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Research Article

In Vivo Evaluation of Antidiabetic Activity with CO- Administration of Ipomoea Quamoclit and Metformin

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ABSTRACT

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycaemia, which may lead to metabolic disturbances and tissue injury. The present study evaluated the antidiabetic potential of the hydroethanolic leaf extract of *Ipomoea quamoclit* L. (HAIQ), administered alone and in combination with metformin, in streptozotocin (STZ)-induced diabetic and dexamethasone-induced insulin-resistant Wistar rats. The extract was subjected to preliminary phytochemical screening, gas chromatography–mass spectrometry (GC–MS) analysis, and an in-vitro α-amylase inhibition assay. The extraction yielded 7 g of extract from 80 g of powdered leaves (8.75% w/w). Phytochemical screening revealed the presence of alkaloids, glycosides, steroids, tannins, flavonoids, carbohydrates, and amino acids, while GC–MS analysis identified 63 chromatographic peaks, including several tentatively identified phytoconstituents. HAIQ demonstrated concentration-dependent α-amylase inhibitory activity, with inhibition increasing from 8.62% at 20 µg/mL to 66.09% at 100 µg/mL and an IC_{50} value of 78.85 µg/mL, compared with 50.80 µg/mL for acarbose. In the STZ-induced diabetic model, 28-day oral treatment with HAIQ (400 mg/kg) reduced fasting blood glucose to 145 mg/dL, compared with 212 mg/dL in diabetic control rats. The combination of HAIQ (400 mg/kg) and metformin (50 mg/kg) produced a greater reduction, with fasting blood glucose reaching 98 mg/dL, compared with 126 mg/dL in the metformin-treated group. In the dexamethasone-induced model, the combination treatment reduced blood glucose to 108 mg/dL, compared with 216 mg/dL in the model control group. Improvements in serum AST and ALT levels and qualitative pancreatic histological findings were also observed in the treated groups. These findings suggest that *Ipomoea quamoclit L.* leaf extract possesses promising in-vitro α-amylase inhibitory and in-vivo antidiabetic potential, with the combination of HAIQ and

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metformin showing greater glucose-lowering effects than either treatment alone under the conditions studied. Further investigations are warranted to establish its mechanism of action, safety, and potential clinical relevance..

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from impaired insulin secretion, reduced insulin action, or both. It affects carbohydrate, lipid, and protein metabolism and represents a major global health concern because of its increasing prevalence and association with long-term complications involving multiple organ systems. The major forms of diabetes include type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM), and gestational diabetes mellitus (GDM). T1DM primarily results from autoimmune destruction of pancreatic β-cells, leading to absolute insulin deficiency, whereas T2DM is characterized by insulin resistance and progressive β-cell dysfunction. GDM is defined as diabetes diagnosed during pregnancy that was not clearly overt diabetes before gestation and is associated with an increased risk of subsequent T2DM.[1]

The pathophysiology of diabetes involves complex interactions among genetic susceptibility, metabolic dysfunction, inflammation, and impaired glucose regulation. In T2DM, reduced insulin sensitivity in skeletal muscle, adipose tissue, and the liver contributes to impaired peripheral glucose utilization and increased hepatic glucose production. Progressive pancreatic β-cell dysfunction further compromises insulin secretion, resulting in sustained hyperglycemia. Persistent hyperglycemia contributes to microvascular complications, including diabetic retinopathy, nephropathy, and neuropathy, as well as macrovascular complications such as cardiovascular and cerebrovascular diseases and peripheral arterial disease. These complications substantially increase disease burden and emphasize the importance of effective glycemic management.

Oxidative stress is implicated in the development and progression of diabetes and its complications. An imbalance between reactive oxygen species (ROS) production and antioxidant defenses can promote the oxidation of cellular lipids, proteins, and nucleic acids. Endogenous antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px), contribute to maintaining cellular redox homeostasis. Excessive oxidative stress may impair pancreatic β-cell function and promote vascular and tissue injury. Consequently, the potential effects of antidiabetic agents on glucose regulation and oxidative stress are important considerations in preclinical investigations.[2]

The pharmacological management of diabetes includes lifestyle modification and various glucose-lowering agents, such as metformin, insulin, sulfonylureas, thiazolidinediones, α-glucosidase inhibitors, dipeptidyl peptidase-4 inhibitors, glucagon-like peptide-1 receptor agonists, and sodium–glucose cotransporter-2 inhibitors. Metformin primarily reduces hepatic glucose production and improves insulin sensitivity. Although established treatments play a central role in glycemic control, treatment selection and long-term management may be influenced by adverse effects, contraindications, tolerability, and individual patient requirements. These considerations have encouraged research into additional therapeutic candidates, including medicinal plants and their bioactive constituents.[3]

Plant-derived compounds, including flavonoids, alkaloids, terpenoids, phenolic compounds, and glycosides, have been investigated for their potential roles in glucose metabolism, insulin secretion, insulin sensitivity, and oxidative stress modulation. The presence of multiple phytoconstituents may contribute to diverse biological activities; however, the efficacy, safety, active constituents, and mechanisms of action of plant extracts require systematic evaluation through appropriate experimental models.[4]

Ipomoea quamoclit L. (IQ), commonly known as cypress vine or cardinal creeper, is an annual climbing plant belonging to the family Convolvulaceae. The plant has attracted scientific interest because of its reported traditional medicinal uses and phytochemical diversity. Preliminary phytochemical investigations of I. quamoclit have reported the presence of various classes of constituents, including flavonoids, tannins, alkaloids, glycosides, and other phytochemicals. These compounds may contribute to different biological activities; however, their specific pharmacological roles depend on the plant material and extract investigated.

The antidiabetic potential of the hydroethanolic leaf extract of I. quamoclit, particularly its effects on glucose regulation and carbohydrate-digesting enzymes, requires further systematic evaluation..[5]

Therefor, the present study evaluated the antidiabetic and insulin-sensitizing effects of hydroethanolic *I. quamoclit* leaf extract (HAIQ) in streptozotocin (STZ)-induced diabetic and dexamethasone-induced insulin-resistant Wistar rats. The study also investigated the effects of combining HAIQ with metformin, assessed the in vitro α-amylase inhibitory activity of the extract, characterized its phytochemical constituents using preliminary screening and gas chromatography–mass spectrometry (GC–MS), and examined biochemical and histopathological changes associated with treatment. These investigations were undertaken to characterize the potential antidiabetic activity of HAIQ and provide experimental evidence to guide further pharmacological research.[6]

MATERIALS AND METHODS

2.1. Chemicals and Reagents

Chemicals and Reagents

Streptozotocin (STZ) was procured from Sisco Research Laboratories Pvt. Ltd., India. Diagnostic kits used for the estimation of serum aspartate aminotransferase (AST/SGOT) and alanine aminotransferase (ALT/SGPT) were obtained from Span Diagnostics, India. All other chemicals and reagents used in the study were of analytical reagent grade. dexamethasone sodium phosphate, carboxymethyl cellulose (CMC), metformin, acarbose, porcine pancreatic α-amylase, soluble starch, and other analytical reagents were obtained from the manufacturers and suppliers specified in the study records. Blood glucose levels were measured using a Dr. Morepen® digital glucometer.[7,8]

2.2. Plant Collection and Extract Preparation

Leaves of *Ipomoea quamoclit L*. were collected from the Western Ghats of Maharashtra, India, and authenticated by Dr. K. H. Patil, Associate Professor, Sadguru Gadage Maharaj College, Karad. The leaves were shade-dried, powdered, and 80 g of the powder was extracted by cold maceration using an ethanol–water mixture (3:5, v/v) for 3–4 days. The filtrate was concentrated on a water bath to obtain 7 g of semisolid extract , which was stored in an airtight container under refrigeration until further use.[9]

2.3. Preliminary Phytochemical Screening

The hydroalcoholic extract of *Ipomoea quamoclit L*. leaves was subjected to preliminary qualitative phytochemical screening using standard chemical tests to detect alkaloids, glycosides, steroids, tannins, flavonoids, carbohydrates, and amino acids.[10,11]

2.4. Gas Chromatography–Mass Spectrometry (GC–MS) Analysis

The hydroethanolic extract was analysed using a Shimadzu QP-2010 Plus GC–MS system equipped with a capillary column (30 m × 0.25 mm × 0.25 µm). Helium was used as the carrier gas at a flow rate of 1.0 mL/min. The oven temperature was maintained at 60°C for 2 min and then increased to 280°C at a rate of 5°C/min. The injector and detector temperatures were set at 250°C and 280°C, respectively. The extract (10 mg/mL in methanol; 1 µL) was injected in split mode (1:10). Mass spectra were recorded over an *m/z* range of 40–700. Compounds were tentatively identified by comparing their mass spectra with those in the NIST library.[11, 12]

2.5. In Vitro Studies

2.5.1. α-Amylase Inhibition Assay

The α-amylase inhibition assay was performed using the Bernfeld method. The extract at concentrations of 20–100 µg/mL was mixed with α-amylase in 0.1 M phosphate buffer (pH 6.9) and incubated at 25°C for 10 min. Starch solution (1% w/v) was then added, followed by incubation for another 10 min. Dinitrosalicylic acid reagent was added, and the mixture was heated in a boiling water bath for 10 min and subsequently cooled. Absorbance was measured at 540 nm using a spectrophotometer.

The percentage inhibition of α-amylase activity was calculated using the following equation:

$$\text{Inhibition (\%)} = \frac{A_c - A_s}{A_c} \times 100$$

where (Ac) represents the absorbance of the control and (As) represents the absorbance of the sample.[12, 13]

2.6. Experimental Animals and Ethical Approval

Wistar albino rats (9–12 weeks old, weighing 150–250 g) were housed under standard laboratory conditions with free access to a standard pellet diet and water *ad libitum*. The animals were acclimatized for one week before the experiment. All experimental procedures were approved by the Institutional Animal Ethics Committee of Shree Santkrupa College of Pharmacy, Ghogaon, Maharashtra, India (Approval No. SNTK/ IAEC/2025/15), in accordance with CPCSEA guidelines.[14,15]

2.7. In Vivo Studies

2.7.1. Acute Oral Toxicity Study

The hydroalcoholic leaf extract of *Ipomoea quamoclit L*. (HAIQ) was evaluated for acute oral toxicity in Swiss albino mice. Groups of six animals received escalating oral doses of 100, 200, 400, 800, 1000, 2000, and 3000 mg/kg body weight, with the extract suspended in 0.5% (w/v) sodium carboxymethyl cellulose (CMC). The animals were observed for behavioural changes during the first 4 h and monitored for mortality up to 72 h after administration. No mortality was observed at any tested dose. Based on these preliminary findings, a dose of 400 mg/kg body weight was selected for subsequent antidiabetic studies.[16]

2.7.2. Induction of Diabetes by Streptozotocin :

Experimental diabetes was induced in overnight-fasted Wistar albino rats by a single intraperitoneal injection of streptozotocin (STZ, 55 mg/kg body weight), freshly dissolved in ice-cold 0.1 M citrate buffer (pH 4.5). STZ induces pancreatic β-cell damage, resulting in impaired insulin secretion and elevated blood glucose levels. After 48 h of STZ administration, blood samples were collected from the tail vein, and fasting blood glucose levels were measured using a digital glucometer. Rats exhibiting fasting blood glucose levels above 200 mg/dL were considered diabetic and included in the study. The selected The animals were assigned to five experimental groups: normal control, diabetic control, metformin-treated (50 mg/kg), HAIQ-treated (400 mg/kg), and combination-treated (metformin 50 mg/kg + HAIQ 400 mg/kg) and received the designated treatments orally for 28 days. Fasting blood glucose levels and body weight were monitored weekly on days 0, 7, 14, 21, and 28 to evaluate the antidiabetic effects of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ), metformin, and their combination.[17, 18]

2.7.3 : Dexamethasone-Induced Insulin Resistance

Insulin resistance was induced in Wistar albino rats by intraperitoneal administration of dexamethasone (8 mg/kg body weight) once daily for six consecutive days. Following induction, the animals were fasted overnight, and fasting blood glucose levels were measured using a digital glucometer. The animals were allocated to five experimental groups: normal control, insulin-resistant control, metformin-treated (50 mg/ kg), HAIQ-treated (400 mg/kg), and combination-treated (metformin 50 mg/kg + HAIQ 400 mg/kg). Treatments were administered orally for 12 days, while body weight and general health were monitored throughout the study. On day 12, following overnight fasting, blood samples were collected for biochemical analysis, including serum AST/SGOT and ALT/SGPT. The animals were subsequently euthanized, and pancreatic tissues were collected and preserved for histopathological examination.[19, 20]

2.8. Biochemical Estimations and Body Weight Assessment

2.8.1. STZ-Induced Diabetes Model

Blood Collection and Biochemical Estimation: On the 30th day of the experimental study, following the final blood glucose assessment, blood samples were collected from the experimental animals by cardiac puncture under appropriate aseptic conditions. The collected blood was allowed to clot and then centrifuged to separate the serum, which was used for biochemical analysis. Serum aspartate aminotransferase (AST/SGOT) and alanine aminotransferase (ALT/SGPT) levels were estimated using standard diagnostic assay kits obtained from Span Diagnostics, India, to evaluate possible changes in liver function associated with STZ-induced diabetes and the effects of the administered treatments.[21,22]

Body Weight Measurement: Body weights of all experimental animals were recorded weekly throughout the 28-day treatment period using a digital animal weighing balance. Weekly body weight monitoring was performed to assess changes in body mass associated with STZ-induced diabetes and to evaluate the effects of HAIQ, metformin, and their combination during the treatment period.23

2.8.2. Dexamethasone-Induced Insulin Resistance Model

Blood Collection and Biochemical Estimation: On the 12th day of the experimental study, following overnight fasting and the final blood glucose assessment, blood samples were collected by cardiac puncture under aseptic conditions. The collected blood samples were processed to obtain serum for biochemical analysis. Serum AST/SGOT and ALT/SGPT levels were determined using standard diagnostic assay kits obtained from Span Diagnostics, India, to assess biochemical changes associated with dexamethasone administration and to evaluate the effects of HAIQ, metformin, and their combination on these parameters.[24, 25]

Body Weight Measurement: Body weights of the experimental animals were recorded daily throughout the experimental period using a digital animal weighing balance. Daily monitoring was performed to observe changes in body weight during dexamethasone administration and subsequent treatment, thereby helping to assess treatment-associated changes in the animals' general physiological condition.[26,27]

2.9. Histopathological Examination

After blood collection, the animals were sacrificed by cervical dislocation, and the pancreas were instantly dissected out, excised and rinsed in icecold saline solution.[28] A portion of liver and kidney were fixed in 10% neutral formalin fixative solution, were fixed in 10% formalin ,dehydrated in alcohol and then embedded in paraffin. Microtome sections of 4–5 µm thickness were made by using a rotary microtome. The sections were stained with haematoxylin–eosin (H&E) dye to observe histopathological changes.[29-31]

2.10. Statistical Analysis

Experimental results were expressed as mean ± standard error of the mean (SEM). Statistical analysis was performed using GraphPad Prism version 9.0. Two-way analysis of variance (ANOVA), followed by Dunnett's multiple-comparison test, was reported for evaluating differences between groups, where appropriate. Statistical significance was determined using the predefined significance threshold.[32-34]

RESULTS

3.1 Invitro assay : α-Amylase Inhibitory Activity of HAIQ

The α-amylase inhibitory activity of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ) was evaluated at concentrations ranging from 20 to 100 µg/mL, using acarbose as the standard inhibitor. The potential anti-hyperglycaemic effect of HAIQ may partly involve inhibition of pancreatic α-amylase, an enzyme responsible for the breakdown of dietary starch into smaller carbohydrates. Inhibition of this enzyme can slow carbohydrate digestion and potentially reduce the rate at which glucose becomes available for intestinal absorption, thereby helping to moderate postprandial blood glucose elevations. The α-amylase inhibitory activity observed in the present study is consistent with a possible contribution of this mechanism to the glucose-lowering effects observed in the STZ-induced diabetic and dexamethasone-induced insulin-resistant rat models. The α-amylase inhibitory activity of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ) was evaluated at concentrations ranging from 20 to 100 µg/mL, using acarbose as the standard inhibitor. HAIQ demonstrated a concentration-dependent increase in α-amylase inhibition, with percentage inhibition increasing from 8.62% at 20 µg/mL to 66.09% at 100 µg/mL. The calculated IC_{50} value of HAIQ was 78.85 µg/mL, compared with 50.80 µg/mL for acarbose, indicating that the extract exhibited lower inhibitory potency than the standard under the experimental conditions. Preliminary phytochemical screening of HAIQ revealed flavonoids, alkaloids, glycosides, steroids, and tannins. Some plant-derived compounds, particularly certain flavonoids and tannins, have been reported to interact with carbohydrate-

digesting enzymes; however, the compounds responsible for the observed inhibitory activity in HAIQ have not yet been identified.

Table 1: Invitro Assay : α-Amylase Inhibitory Activity of HAIQ

			α-amylase enzyme inhibition assay					IC50 (µg/ml)
SR.NO	Sample code	Concentration (µg/ml)	Absorbance at 540nm				% Inhibition	
			Test 1	Test 2	Test 3	Mean		
1	Control		1.74	1.74	1.74	**1.74**	-	**50.80**
2	Standard (Acarbose)	**20**	1.42	1.45	1.40	**1.42**	**18.39%**	
		40	1.07	1.09	1.05	**1.07**	**38.51%**	
		60	0.71	0.74	0.69	**0.71**	**59.20%**	
		80	0.50	0.53	0.48	**0.50**	**71.26%**	
		100	0.29	0.28	0.25	**0.27**	**84.48%**	
3	Ipomoea quamoclit	**20**	1.55	1.59	1.62	**1.59**	**8.62%**	**78.85**
		40	1.24	1.29	1.25	**1.26**	**27.59%**	
		60	1.10	1.12	1.15	**1.12**	**35.63%**	
		80	0.88	0.85	0.81	**0.85**	**51.15%**	
		100	0.59	0.61	0.57	**0.59**	**66.09%**	

3.2. Acute oral toxicity

The acute oral toxicity study was conducted to evaluate the safety of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ) and to determine a suitable dose for further pharmacological investigations. The animals were observed for changes in their normal behavioural patterns, signs of toxicity, and mortality following oral administration of the extract. The extract was found to be well tolerated up to a maximum dose of 2000 mg/kg body weight, with no mortality observed during the study period. Based on the findings of the acute toxicity assessment and considering the requirements of the subsequent pharmacological evaluation, a dose of 400 mg/kg body weight was selected for further biological studies.

3.3. Effect of different treatments on blood Glucose Levels in STZ-Induced Diabetic Rats.

Table 1. illustrates the effect of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ), metformin, and their combination on blood glucose levels in STZ-induced diabetic rats. Streptozotocin (STZ) is widely used to induce experimental diabetes in rodents through its toxic effects on pancreatic β-cells, resulting in impaired insulin secretion and hyperglycemia. In the present study, STZ-induced diabetic control rats exhibited persistently elevated blood glucose levels, increasing from 205.0 ± 1.12 mg/dL in Week 1 to 212.0 ± 1.10 mg/dL in Week 4, confirming the maintenance of hyperglycemia throughout the experimental period. In contrast, treatment with metformin (50 mg/kg) produced a progressive reduction in blood glucose levels from 175.0 ± 1.04 mg/dL in Week 1 to 126.0 ± 1.06 mg/dL in Week 4. Similarly, administration of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ) at 400 mg/kg reduced blood glucose levels from 188.0 ± 1.07 mg/dL to 145.0 ± 1.01 mg/dL over the four-week treatment period, indicating a potential antihyperglycemic effect. Notably, the combination of metformin (50 mg/kg) and HAIQ (400 mg/kg) demonstrated the greatest reduction, with blood glucose levels decreasing from 166.0 ± 1.00 mg/dL in Week 1 to 98.0 ± 1.08 mg/dL in Week 4, approaching the values observed in the normal control group, which

ranged from 90.0 ± 0.96 to 94.0 ± 1.05 mg/dL. These findings indicate that HAIQ may possess antidiabetic activity and that its co-administration with metformin was associated with a greater reduction in blood glucose levels than either treatment alone under the conditions of this study. The observed antidiabetic activity of *Ipomoea quamoclit L*. may be associated with its phytochemical constituents, including flavonoids, alkaloids, glycosides, tannins, and other bioactive compounds identified during preliminary phytochemical screening. These constituents may contribute to the observed reduction in blood glucose levels through potential mechanisms such as modulation of carbohydrate metabolism, inhibition of intestinal carbohydrate-digesting enzymes, and effects on glucose utilization as shown in Figure 1.

Table 1: Effect of Different Treatments.on Blood Glucose Levels of STZ Induced Diabetic Rats.

Sr. No.	Group	Week 1 (Mean ± SEM)	Week 2 (Mean ± SEM)	Week 3 (Mean ± SEM)	Week 4 (Mean ± SEM)	Interpretation
1	Normal Control (NC)	90.0 ± 0.96	92.0 ± 1.01	93.0 ± 0.98	94.0 ± 1.05	Normal glycemic homeostasis
2	Diabetic Control (DM)	205.0 ± 1.12	208.0 ± 1.08	210.0 ± 1.15	212.0 ± 1.10	Persistent hyperglycemia
3	Metformin (50 mg/kg)	175.0 ± 1.04	156.0 ± 0.99	141.0 ± 1.02	126.0 ± 1.06	Progressive reduction in blood glucose
4	HAIQ (400 mg/kg)	188.0 ± 1.07	170.0 ± 1.03	158.0 ± 1.09	145.0 ± 1.01	Reduction in blood glucose
5	Metformin (50 mg/kg) + HAIQ (400 mg/kg)	166.0 ± 1.00	142.0 ± 0.97	121.0 ± 1.05	98.0 ± 1.08	Greatest reduction among the treatment groups

Data are presented as mean ± SEM (n = 6). Statistical analysis was performed using two-way ANOVA followed by Tukey's multiple comparison test, with $p < 0.05$ considered statistically significant.

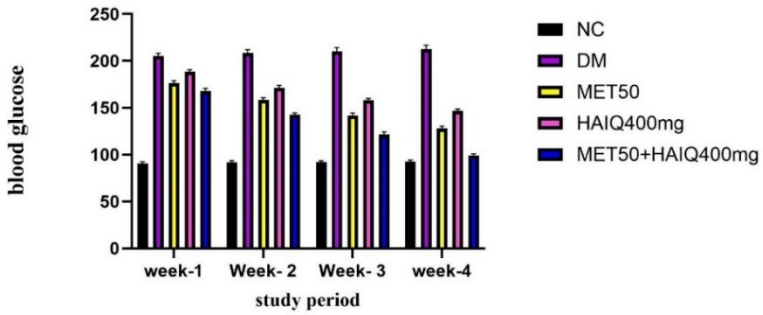


Figure 1: Effect of Different Treatments.on Blood Glucose Levels of STZ Induced Diabetic Rats

3.4. Effect of HAIQ, Metformin, and Their Combination on Body Weight in STZ-Induced Diabetic Rats

STZ-induced diabetes is commonly associated with alterations in body weight due to disturbed glucose utilization and metabolic imbalance. In the present study, the normal control group showed relatively stable body weight, with values ranging from 200.0 ± 1.12 g to 210.0 ± 1.18 g throughout the four-week observation period. In contrast, the diabetic control group exhibited a gradual decline in body weight from 188.0 ± 1.09 g in Week 1 to 183.0 ± 1.17 g in Week 4, indicating diabetes-associated weight loss. Treatment with metformin showed an overall improvement in body weight, increasing from 192.0 ± 1.05 g in Week 1 to 197.0 ± 1.10 g in Week 4. Similarly, the HAIQ-treated group showed an initial decrease from 185.0 ± 1.13 g to 181.0 ± 1.15 g by Week 3, followed by an increase to 185.0 ± 1.07 g in Week 4 as shown in Table 2. Notably, the combination treatment group demonstrated a progressive increase in body weight from 195.0 ± 1.02 g in Week 1 to 201.0 ± 1.08 g in Week 4, suggesting a potential beneficial effect on maintaining

body weight during the diabetic condition. Overall, these findings indicate that treatment with HAIQ, particularly in combination with metformin, was associated with improved body weight trends compared with the diabetic control group as Figure 2.

Table 2: Effect of Different Treatments.on Body Weight in STZ Induced Diabetic Rats

Sr. No.	Group	Week-1 (Mean ± SEM)	Week-2 (Mean ± SEM)	Week-3 (Mean ± SEM)	Week-4 (Mean ± SEM)	Interpretation
1	Nor.mal Cont.rol	210.0 ± 1.18	200.0 ± 1.12	205.0 ± 1.20	208.0 ± 1.15	Nor.mal Body Wei.ght Mainte.nance
2	Diab.etic Cont.rol	188.0 ± 1.09	184.0 ± 1.14	186.0 ± 1.11	183.0 ± 1.17	Progre.ssive Body Wei.ght Loss
3	Stan.dard (Met-f.ormin)	192.0 ± 1.05	195.0 ± 1.08	198.0 ± 1.06	197.0 ± 1.10	Preve.ntion of Dia-betes.-Induced Wei.ght Loss
4	Test Gro.up (HAIQ)	185.0 ± 1.13	183.0 ± 1.09	181.0 ± 1.15	185.0 ± 1.07	Mode.rate Improv.e-ment in Body Wei.ght
5	Combin.ation Gro.up	195.0 ± 1.02	198.0 ± 1.06	200.0 ± 1.04	201.0 ± 1.08	Near-.Normal Body Wei.ght Restor.ation

Data are presented as mean ± SEM (n = 6). Statistical analysis was performed using two-way ANOVA followed by Tukey's multiple comparison test, with $p < 0.05$ considered statistically significant.

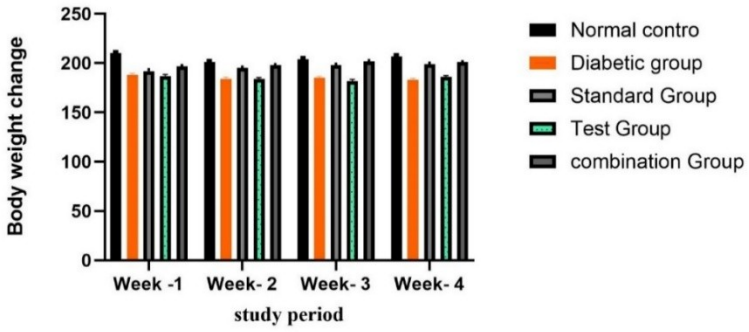


Figure 2: Effect of Different Treatments.on Body Weight of STZ Induced Diabetic Rats.

3.5 Effect of HAIQ, Metformin, and Their Combination on AST, ALT in STZ-Induced Diabetic Rats.

STZ-induced diabetes can be associated with hepatic dysfunction, which may be reflected by elevated serum aspartate aminotransferase (AST/SGOT) and alanine aminotransferase (ALT/SGPT) levels. In the present study, the diabetic control group exhibited markedly elevated AST and ALT levels of 156.00 ± 2.37 U/L and 118.00 ± 1.88 U/L, respectively, compared with the normal control group (68.00 ± 1.31 U/L and 42.00 ± 0.86 U/L), suggesting possible hepatic alterations associated with diabetes. Treatment with metformin reduced AST and ALT levels to 92.00 ± 1.67 U/L and 63.00 ± 1.22 U/L, respectively. Similarly, administration of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ) resulted in AST and ALT levels of 101.00 ± 1.59 U/L and 71.00 ± 1.31 U/L as shown in Table 3, indicating an improvement compared with the diabetic control group. Notably, combination treatment with metformin (50 mg/kg) and HAIQ (400 mg/kg) produced the lowest enzyme levels among the diabetic treatment groups, at 76.00 ± 1.14 U/L for AST and 49.00 ± 0.98 U/L for ALT, approaching the values observed in the normal control group. These findings suggest that the combination treatment was associated with a greater improvement in serum liver enzyme levels than either treatment alone as Figure 3.

The observed reduction in serum AST and ALT levels following combined administration of HAIQ and metformin may be related to improved glycemic control

and reduced metabolic stress associated with STZ-induced diabetes. Metformin lowers blood glucose primarily by reducing hepatic glucose production and improving glucose homeostasis. Meanwhile, the phytochemical constituents identified in HAIQ, including flavonoids, alkaloids, and sterols, may contribute to its potential biological activity through antioxidant and other metabolic effects. These complementary actions may help limit diabetes-associated cellular injury and contribute to the improvement in serum liver enzyme levels observed in the combination-treated group.

Sr. No.	Group	SGOT (AST) (U/L), Mean ± SEM	SGPT (ALT) (U/L), Mean ± SEM	Interpretation
1	Normal Control (NC)	68.00 ± 1.31	42.00 ± 0.86	Normal liver enzyme levels
2	Diabetic Control (DM)	156.00 ± 2.37	118.00 ± 1.88	Elevated liver enzyme levels
3	Metformin (50 mg/kg)	92.00 ± 1.67	63.00 ± 1.22	Reduced liver enzyme levels
4	HAIQ (400 mg/kg)	101.00 ± 1.59	71.00 ± 1.31	Improvement in liver enzyme levels
5	Metformin (50 mg/kg) + HAIQ (400 mg/kg)	76.00 ± 1.14	49.00 ± 0.98	Levels approaching normal control

Table 3: Effect of HAIQ, Metformin, and Their Combination on Serum AST and ALT Levels in STZ-Induced Diabetic Rats.

Data are presented as mean ± SEM (n = 6). Statistical significance was assessed using one-way ANOVA followed by Dunnett's post hoc test, with $p < 0.05$ considered statistically significant.

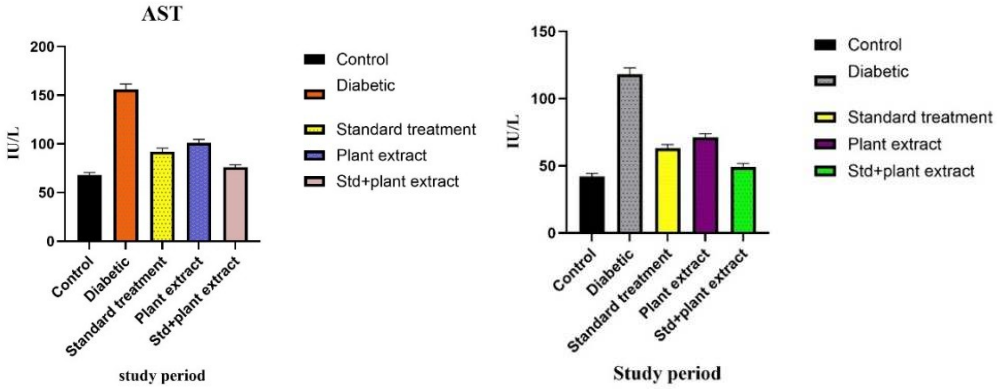


Figure 3: Effect of HAIQ, Metformin, and Their Combination on Serum AST and ALT Levels in STZ-Induced Diabetic Rats.

3.6 Effect of different treatments on Blood Glucose Levels in Dexamethasone induced Diabetic Rats.

Dexamethasone administration can induce hyperglycemia by promoting hepatic gluconeogenesis and impairing insulin-mediated glucose utilization. In the present study, the diabetic control group exhibited a progressive increase in blood glucose levels from 195.0 ± 2.6 mg/dL on Day 6 to 216.0 ± 3.1 mg/dL on Day 12, indicating persistent hyperglycemia. In contrast, metformin treatment reduced blood glucose levels from 175.0 ± 2.3 mg/dL to 118.0 ± 1.8 mg/dL over the same period. Treatment with HAIQ (400 mg/kg) also produced a progressive reduction, with glucose levels decreasing from 180.0 ± 2.5 mg/dL to 128.0 ± 1.9 mg/dL. Notably, the combination of metformin (50 mg/kg) and HAIQ (400 mg/kg) showed the greatest reduction among the treatment groups, with blood glucose levels declining from 168.0 ± 2.2 mg/dL on Day 6 to 108.0 ± 1.6 mg/dL as showm in Fig.4. On Day 12, approaching the normal control value of 102.0 ± 1.9 mg/dL. These findings suggest that HAIQ may have beneficial antihyperglycemic activity and that its combination with metformin was associated with

improved glycemic control compared with either treatment alone.

Dexamethasone-induced hyperglycemia is primarily associated with increased hepatic glucose production and impaired peripheral glucose utilization. Metformin may counteract these effects by suppressing hepatic gluconeogenesis and improving insulin-mediated glucose handling. The observed α-amylase inhibitory activity and phytochemical constituents of HAIQ, including flavonoids, alkaloids, glycosides, and tannins, may contribute to its potential effects on carbohydrate metabolism and postprandial glucose regulation. When administered together, HAIQ and metformin may exert complementary effects on glucose homeostasis, contributing to the greater reduction in blood glucose observed in the combination group.

Table 4: Effect of HAIQ, Metformin, and Their Combination on Blood Glucose Levels in Dexamethasone-Induced Insulin-Resistant Rats

Sr. No.	Group	Day 6 (mg/dL)	Day 8 (mg/dL)	Day 10 (mg/dL)	Day 12 (mg/dL)
1	Normal Control (NC)	96.0 ± 1.8	98.0 ± 1.6	100.0 ± 1.7	102.0 ± 1.9
2	Insulin-Resistant Control	195.0 ± 2.6	198.0 ± 2.4	208.0 ± 2.8	216.0 ± 3.1
3	Metformin (50 mg/kg)	175.0 ± 2.3	158.0 ± 2.1	138.0 ± 1.9	118.0 ± 1.8
4	HAIQ (400 mg/kg)	180.0 ± 2.5	165.0 ± 2.2	148.0 ± 2.0	128.0 ± 1.9
5	Metformin (50 mg/kg) + HAIQ (400 mg/kg)	168.0 ± 2.2	145.0 ± 2.0	122.0 ± 1.8	108.0 ± 1.6

Data are presented as mean ± SEM (n = 6). Statistical analysis was performed using two-way ANOVA followed by Tukey's multiple comparison test, with $p < 0.05$ considered statistically significant.

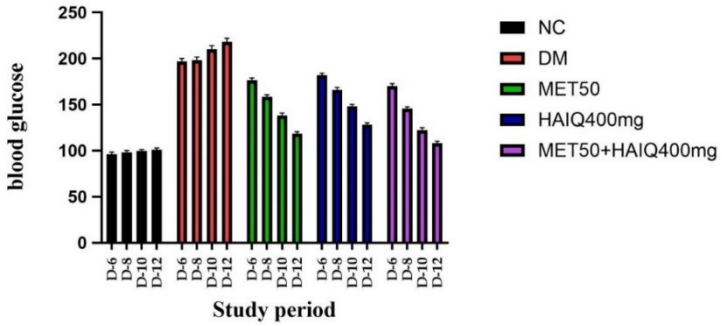


Figure 4: Effect of HAIQ, Metformin, and Their Combination on Blood Glucose Levels in Dexamethasone-Induced Insulin-Resistant Rats

3.7 Effect of HAIQ, Metformin, and Their Combination on Body Weight in Dexamethasone-Induced Insulin-Resistant Rats.

Dexamethasone, a synthetic glucocorticoid, can disturb glucose and energy homeostasis by promoting hepatic gluconeogenesis, reducing insulin-mediated glucose uptake in peripheral tissues, and altering protein and lipid metabolism. These effects may contribute to changes in body weight and glycemic control. Body weight changes provide a useful supportive indicator of metabolic alterations in experimental insulin resistance. In the present study, the normal control group showed a progressive increase in body weight, from 198.0 ± 1.6 g on Day 4 to 207.0 ± 1.9 g on Day 10. In contrast, the insulin-resistant control group exhibited a reduction from 182.0 ± 1.9 g to 170.0 ± 2.1 g by Day 8, followed by a partial increase to 176.0 ± 2.2 g on Day 10. This pattern may reflect metabolic disturbances associated with dexamethasone exposure, although body weight alone cannot establish the underlying cause.

Table 5: Effect of HAIQ, Metformin, and Their Combination on Body Weight in Dexamethasone-Induced Insulin-Resistant Rats.

Sr. No.	Experimental Group	Day 4 (g)	Day 6 (g)	Day 8 (g)	Day 10 (g)	Interpretation
1	Normal Control (NC)	198.0 ± 1.6	201.0 ± 1.7	204.0 ± 1.8	207.0 ± 1.9	Progressive body weight gain
2	Insulin-Resistant Control	182.0 ± 1.9	178.0 ± 2.0	170.0 ± 2.1	176.0 ± 2.2	Body weight reduction followed by partial recovery
3	Metformin (50 mg/kg)	200.0 ± 1.8	203.0 ± 1.9	202.0 ± 1.8	206.0 ± 2.0	Overall maintenance of body weight
4	HAIQ (400 mg/kg)	190.0 ± 1.7	188.0 ± 1.8	186.0 ± 1.9	191.0 ± 2.0	Initial weight reduction followed by partial recovery
5	Metformin (50 mg/kg) + HAIQ (400 mg/kg)	194.0 ± 1.6	197.0 ± 1.7	200.0 ± 1.8	198.0 ± 1.9	Overall increase in body weight, with a slight decline on Day 10

Metformin-treated rats maintained body weight relatively well, increasing from 200.0 ± 1.8 g to 206.0 ± 2.0 g over the observation period. HAIQ-treated rats showed an initial decline in body weight, followed by a partial recovery to 191.0 ± 2.0 g on Day 10. The combination-treated group demonstrated an overall increase from 194.0 ± 1.6 g on Day 4 to 198.0 ± 1.9 g on Day 10, although a slight decline occurred between Days 8 and 10. These observations suggest that treatment, particularly the combination of metformin and HAIQ, was associated with maintenance of body weight compared with the insulin-resistant control group. The observed effects of HAIQ may be related, in part, to its phytochemical constituents, including flavonoids, alkaloids, glycosides, and tannins, as well as its demonstrated in vitro α-amylase inhibitory activity. These constituents may influence carbohydrate digestion or other processes involved in glucose homeostasis; however, their specific contribution to body weight regulation has not been established. When administered with metformin, HAIQ may potentially complement the drug's glucose-lowering effects through different or overlapping mechanisms.

Data are presented as mean ± SEM (n = 6). Statistical significance was assessed using one-way ANOVA followed by Dunnett's post hoc test, with $p < 0.05$ considered statistically significant.

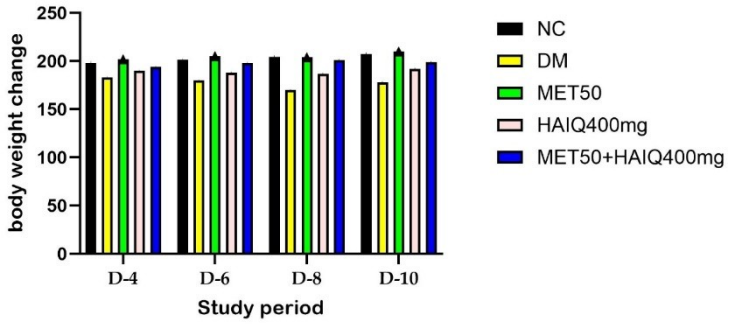


Figure 5: Effect of HAIQ, Metformin, and Their Combination on Body Weight in Dexamethasone-Induced Insulin-Resistant Rats

3.8 Effect of Standard Treatment, HAIQ, and Their Combination on Serum ALT and AST Levels in Dexamethasone-Induced Insulin-Resistant Rats

Dexamethasone-induced metabolic disturbances may be accompanied by altered hepatic glucose and lipid metabolism, oxidative stress, and changes in cellular integrity. These processes could contribute to increased leakage of intracellular aminotransferases into the circulation. Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were evaluated as biochemical indicators of possible hepatic alterations associated with dexamethasone-induced insulin resistance and the effects of treatment. The insulin-resistant control group exhibited higher ALT and AST levels (76.5 ± 1.8 and 75.8 ± 1.9 IU/L, respectively) than the normal control group (39.0 ± 1.2

and 38.5 ± 1.3 IU/L, respectively). These elevations may indicate hepatic stress or injury associated with the experimental condition. Treatment with metformin (50 mg/kg) reduced ALT and AST levels to 45.2 ± 1.4 and 46.0 ± 1.5 IU/L, respectively. Similarly, HAIQ (400 mg/kg) treatment reduced ALT to 48.0 ± 1.6 IU/L and AST to 47.5 ± 1.6 IU/L. The combination of metformin and HAIQ produced the lowest values among the treatment groups, with ALT at 37.2 ± 1.3 IU/L and AST at 36.8 ± 1.2 IU/L, which were close to the normal control value

The effects observed with HAIQ may potentially be associated with its phytochemical constituents, including flavonoids, alkaloids, glycosides, and tannins, which could influence metabolic or cellular stress pathways. When administered in combination, HAIQ and metformin may produce complementary effects that contribute to the observed reduction in serum ALT and AST levels.

Sr. No.	Experimental Group	ALT (IU/L), Mean ± SEM	AST (IU/L), Mean ± SEM	Interpretation
1	Normal Control	39.0 ± 1.2	38.5 ± 1.3	Baseline serum enzyme levels
2	Insulin-Resistant Control	76.5 ± 1.8	75.8 ± 1.9	Elevated serum liver enzyme levels
3	Metformin (50 mg/kg)	45.2 ± 1.4	46.0 ± 1.5	Reduction in elevated serum enzyme levels
4	HAIQ (400 mg/kg)	48.0 ± 1.6	47.5 ± 1.6	Reduction in elevated serum enzyme levels
5	Metformin (50 mg/kg) + HAIQ (400 mg/kg)	37.2 ± 1.3	36.8 ± 1.2	Serum enzyme levels close to control values

Table 6: Effect of Metformin, HAIQ, and Their Combination on Serum ALT and AST Levelsin Dexamethasone-Induced Insulin-Resistant Rats

Data are presented as mean ± SEM (n = 6). Statistical significance was assessed using one-way ANOVA followed by Dunnett's post hoc test, with $p < 0.05$ considered statistically significant.

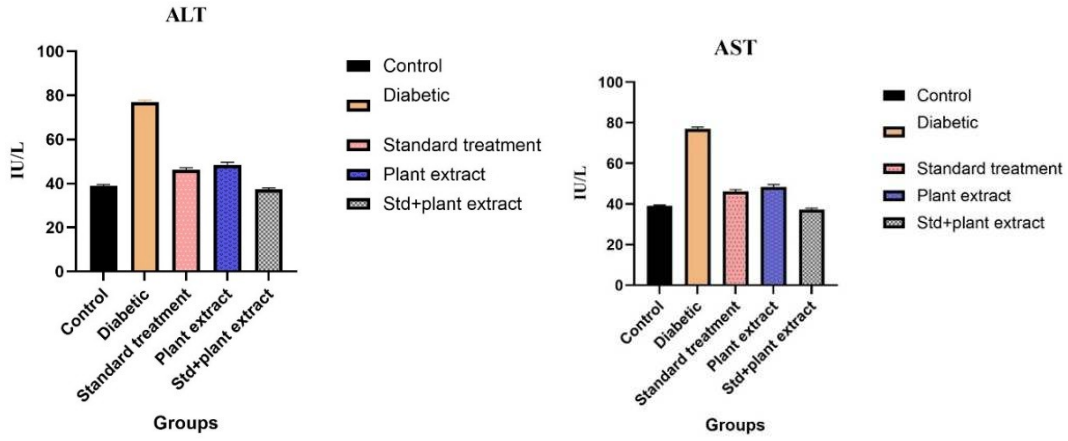
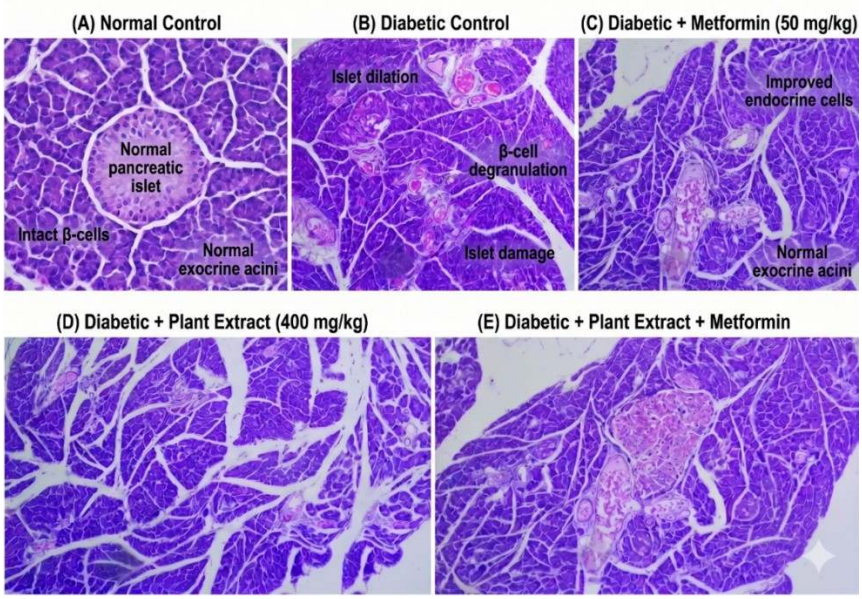


Figure 6: Effect of Metformin, HAIQ, and Their Combination on Serum ALT and AST Levels in Dexamethasone-Induced Insulin-Resistant Rats

3.9 Histopathological Changes in Pancreatic Tissue of Experimental Animals Stained with Hematoxylin and Eosin (H&E) Fig.1 : STZ-Induced Diabetic Rats.



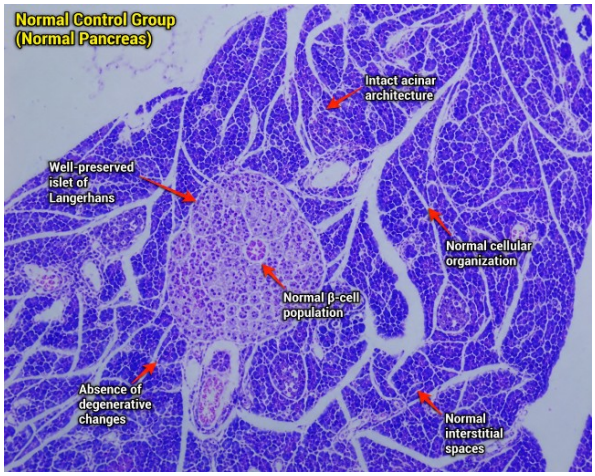
(A) Normal Control: The pancreatic tissue shows normal histological architecture, with well-defined islets of Langerhans, intact endocrine cells, and normally arranged exocrine acini.

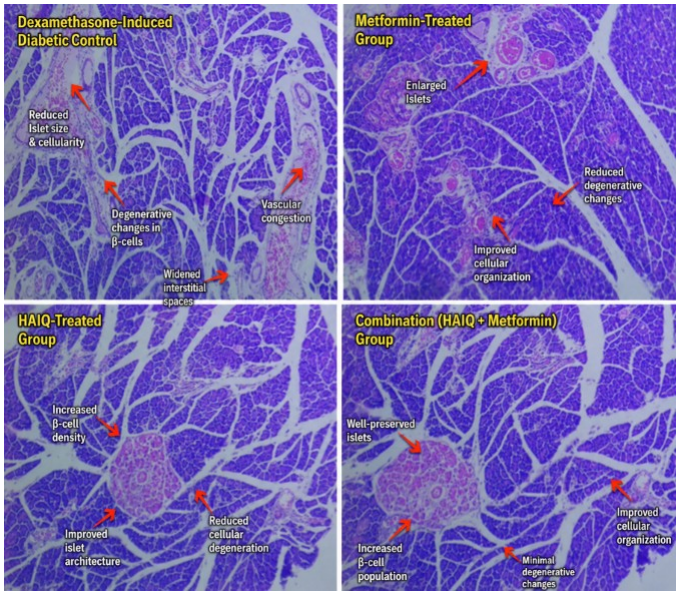
(B) Diabetic Control: The pancreatic tissue shows disrupted islet architecture, islet shrinkage and damage, β-cell degranulation, and reduced cellular organization, consistent with STZ-induced pancreatic injury.

(C) Diabetic + Metformin (50 mg/kg): The pancreatic section shows partial preservation of islet architecture, with improved endocrine cellular organization and relatively preserved exocrine acini compared with the diabetic control.

(D) Diabetic + Plant Extract (HAIQ, 400 mg/kg): The pancreatic section shows apparent preservation of pancreatic tissue architecture, with improved organization of the islet region and endocrine cellularity compared with the diabetic control.

(E) Diabetic + Plant Extract (HAIQ, 400 mg/kg) + Metformin (50 mg/kg): The pancreatic section shows comparatively better preservation of islet architecture and pancreatic cellular organization than the diabetic control, suggesting a potential protective effect of the combined treatment.





3.10 Histopathological Changes in Pancreatic Tissue of Experimental Animals Stained with Hematoxylin and Eosin (H&E) : Dexamethasone induced insulin resistance

(A) Normal Control: The pancreatic tissue exhibits well-preserved islets of Langerhans, normal endocrine cellularity, intact acinar architecture, and normal interstitial spaces, with no apparent degenerative changes.

(B) Dexamethasone-Induced Insulin-Resistant Control: The pancreatic section shows reduced islet size and cellularity, degenerative changes in β-cells, vascular congestion, and widened interstitial spaces, indicating alterations in pancreatic tissue architecture.

(C) Dexamethasone + Metformin (50 mg/kg): The pancreatic tissue shows apparent improvement in cellular organization, enlarged islet profiles, and reduced degenerative changes compared with the dexamethasone-induced control.

(D) Dexamethasone + HAIQ (400 mg/kg): The pancreatic section exhibits improved islet architecture, increased apparent β-cell density, and reduced cellular degeneration compared with the untreated insulin-resistant control.

(E) Dexamethasone + HAIQ (400 mg/kg) + Metformin (50 mg/kg): The pancreatic tissue shows comparatively well-preserved islet architecture, increased apparent endocrine cellularity, improved cellular organization, and fewer apparent degenerative changes than the dexamethasone-induced control.

DISCUSSION :

The present investigation evaluated the antidiabetic potential of the hydroalcoholic extract of *Ipomoea quamoclit L.* (HAIQ) in streptozotocin (STZ)-induced diabetic and dexamethasone-induced insulin-resistant rats. STZ induces experimental diabetes primarily through pancreatic β-cell damage, resulting in impaired insulin secretion and persistent hyperglycaemia, whereas dexamethasone promotes hyperglycaemia predominantly by inducing insulin resistance and altering glucose metabolism. The elevated fasting blood glucose levels observed in the respective untreated control groups indicated successful establishment of both experimental models. These models provided a basis for evaluating the glucose-lowering effects of HAIQ administered alone and in combination with metformin.

Oral administration of HAIQ (400 mg/kg) produced a progressive reduction in fasting blood glucose levels throughout the 28-day treatment period in STZ-induced diabetic rats. Although the extract was less effective numerically than metformin alone, the combination of HAIQ with metformin produced the greatest reduction in blood glucose levels among the treated groups. A similar glucose-lowering trend was observed in dexamethasone-induced insulin-resistant rats, suggesting

that the effects of HAIQ may extend across experimental models involving insulin deficiency and insulin resistance. The gradual reduction in blood glucose levels suggests a potential role for HAIQ in improving glycaemic regulation over the treatment period. Its observed α-amylase inhibitory activity may contribute to this effect by limiting the enzymatic breakdown of complex carbohydrates and potentially reducing glucose availability following carbohydrate intake. Other mechanisms, including enhanced peripheral glucose utilization, improved insulin sensitivity, or protection of residual pancreatic β-cells, remain possible but require direct experimental confirmation.

Phytochemical screening of HAIQ revealed the presence of flavonoids, alkaloids, glycosides, steroids, tannins, carbohydrates, and amino acids. Among these constituents, flavonoids and other plant-derived compounds have been reported to influence glucose homeostasis through several potential mechanisms, including modulation of carbohydrate-digesting enzymes, insulin signalling, and cellular glucose utilization. The α-amylase inhibitory activity observed in the present study provides experimental support for the possibility that HAIQ may influence carbohydrate digestion and contribute to its antihyperglycaemic effect.

Changes in body weight were observed across the experimental groups in both STZ-induced diabetic and dexamethasone-induced insulin-resistant rats. In STZ-induced diabetic rats, the diabetic control group showed a slight overall reduction in body weight during the study, whereas the metformin-treated and combination-treated groups exhibited comparatively better maintenance of body weight. HAIQ treatment alone did not produce a consistent improvement throughout the observation period. In the dexamethasone model, body weight also varied among the treatment groups, with the metformin-treated group showing an overall increase and the HAIQ-treated group showing fluctuations over time. These changes may reflect differences in metabolic status and treatment responses. In diabetes, inadequate glucose utilization and increased protein and fat catabolism can contribute to body weight loss.

In the present study, HAIQ at 400 mg/kg demonstrated antihyperglycaemic activity in both STZ-induced diabetic and dexamethasone-induced insulin-resistant rats. However, the combination of HAIQ (400 mg/kg) and metformin (50 mg/kg) produced the greatest numerical reduction in blood glucose levels among the treated groups, while metformin alone also demonstrated a glucose-lowering effect.

These findings suggest that HAIQ may have potential as an adjunct to conventional antidiabetic treatment. The observed α-amylase inhibitory activity and comparatively improved pancreatic histological features in treated STZ-induced diabetic rats provide additional support for further investigation of its antidiabetic potential. Overall, the findings provide preliminary experimental evidence supporting further phytopharmacological investigation of *Ipomoea quamoclit L*., including dose-ranging studies, mechanistic evaluation, and comprehensive safety assessment.

CONCLUSION :

The present study was undertaken to evaluate the antidiabetic potential of the hydroalcoholic extract of *Ipomoea quamoclit L*. (HAIQ) in streptozotocin (STZ)-induced diabetic and dexamethasone-induced insulin-resistant rats. The findings demonstrated a progressive reduction in fasting blood glucose levels following HAIQ administration, with the combination of HAIQ (400 mg/kg) and metformin (50 mg/kg) producing the greatest numerical glucose-lowering effect among the treated groups. HAIQ also exhibited α-amylase inhibitory activity, while biochemical findings showed lower serum AST and ALT levels in treated animals compared with the respective untreated disease-control groups. Changes in body weight and the comparatively improved pancreatic histological features observed in treated STZ-induced diabetic rats provided additional findings relevant to the extract's potential antidiabetic effects.

The observed activity may be associated, in part, with the phytochemical constituents identified during preliminary screening, including flavonoids, alkaloids, glycosides, steroids, and tannins. The α-amylase inhibitory activity suggests a possible contribution

through modulation of carbohydrate digestion, although the mechanisms underlying the in vivo glucose-lowering effects remain to be elucidated. As only one dose of HAIQ was evaluated in the animal models, a dose-dependent response cannot be confirmed. Furthermore, the present findings do not establish antidyslipidaemic activity because serum lipid parameters were not assessed.

Overall, this study provides preliminary experimental evidence supporting further investigation of *Ipomoea quamoclit L*. as a potential source of antidiabetic agents and as a possible adjunct to metformin therapy. Further studies involving dose-ranging experiments, insulin and glucose-metabolism markers, quantitative histopathological assessment, isolation and confirmation of active constituents, and comprehensive toxicity evaluation are warranted to clarify its pharmacological effects and establish its safety and therapeutic potential.

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