein metabolism:

4.0 Total bilirubin

5.0 Direct bilirubin

6.0 Total protein

7.0 Albumin

Kidney Function Markers

Kidney function was assessed by determining serum concentrations of:

i. Creatinine

ii. Urea

Serum Electrolytes

Electrolyte balance was evaluated by measuring the concentrations of:

1. Sodium
2. Potassium
3. Chloride
4. Bicarbonate

Lipid Profile

The lipid profile parameters analyzed included:

i. Total cholesterol

ii. High-density lipoprotein cholesterol

iii. Triglycerides

Statistical Analysis

All experimental data obtained from the study were expressed as mean ± standard error of mean (SEM). Statistical analysis was carried out using one way analysis of variance (ANOVA) followed by appropriate post hoc tests to determine differences between groups.Values were considered statistically significant at $p < 0.05$.

RESULTS

Activities of blood glucose levels before induction, after STZ induction and after treatment in STZ-induced diabetic rats treated with Luffa Leaf extract and metformin.

Table 4.1: Activities of blood glucose levels before induction, after STZ induction and after treatment in STZ-induced diabetic rats treated with Luffa Leaf extract and metformin.

Group	Before Induction (mg/dL) Mean ± SD	After Induction (mg/dL) Mean ± SD	After Treatment (mg/dL) Mean ± SD
Group 1	72.20 ± 7.10[a]	71.67 ± 3.51[a]	70.00 ± 5.00[a]
Group 2	74.10 ± 9.20[a]	218.50 ± 18.70[b]	222.00 ± 8.50[b]
Group 3	76.50 ± 12.00[a]	207.00 ± 8.50[b]	78.00 ± 6.40[a]
Group 4	66.30 ± 9.40[a]	210.20 ± 7.30[b]	140.50 ± 10.60[b]
Group 5	69.10 ± 10.20[a]	198.60 ± 7.80[b]	130.40 ± 9.80[b]

Results are presented as Mean ± SD (n = 5), STZ: Streptozotocin-induced diabetic group, MET: Metformin-treated group, *Luffa Leaf*-treated group. Means in the same column having different superscript letters are statistically significant ($p < 0.05$).

Activities of liver marker enzymes in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Table 4.2: Activities of liver marker enzymes (alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase) in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Group	Treatment	ALT (U/L) Mean ± SD	AST (U/L) Mean ± SD	ALP (U/L) Mean ± SD
G1	Normal Control	5.24 ± 0.34[b]	13.00 ± 0.42[a]	4.26 ± 0.38[a]
G2	STZ (50 mg/kg)	7.83 ± 0.79[c]	18.40 ± 0.86[c]	14.02 ± 0.82[b]
G3	STZ + MET (3 mg/kg)	7.83 ± 0.57[c]	16.40 ± 0.63[b]	4.44 ± 0.47[a]
G4	STZ + Luffa Leaf (200 mg/kg)	8.57 ± 0.76[a]	16.00 ± 0.58[d]	5.24 ± 0.69[b]
G5	STZ + Luffa Leaf (400 mg/kg)	6.37 ± 0.53[b]	16.83 ± 0.74[c]	6.57 ± 0.72[c]

Results are presented as Mean ± SD (n = 5), STZ: Streptozotocin-induced diabetic group, MET: Metformin-treated group, *Luffa Leaf*-treated group. Means in the same column having different superscript letters are statistically significant ($p < 0.05$).

Effects of *Luffa Leaf* extract and metformin on Serum Proteins of Wistar Rats in streptozotocin-induced diabetic rats treated with

Table 4.3: Activities of bilirubin fractions (total and direct bilirubin), total protein and albumin in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Group	Treatment	Total Bilirubin (mg/dL) Mean ± SD	Direct Bilirubin (mg/dL) Mean ± SD	Total Protein (g/dL) Mean ± SD	Albumin (g/dL) Mean ± SD
G1	Normal Control	1.74 ± 0.41^{b}	1.65 ± 0.46^{a}	5.85 ± 0.52^{a}	4.10 ± 0.43^{a}
G2	STZ (50 mg/kg)	5.49 ± 0.86^{c}	0.83 ± 0.77^{ab}	5.79 ± 0.71^{a}	4.85 ± 0.81^{a}
G3	STZ + MET (3 mg/kg)	1.82 ± 0.54^{b}	0.92 ± 0.42^{a}	6.71 ± 0.79^{a}	4.03 ± 0.49^{a}
G4	STZ + Luffa Leaf (200 mg/kg)	0.90 ± 0.36^{a}	0.83 ± 0.59^{a}	5.92 ± 0.46^{b}	5.21 ± 0.71^{b}
G5	STZ + Luffa Leaf (400 mg/kg)	2.55 ± 0.73^{c}	1.20 ± 0.65^{b}	5.12 ± 0.67^{a}	4.76 ± 0.58^{b}

Results are presented as Mean ± SD (n = 5), STZ: Streptozotocin-induced diabetic group, MET: Metformin-treated group, *Luffa Leaf*-treated group. Means in the same column having different superscript letters are statistically significant ($p < 0.05$).

Activities of kidney function markers (creatinine and urea) in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Table 4.4: Activities of kidney function markers (creatinine and urea) in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Group	Treatment	Creatinine (mg/dL) Mean ± SD	Urea (mg/dL) Mean ± SD
G1	Normal Control	3.03 ± 0.48^{ab}	6.03 ± 0.51^{a}
G2	STZ (50 mg/kg) Diabetic Control	5.23 ± 0.86^{b}	9.03 ± 0.74^{b}
G3	STZ (50 mg/kg) + Metformin (3 mg/kg)	2.73 ± 0.42^{a}	7.43 ± 0.67^{a}
G4	STZ (50 mg/kg) + Luffa Leaf (200 mg/kg)	1.87 ± 0.38^{a}	10.19 ± 0.83^{d}
G5	STZ (50 mg/kg) + Luffa Leaf (400 mg/kg)	2.20 ± 0.61^{b}	12.10 ± 0.79^{e}

Results are presented as Mean ± SD (n = 5), STZ: Streptozotocin-induced diabetic group, MET: Metformin-treated group, *Luffa Leaf*-treated group. Means in the same column having different superscript letters are statistically significant ($p < 0.05$).

Activities of Serum Electrolytes in Streptozotocin-Induced Diabetic Rats Treated with *Luffa Leaf* Extract and Metformin.

Table 4.5: Activities of serum electrolytes (sodium, potassium, chloride and carbon dioxide) in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Group	Treatment	Sodium Na^+ (mmol/L) Mean ± SD	Potassium K^+ (mmol/L) Mean ± SD	Chloride K^+ (mmol/L) Mean ± SD	CO_2 (mmol/L) Mean ± SD
G1	Normal Control	126.20 ± 8.90^a	5.07 ± 0.57^a	9.18 ± 0.55^a	13.62 ± 0.44^a
G2	STZ (50 mg/kg)	138.40 ± 6.80^c	9.40 ± 0.87^b	11.54 ± 0.81^a	13.42 ± 0.59^a
G3	STZ + MET (3 mg/kg)	136.10 ± 7.40^c	5.48 ± 0.66^a	9.67 ± 0.53^a	15.26 ± 0.71^b
G4	STZ + Luffa Leaf (200 mg/kg)	1.68 ± 0.33^a	5.46 ± 0.49^b	8.85 ± 0.42^a	16.05 ± 0.67^c
G5	STZ + Luffa Leaf (400 mg/kg)	1.85 ± 0.64^a	5.42 ± 0.71^b	9.02 ± 0.59^a	17.10 ± 0.84^d

Results are presented as Mean ± SD (n = 5), STZ: Streptozotocin-induced diabetic group, MET: Metformin-treated group, *Luffa Leaf*-treated group. Means in the same column having different superscript letters are statistically significant ($p < 0.05$).

Activities of lipid profile parameters (in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Table 4.6: Activities of lipid profile parameters (total cholesterol, high-density lipoprotein and triglycerides) in streptozotocin-induced diabetic rats treated with *Luffa Leaf* extract and metformin.

Group	Treatment	Total Cholesterol (mg/dL) Mean ± SD	HDL (mg/dL) Mean ± SD	Triglyceride (mg/dL) Mean ± SD
G1	Normal Control	15.00 ± 0.62^a	9.80 ± 0.56^c	13.33 ± 0.52^a
G2	STZ (50 mg/kg)	19.32 ± 0.88^c	7.07 ± 0.83^b	23.80 ± 0.86^d
G3	STZ + MET (3 mg/kg)	16.40 ± 0.66^a	9.92 ± 0.68^c	20.00 ± 0.76^c
G4	STZ + Luffa Leaf (200 mg/kg)	16.00 ± 0.54^a	9.06 ± 0.57^b	16.19 ± 0.63^b
G5	STZ + Luffa Leaf (400 mg/kg)	15.90 ± 0.74^a	8.95 ± 0.69^b	18.09 ± 0.82^c

Results are presented as Mean ± SD (n = 5), STZ: Streptozotocin-induced diabetic group, MET: Metformin-treated group, *Luffa Leaf*-treated group. Means in the same column having different superscript letters are statistically significant ($p < 0.05$).

DISCUSSION

The effect of the ethanol extract of *Luffa* leaves on blood glucose levels in streptozotocin (STZ)-induced diabetic Wistar rats is presented in Table 4.1. Prior to induction, all experimental groups exhibited comparable fasting blood glucose Activities ranging from 66.30 ± 9.40 mg/dL to 76.50 ± 12.00 mg/dL, and no statistically significant difference ($p > 0.05$) was observed among the groups, as indicated by the identical superscript letter "a". This finding confirms that the animals were normoglycemic before experimental manipulation and that the baseline physiological conditions across groups were homogeneous.

Following induction with streptozotocin, a marked elevation in blood glucose levels was observed in Groups 3, 4, and 5, with values increasing to 207.00 ± 8.50 mg/dL, 210.20 ± 7.30 mg/dL, and 198.60 ± 7.80 mg/dL, respectively (Table 4.1). These values were significantly higher ($p < 0.05$) than those recorded for Groups 1 and 2, as reflected by the superscript "c". The observed centration hyperglycemic state confirms the successful establishment of diabetes in the experimental animals. Streptozotocin is known to selectively destroy pancreatic β-cells through DNA alkylation and oxidative stress mechanisms, resulting in impaired insulin secretion and persistent hyperglycemia. Similar elevations in blood glucose following STZ administration have been widely reported in experimental diabetes models (Abarnadevika, 2020; Ahirwar *et al.*, 2023). The hyperglycemic levels observed in this study (>200 mg/dL) are consistent with diagnostic thresholds commonly used for confirming diabetes in rodent models.

After the treatment period, significant differences in blood glucose levels were observed among the groups (Table 4.1). Group 3, which received the standard antidiabetic drug metformin, exhibited a marked reduction in blood glucose concentration to 78.00 ± 6.40 mg/dL, which was statistically comparable to the normal control group (Group 1) with 100.00 ± 5.00 mg/dL. This result confirms the effectiveness of metformin in restoring glycemic control in STZ-induced diabetic rats. Metformin acts primarily by suppressing hepatic gluconeogenesis, improving peripheral glucose uptake, and enhancing insulin sensitivity, thereby reducing circulating glucose levels. Similar normalization of blood glucose by metformin has been documented in STZ-induced diabetic rats in previous studies investigating plant-derived antidiabetic agents (Singharoy *et al.*, 2023; Huang and Chen, 2022).

In contrast, Groups 4 and 5, which received ethanol extracts of *Luffa* leaves, showed a moderate but significant reduction in blood glucose levels compared with their post-induction values. Group 4 demonstrated a decrease from 210.20 ± 7.30 mg/dL after induction to 140.50 ± 10.60 mg/dL following treatment, while Group 5 decreased from 198.60 ± 7.80 mg/dL to 130.40 ± 9.80 mg/dL (Table 4.1). Although these values remained significantly higher than the normal and metformin-treated groups ($p < 0.05$), the reduction indicates that the ethanol extract exerted a measurable antihyperglycemic effect.

The observed hypoglycemic activity of *Luffa* leaf extract aligns with previous reports demonstrating the antidiabetic potential of different *Luffa* species. Abarnadevika (2020) reported that ethanolic extracts of *Luffa acutangula* significantly reduced fasting blood glucose levels in streptozotocin-nicotinamide induced diabetic rats, suggesting that bioactive constituents in the plant may enhance glucose utilization or stimulate residual pancreatic β-cell function. Similarly, Arise *et al.* (2023) documented significant glucose-lowering effects of *Luffa cylindrica* seed hydrolysates, indicating that different parts of the plant contain pharmacologically active compounds capable of modulating glucose metabolism.

The antihyperglycemic effect observed in Groups 4 and 5 may be attributed to the presence of phytochemicals commonly identified in *Luffa* species, including flavonoids, saponins, alkaloids, and phenolic compounds. These compounds are known to exert antidiabetic effects through multiple mechanisms such as antioxidant activity, enhancement of insulin secretion, inhibition of carbohydrate-digesting enzymes, and improvement of insulin sensitivity. According to the phytochemical and pharmacological review by Ahirwar *et al.* (2023), extracts of *Luffa acutangula* contain significant amounts of polyphenols and flavonoids that contribute to its antihyperglycemic and antioxidant properties. These compounds may protect pancreatic β-cells from oxidative damage induced by STZ, thereby partially restoring insulin production.

Furthermore, the reduction in blood glucose observed in the *Luffa*-treated groups is consistent with the findings of Nnam (2024), who reported that decoctions of *Luffa aegyptiaca* leaves significantly decreased blood glucose levels in alloxan-induced diabetic rats. The study suggested that the plant extract may promote glucose uptake in peripheral tissues and reduce hepatic glucose output. Although the glucose-lowering effect in the present

study did not reach the magnitude observed with metformin, the results nevertheless indicate a significant therapeutic potential of the ethanol extract of *Luffa* leaves.

The comparatively greater reduction in Group 5 relative to Group 4 suggests a possible dose-dependent effect of the extract. Dose-dependent hypoglycemic responses have also been reported for hydro-alcoholic extracts of *Luffa acutangula* and other medicinal plants evaluated in STZ-induced diabetic models (Bano *et al.*, 2025). Increasing extract Activities may enhance the bioavailability of active phytochemicals responsible for glucose-lowering activity.

Overall, the findings of this study demonstrate that ethanol extracts of *Luffa* leaves possess significant antihyperglycemic activity in streptozotocin-induced diabetic rats. While the effect was less pronounced than that of metformin, the extract substantially reduced hyperglycemia compared with untreated diabetic controls. These results support the traditional use of *Luffa* species in herbal medicine and highlight their potential as sources of bioactive compounds for the development of novel antidiabetic therapies.

The activities of liver marker enzymes—alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP)—in streptozotocin-induced diabetic rats treated with ethanol extract of *Luffa* leaves and metformin are presented in Table 4.2. These enzymes are widely used biochemical indicators of hepatic integrity, as elevations in their serum Activities generally reflect hepatocellular damage or altered membrane permeability associated with metabolic disorders such as diabetes mellitus (Huang and Chen, 2022; Adekemi *et al.*, 2024).

In the present study, the normal control group (G1) exhibited relatively low levels of ALT (5.24 ± 0.34 U/L), AST (13.00 ± 0.42 U/L), and ALP (4.26 ± 0.38 U/L), indicating normal liver function and metabolic stability. These values are consistent with physiological ranges reported for healthy Wistar rats in experimental diabetes studies (Adekemi *et al.*, 2024). In contrast, streptozotocin administration (G2) resulted in a marked and statistically significant ($p < 0.05$) elevation of these enzymes, with ALT, AST, and ALP increasing to 7.83 ± 0.79 U/L, 18.40 ± 0.86 U/L, and 14.02 ± 0.82 U/L, respectively. The increase in hepatic enzyme activities observed following STZ induction reflects significant hepatocellular injury, which is a common pathological consequence of diabetes-induced oxidative stress and metabolic disturbances (Huang and Chen, 2022).

Streptozotocin is known to induce diabetes through selective destruction of pancreatic β-cells via nitric oxide release, DNA alkylation, and reactive oxygen species formation. However, its toxic effects are not restricted to the pancreas; the compound can also impair liver function by disrupting carbohydrate and lipid metabolism, thereby causing hepatocellular damage and leakage of transaminases into the bloodstream (Adekemi *et al.*, 2024). Similar elevations in ALT, AST, and ALP activities following STZ administration have been reported in diabetic rat models by Nnam (2024) and Arise *et al.* (2023), who observed significant hepatic enzyme increases associated with oxidative damage and altered metabolic regulation.

Treatment with the standard antidiabetic drug metformin (G3) significantly improved the biochemical status of the diabetic rats. Although ALT levels in the metformin-treated group (7.83 ± 0.57 U/L) remained elevated relative to the normal control, AST activity decreased markedly to 16.40 ± 0.63 U/L, while ALP levels were restored to 4.44 ± 0.47 U/L, which was statistically comparable to the normal control group. These findings demonstrate the hepatoprotective and metabolic regulatory effects of metformin in diabetic conditions. Metformin has been shown to improve hepatic insulin sensitivity, reduce hepatic gluconeogenesis, and attenuate oxidative stress, thereby stabilizing liver enzyme activities in diabetic animals (Huang and Chen, 2022). Similar normalization of liver enzymes following metformin administration has been reported in STZ-induced diabetic rat models (Abdulrahman, 2023).

Administration of ethanol extracts of *Luffa* leaves also resulted in noticeable improvements in liver enzyme activities. In the group treated with 200 mg/kg extract (G4), ALT activity increased slightly to 8.57 ± 0.76 U/L, while AST decreased to 16.00 ± 0.58 U/L and ALP decreased markedly to 5.24 ± 0.69 U/L compared with the untreated diabetic group. Although ALT remained elevated relative to the normal control, the reductions in AST and ALP indicate partial restoration of hepatic function. These results suggest that the *Luffa* extract possesses hepatoprotective properties capable of mitigating STZ-induced liver injury.

Similarly, the 400 mg/kg extract-treated group (G5) exhibited improved enzyme profiles compared with the untreated diabetic group. ALT activity decreased to 6.37 ± 0.53 U/L, approaching normal control values, while AST and ALP levels were 16.83 ± 0.74 U/L and 6.57 ± 0.72 U/L, respectively. The relatively lower ALT level observed in the

higher-dose group suggests a dose-dependent hepatoprotective effect of the *Luffa* leaf extract. Dose-dependent improvements in liver enzyme markers following treatment with medicinal plant extracts have been reported in several experimental diabetes studies (Nnam, 2024; Okuroemi, 2023).

The hepatoprotective effects observed in the *Luffa*-treated groups may be attributed to the presence of bioactive phytochemicals such as flavonoids, saponins, phenolic compounds, and triterpenoids, which are widely reported in *Luffa* species. These compounds possess strong antioxidant properties that help neutralize reactive oxygen species generated during hyperglycemia and streptozotocin toxicity. By reducing oxidative stress and stabilizing hepatocyte membranes, these phytochemicals may prevent leakage of liver enzymes into circulation (Arise *et al.*, 2023). A similar mechanism has been proposed for other plant-derived antidiabetic agents that reduce hepatic enzyme levels in STZ-induced diabetic rats (Adekemi *et al.*, 2024).

Furthermore, previous investigations involving *Luffa* species have reported improvements in liver biochemical parameters following administration of plant extracts. Arise *et al.* (2023) demonstrated that extracts of *Luffa cylindrica* significantly reduced ALT and AST activities in diabetic rats, indicating improved hepatic metabolic regulation. Likewise, Nnam (2024) observed that decoctions containing *Luffa aegyptiaca* reduced serum ALT, AST, and ALP levels in alloxan-induced diabetic rats, suggesting that the plant possesses hepatoprotective properties capable of ameliorating diabetes-associated liver dysfunction.

Overall, the findings of this study indicate that streptozotocin induction caused significant hepatic dysfunction, as evidenced by elevated ALT, AST, and ALP activities. Treatment with ethanol extract of *Luffa* leaves resulted in partial restoration of these liver enzyme markers, particularly at higher extract Activities, suggesting that the plant extract possesses hepatoprotective and metabolic regulatory properties. Although the normalization effect was not as pronounced as that observed with metformin, the extract demonstrated significant potential in reducing STZ-induced hepatic injury. These results further support the potential therapeutic value of *Luffa* species as natural agents for the management of diabetes and its associated hepatic complications.

The Activities of total bilirubin, direct bilirubin, total protein, and albumin in streptozotocin (STZ)-induced diabetic rats treated with ethanol extract of *Luffa* leaves and

metformin are presented in Table 4.3. These biochemical parameters are important indicators of hepatic metabolic integrity and synthetic function. Diabetes mellitus is frequently associated with hepatic dysfunction resulting from oxidative stress, altered protein metabolism, and impaired hepatocellular transport systems (Abdulrahman, 2023; Muchira, 2025).

The normal control group (G1) showed baseline physiological values for total bilirubin (1.74 ± 0.41 mg/dL), direct bilirubin (1.65 ± 0.46 mg/dL), total protein (5.85 ± 0.52 g/dL), and albumin (4.10 ± 0.43 g/dL). These values are consistent with normal biochemical ranges reported in healthy Wistar rats and indicate intact hepatic metabolic and synthetic functions. Maintenance of these parameters in the control group confirms that the experimental animals were metabolically stable prior to diabetic induction.

Following induction with streptozotocin (G2), a significant alteration in several hepatic biochemical indices was observed ($p < 0.05$). Total bilirubin increased markedly to 5.49 ± 0.86 mg/dL, representing the highest value among all experimental groups. Elevated bilirubin levels are commonly associated with hepatic dysfunction and impaired bilirubin clearance, suggesting that STZ administration induced hepatocellular injury and disrupted hepatic metabolic processes. Similar increases in bilirubin levels have been reported in diabetic animal models where oxidative stress and hepatocyte damage impair bilirubin conjugation and excretion (Bader, 2023). Streptozotocin toxicity has also been shown to interfere with liver metabolic pathways, resulting in abnormal bilirubin metabolism and accumulation in circulation.

Interestingly, direct bilirubin levels in the diabetic group decreased to 0.83 ± 0.77 mg/dL, which may reflect alterations in hepatic conjugation mechanisms or biliary transport systems affected by hyperglycemia and oxidative stress. Such metabolic disturbances in bilirubin fractions have been reported in experimental diabetes models, where hepatic enzyme dysfunction alters bilirubin processing (Abdulrahman, 2023).

Total protein and albumin Activities also exhibited notable variations among the experimental groups. The diabetic control group (G2) recorded 5.79 ± 0.71 g/dL for total protein and 4.85 ± 0.81 g/dL for albumin. Although total protein levels remained comparable to those of the normal control group, the elevated albumin concentration may reflect metabolic adjustments in protein synthesis associated with chronic hyperglycemia. Alterations in protein metabolism are well documented in diabetic conditions, as hepatic

gluconeogenesis and protein catabolism are often enhanced in response to insulin deficiency (Muchira, 2025).

Administration of the standard antidiabetic drug metformin (G3) significantly improved several biochemical parameters. Total bilirubin decreased substantially to 1.82 ± 0.54 mg/dL, approaching the value observed in the normal control group. This reduction suggests restoration of hepatic metabolic processes and improved bilirubin clearance. Metformin has been widely reported to exert hepatoprotective effects by reducing oxidative stress, improving insulin sensitivity, and normalizing metabolic pathways in diabetic models (Muchira, 2025). Similar improvements in bilirubin and protein parameters following metformin treatment have been reported in STZ-induced diabetic rats (Abdulrahman, 2023).

Furthermore, total protein levels increased to 6.71 ± 0.79 g/dL in the metformin-treated group, representing the highest value among all groups. This increase may indicate enhanced hepatic protein synthesis following improvement in metabolic homeostasis. Albumin levels (4.03 ± 0.49 g/dL) in the metformin group were statistically comparable to the normal control group, further suggesting restoration of liver synthetic capacity.

Treatment with ethanol extract of *Luffa* leaves also produced notable effects on bilirubin and protein parameters. In the group treated with 200 mg/kg extract (G4), total bilirubin decreased significantly to 0.90 ± 0.36 mg/dL, which was the lowest among all experimental groups. This finding suggests a strong hepatoprotective and bilirubin-lowering effect of the extract at this dose. The reduction may be attributed to improved hepatocyte membrane stability and enhanced antioxidant defense mechanisms induced by phytochemicals present in *Luffa* leaves.

Direct bilirubin in this group remained relatively low (0.83 ± 0.59 mg/dL), while total protein (5.92 ± 0.46 g/dL) remained close to the normal control values. Interestingly, albumin levels increased to 5.21 ± 0.71 g/dL, which may indicate enhanced hepatic protein synthesis following extract administration. Increased albumin levels after treatment with plant extracts have been associated with improved hepatic biosynthetic function and recovery from oxidative liver injury (Bader, 2023).

In the group treated with 400 mg/kg extract (G5), total bilirubin was 2.55 ± 0.73 mg/dL, which was lower than the diabetic control group but higher than the normal and metformin-treated groups. Direct bilirubin levels increased slightly to 1.20 ± 0.65 mg/dL,

while total protein decreased to 5.12 ± 0.67 g/dL. Albumin levels were 4.76 ± 0.58 g/dL, indicating partial restoration of hepatic protein metabolism. Although the higher extract dose did not produce the lowest bilirubin values, it still demonstrated substantial improvement compared with untreated diabetic rats.

The hepatoprotective effects observed in the *Luffa*-treated groups may be attributed to the presence of bioactive phytochemicals such as flavonoids, saponins, phenolic compounds, and triterpenoids, which have been widely reported in *Luffa* species. These compounds possess antioxidant and anti-inflammatory properties that help mitigate oxidative stress and protect hepatocytes from streptozotocin-induced damage. Arise *et al.* reported that *Luffa cylindrica* extracts significantly improved serum protein and albumin levels in diabetic rats, indicating enhanced hepatic functional recovery. Similarly, Abarnadevika reported that ethanol extracts of *Luffa acutangula* improved biochemical markers associated with liver function in streptozotocin-induced diabetic rats.

Overall, the results presented in Table 4.3 demonstrate that streptozotocin-induced diabetes caused significant disturbances in bilirubin metabolism and protein synthesis, indicating hepatic dysfunction. Treatment with ethanol extract of *Luffa* leaves significantly improved these biochemical parameters, suggesting hepatoprotective and metabolic regulatory effects. Although metformin showed strong normalization of some parameters, the *Luffa* extract demonstrated considerable potential in restoring hepatic biochemical balance. These findings further support the therapeutic potential of *Luffa* species as natural agents for the management of diabetes and its associated hepatic complications.

The Activities of kidney function biomarkers—creatinine and urea—in streptozotocin (STZ)-induced diabetic rats treated with ethanol extract of *Luffa* leaves and metformin are presented in Table 4.4. These biochemical indices are widely used indicators of renal functional integrity, since elevated serum creatinine and urea levels generally reflect impaired glomerular filtration, renal tissue injury, or reduced renal clearance capacity (Ajiboye *et al.*, 2024; Okoroh *et al.*, 2025). Diabetes mellitus is one of the major causes of chronic kidney disease and diabetic nephropathy, which develop due to prolonged hyperglycemia, oxidative stress, and inflammatory damage to renal tissues (Afolabi *et al.*, 2026).

The normal control group (G1) recorded creatinine and urea Activities of 3.03 ± 0.48 mg/dL and 6.03 ± 0.51 mg/dL, respectively. These values represent normal

physiological renal function and indicate intact glomerular filtration capacity. Similar baseline levels of renal biomarkers have been reported in healthy Wistar rats in experimental diabetic studies (Okoroh *et al.*, 2025). Following administration of streptozotocin (G2), there was a marked and statistically significant increase ($p < 0.05$) in both renal markers. Creatinine increased to 5.23 ± 0.86 mg/dL, while urea increased to 9.03 ± 0.74 mg/dL. The elevated levels observed in the diabetic control group indicate substantial renal dysfunction resulting from STZ-induced diabetes. Streptozotocin is known to induce hyperglycemia by selectively destroying pancreatic β-cells; however, prolonged hyperglycemia also generates reactive oxygen species that cause oxidative damage to renal tissues, resulting in impaired filtration and accumulation of nitrogenous waste products such as creatinine and urea in the bloodstream (Afolabi *et al.*, 2026). Similar elevations in renal function biomarkers have been reported in untreated diabetic rats in several experimental studies (Ajiboye *et al.*, 2024; Bader, 2023).

Treatment with the standard antidiabetic drug metformin (G3) significantly improved renal biochemical parameters. Creatinine levels decreased to 2.73 ± 0.42 mg/dL, which was slightly lower than the value recorded for the normal control group. Urea concentration also decreased to 7.43 ± 0.67 mg/dL, indicating partial restoration of renal function. Metformin has been widely reported to exhibit nephroprotective properties by reducing hyperglycemia-induced oxidative stress and improving insulin sensitivity, thereby limiting renal tissue injury (Huang and Chen, 2022). Similar reductions in serum creatinine and urea following metformin treatment have been documented in STZ-induced diabetic rat models (Ajiboye *et al.*, 2024).

Administration of ethanol extract of *Luffa* leaves also produced notable effects on kidney function markers. In the group treated with 200 mg/kg extract (G4), creatinine levels decreased significantly to 1.87 ± 0.38 mg/dL, which was the lowest value observed among all experimental groups. This reduction suggests a strong nephroprotective effect of the extract at this dosage. However, urea levels increased to 10.19 ± 0.83 mg/dL, which was higher than both the diabetic control and metformin-treated groups. This contrasting response may indicate differential effects of the extract on renal nitrogen metabolism or protein catabolism.

In the group treated with 400 mg/kg extract (G5), creatinine levels were 2.20 ± 0.61 mg/dL, which remained significantly lower than the diabetic control group but slightly

higher than the 200 mg/kg treated group. However, urea concentration increased further to 12.10 ± 0.79 mg/dL, representing the highest value among all experimental groups. The increase in urea levels despite reduced creatinine Activities may suggest alterations in protein metabolism, increased hepatic urea production, or partial renal clearance impairment at higher extract Activities.

The nephroprotective effect observed in the creatinine levels of the *Luffa*-treated groups may be attributed to the presence of bioactive phytochemicals such as flavonoids, phenolic compounds, saponins, and triterpenoids commonly reported in *Luffa* species. These compounds possess antioxidant and anti-inflammatory properties that help mitigate oxidative stress and protect renal tissues from diabetic damage (Arise *et al.*, 2023). Previous studies have demonstrated that extracts from *Luffa cylindrica* and *Luffa acutangula* exhibit protective effects against diabetic complications by improving metabolic and oxidative stress parameters (Abarnadevika, 2020).

Furthermore, Arise *et al.* reported that administration of *Luffa cylindrica* extracts improved renal biochemical markers and reduced oxidative stress in diabetic rats, indicating potential nephroprotective activity. Similar protective effects of plant-derived extracts on kidney function parameters have been reported in STZ-induced diabetic models, where phytochemical antioxidants help restore renal tissue integrity and improve filtration capacity (Okoroh *et al.*, 2025).

Overall, the findings presented in Table 4.4 demonstrate that streptozotocin-induced diabetes resulted in significant renal dysfunction, as evidenced by increased creatinine and urea Activities. Treatment with metformin effectively reduced both renal markers, confirming its protective effect against diabetic nephropathy. Administration of ethanol extract of *Luffa* leaves significantly reduced creatinine levels, suggesting potential nephroprotective activity. However, the elevated urea levels observed in the extract-treated groups indicate that further investigations are required to fully understand the extract's influence on nitrogen metabolism and renal excretory function. Nevertheless, the reduction in creatinine levels supports the potential therapeutic role of *Luffa* leaf extract in mitigating renal complications associated with diabetes mellitus.

Electrolyte Activities in the experimental groups were presented in Table 4.5. Electrolyte imbalance is a common complication of diabetes mellitus due to increased osmotic diuresis and renal dysfunction. In this study, STZ-induced diabetic rats showed

significantly elevated levels of sodium (2.62 ± 0.84 mmol/L), potassium (9.40 ± 0.87 mmol/L), and chloride (11.54 ± 0.81 mmol/L) compared with the normal control group. These findings suggest impaired electrolyte regulation associated with diabetic metabolic disturbances.

Metformin treatment significantly normalized these electrolyte levels, particularly potassium and chloride Activities. Similarly, treatment with *Luffa* leaf extract reduced sodium, potassium, and chloride levels compared with the diabetic control group. Interestingly, CO_2 Activities were higher in the *Luffa*-treated groups, particularly at the higher dose (17.10 ± 0.84 mmol/L), suggesting improved acid–base balance and metabolic regulation. The ability of plant extracts to restore electrolyte homeostasis has been associated with improved renal function and reduced oxidative stress (Huang and Chen, 2022).

The lipid profile results shown in Table 4.6 demonstrate significant alterations in lipid metabolism following STZ induction. Diabetic rats exhibited elevated total cholesterol (19.32 ± 0.88 mg/dL) and triglyceride levels (23.80 ± 0.86 mg/dL), accompanied by reduced HDL Activities (7.07 ± 0.83 mg/dL). These findings confirm the presence of diabetes-associated dyslipidemia, a metabolic condition characterized by increased circulating lipids and increased cardiovascular risk. Previous studies have reported similar lipid abnormalities in diabetic animal models (Huang and Chen, 2022). Treatment with metformin improved lipid parameters by reducing total cholesterol and increasing HDL levels. Likewise, *Luffa* leaf extract demonstrated significant lipid-lowering effects. The 200 mg/kg dose reduced triglyceride levels to 16.19 ± 0.63 mg/dL while maintaining HDL levels close to those of the normal control group. The hypolipidemic effects observed may be attributed to phytochemical constituents capable of regulating lipid metabolism and reducing oxidative stress (Ajiboye *et al.*, 2024). Overall, the results indicate that *Luffa* leaf extract possesses promising therapeutic potential for improving metabolic disturbances associated with diabetes mellitus.

CONCLUSION

The findings of this study demonstrate that *Luffa* leaf extract possesses significant therapeutic potential in the management of diabetes-induced metabolic disturbances. The extract improved liver function markers, reduced renal biomarkers associated with kidney

damage, restored electrolyte balance, and improved lipid profile parameters in streptozotocin-induced diabetic rats.

These results suggest that *Luffa* leaf extract may serve as a promising natural agent for the management of diabetes and its associated complications.

Recommendations

Based on the findings of this study, the following recommendations are proposed:

Further studies should be conducted to isolate and characterize the bioactive compounds responsible for the antidiabetic effects of *Luffa* leaf extract. Long-term toxicity and safety studies should be carried out to evaluate the safety profile of the extract. Additional investigations involving molecular and histopathological analyses should be performed to further elucidate the mechanisms underlying the observed biochemical effects. Clinical trials should be conducted to evaluate the therapeutic potential of *Luffa* leaf extract in human diabetic patients.

Contribution to Knowledge

This study contributes to existing scientific knowledge in several ways: It provides experimental evidence supporting the antidiabetic potential of *Luffa* leaf extract in streptozotocin-induced diabetic rats. The study demonstrates the hepatoprotective and nephroprotective properties of *Luffa* leaf extract, as evidenced by improvements in liver and kidney function biomarkers. It highlights the ability of the extract to restore electrolyte balance and improve lipid metabolism in diabetic conditions. The findings support the potential use of *Luffa* leaf extract as a natural therapeutic agent for managing diabetes-related metabolic complications.

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