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EVALUATION OF THE ANTIDIABETIC ACTIVITY OF METHANOLIC EXTRACT OF COPTIS TRIFOLIA IN STREPTOZOTOCIN-INDUCED DIABETES RATS

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Abstract:

The present study aimed to evaluate the antidiabetic, hypolipidemic, and antioxidant activities of the methanolic extract of Coptis trifolia in a streptozotocin-induced hypertensive rat model. Rats were divided into five groups: normal control, Streptozotocin control, standard drug (hydrochlorothiazide), and two doses of Coptis trifolia extract (200 mg/kg and 500 mg/kg). Treatment was administered orally, and parameters including fasting blood glucose, serum insulin, lipid profile, liver glycogen content, antioxidant enzyme levels (GST, GSH, SOD), and body weight changes were measured.

Streptozotocin treatment induced significant hyperglycemia, dyslipidemia, oxidative stress, and weight loss. Administration of Coptis trifolia extract significantly reduced fasting blood glucose and improved glucose tolerance, serum insulin levels, and liver glycogen content in a dose-dependent manner. Additionally, the extract normalised serum lipid profiles by lowering total cholesterol, triglycerides, LDL-C, and VLDL-C, while increasing HDL-C. Antioxidant enzyme activities and total protein content were markedly restored by Coptis trifolia treatment, indicating a potent antioxidative effect.

The Coptis trifolia methanolic extract exerts multi-faceted protective effects against metabolic disturbances associated with Streptozotocin induced hypertension. This supports its potential therapeutic application as a natural agent for managing diabetes and cardiovascular complications.

Keywords: *Coptis trifolia , hypertension, hypertension, LDL-C, cholesterols*

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INTRODUCTION:

DIABETES MELLITUS

Diabetes mellitus is a group of diseases characterised by high levels of blood glucose resulting from defects in insulin production, insulin action, or both, altered metabolism of lipids, carbohydrates, and proteins, and an increased risk of complications of vascular diseases[1]. It is the most common endocrine disorder and can be associated with serious complications and premature death.

HISTORY [2, 3]

Knowledge of diabetes mellitus dates back to many centuries. Eber's Egyptian Papyrus (1500 BC) described it as an illness associated with the passage of much urine. A renowned Greek physician Arataeus of Cappodica coined the name diabetes (a siphon which described it as melting down of flesh and limbs into urine.

Scholars in China, Japan, and India during the 3rd and 6th centuries AD wrote of a condition with polyurea in which urine was 'sweet and sticky'. Although it has been for centuries, it was until 1674, that Willis added the observation 'as it is imbued with honey and sugar'. The name diabetes mellitus (mellitus- honey) was thus established. A century after Willis, Dobson demonstrated that the sweetness was due to sugar. From the earliest recorded history of diabetes, progress in understanding came slowly until the middle of the 19th century. An association was established with a disturbance in the beta cells, clustered as tiny islets of tissue in the exocrine pancreas. Brockman first noticed the presence of these islets in fish during the 19th century. Soon after, the German scientists, Mering and Minkowski, found that surgical removal of the pancreas produced diabetes in dogs. The breakthrough of the discovery of insulin was by Fredrick Banting and Charles Best in 1921. By 1952, various purified modified versions of insulin were available and by 1982, recombinant DNA insulin became available. Thus diabetes, which was synonymous with an inevitably fatal disease, became a more benign controllable disease.

EPIDEMIOLOGY [4]

In the year 2000, according to the World Health Organization, at least 171 million people worldwide suffered from diabetes. Its incidence is increasing rapidly, and it is estimated that by 2030, this number will almost double. Diabetes mellitus occurs throughout the world but is more common (especially type 2) in the more developed countries. The greatest increase in prevalence is, however, expected to occur in Asia and Africa, where most patients will probably be found by 2030. The increase in the incidence of diabetes in developing countries follows the trend of urbanization and lifestyle changes, perhaps most importantly a "Western-style" diet. This has suggested an environmental (i.e., dietary) effect, but there is little understanding of the mechanism(s) at present, though there is much speculation, some of it most compellingly presented. For at least 20 years, diabetes rates in North America have been increasing substantially. In 2010 nearly 26 million people had diabetes in the United States alone, and from those 7 million people remain undiagnosed. Other 57 million people are estimated to have pre-diabetes. The Centres for Disease Control has termed the change an epidemic. The National Diabetes Information Clearinghouse estimates that diabetes costs $132 billion in the United States alone every year. About 5%–10% of diabetes cases in North America are type 1, with the rest being type 2. The fraction of type 1 in other parts of the world differs. Most of this difference is not currently understood. The American Diabetes Association cites the 2003 assessment of the National Centred for Chronic Disease Prevention and Health Promotion (Centres for Disease Control and Prevention) that 1 in 3 Americans born after 2000 will develop diabetes in their lifetime. The diabetes epidemic was more pronounced in India as the World Health Organization (WHO) reports showed that 32 million people had diabetes in the year 2002 and expected that it may rise to 80 million in 2030. According to the American Diabetes Association, approximately 18.3% (8.6 million) of Americans age 60 and older have diabetes. Diabetes mellitus prevalence increases with age, and the numbers of older persons with diabetes are expected to grow as the elderly population increases in number. The National Health and Nutrition Examination Survey (NHANES III) demonstrated that, in the population over 65 years old, 18% to 20% have diabetes, with 40% having either diabetes or its precursor form of impaired glucose tolerance. Indigenous populations in first-world countries have a higher prevalence and increasing incidence of diabetes than their corresponding non-indigenous populations.

2. MATERIALS AND METHODS:

Collection and Authentication of plant material:

The plant *Coptis* trifolia was collected from natural habitat of Ranga Reddy District, Telangana, India. The taxonomic identification of the plant was confirmed by Department of Botany, Government Degree College, Kukatpally, Medchal District, Telangana State. The Macroptilium Atropurpureum were cleaned and drying under sunlight. The dried plant was grind with mortar pestle and passed through sieve no. 14. The powdered plant was kept in airtight container for extraction.

Extraction

The fresh leaves of Coptis trifolia are collected and shade dried for one week, the dried leaves are pulverized by a mechanical grinder reduced to coarse powder and around 60g of powder material was subjected to successively hot continuous extraction .a weight quantity of each of the plant powdered material was extracted by Soxhlet with ethanol of 300ml for 24hrs with intermediate heating at 40 ^{0} c after that the residues were extracted the extract was filtered using Whatmann filter paper and then the extract was evaporated in a rota evaporator and further dried under vacuum.

Percentage of yield

$$Percentage\ of\ yield = \frac{Weight\ of\ crude\ extract}{Weight\ of\ dried\ material} \times 100$$

$$Percentage\ of\ yield = \frac{26g}{200g} \times 100 = 13\%$$

200gms of drug dissolved in 800ml of solvent in 1:4 Ratio

PARAMETERS FOR EVALUATION

Fasting Blood Sugar (FBS):

Fasting blood sugar level was determined by using glucose oxidase peroxidase reactive strips. **Lipid Profile**:

The lipid profiles viz Serum cholesterol, Serum triglycerides, and HDL Cholesterol were estimated by using an auto analyser. On the 15th day, animals were sacrificed by cervical dislocation, and the following parameters were monitored.

Liver Glycogen Content:

The liver glycogen was estimated by the Anthrone method.

Estimation of liver glycogen:

Each liver tissue was minced and placed in an efficient blender containing an approximate volume (about 5ml) of 5% Trichloroacetic acid and homogenized for 3 minutes. The homogenate was centrifuged at 3000 rpm for 5 minutes. The supernatant was decanted upon an acid-washed filter paper (glass wool was used) in a funnel, draining into a graduated cylinder. The residue was then transferred into the blender with an appropriate volume of TCA (5 ml) and homogenized again for 1 minute. The homogenate was centrifuged and the supernatant was poured through the same filter. This was repeated two more times. The total volume of filtrate collected in the graduated cylinder was noted. 10µl of this filtrate was pipetted into 15-ml Pyrex centrifuge tubes. To each tube, 5 ml of 95% ethanol was added and the tubes were allowed to stand overnight at room temperature. After completion of the precipitation of glycogen, the tubes were centrifuged at 3000 rpm for 15 minutes. The clear liquid was gently decanted out from the packed glycogen and the tubes were kept in an inverted position for 10 minutes to make the packed glycogen free from alcohol. The glycogen was dissolved in 2 ml of distilled water.

A reagent blank was prepared by pipetting 2 ml of distilled water into a clean boiling tube. A standard was prepared by pipetting out 2 ml of standard glucose solution (containing 0.1mg glucose), into another tube. The dissolved glycogen was added to different boiling tubes. Then to each tube, 10 ml of anthrone reagent was added. All the tubes were capped tightly with an air condenser and placed in the cold-water bath. After allowing the contents of the tubes to reach the temperature of cold water, (20 minutes) all tubes were taken out and immersed in a boiling water bath to a depth, a little above the level of contents in the tubes for 15 minutes. They were taken out again immersed in a cold-water bath and cooled to room temperature (about 10 minutes). Then the contents of each were transferred to colorimeter tubes and the optical density was read at 620 nm, after adjusting the colorimeter with reagent blank.

Calculation:

$$mg\ of\ glycogen/gm\ of\ liver \frac{DU}{DS} \times 0.05 \times \frac{Total\ volume\ of\ extract\ (ml)}{Weight\ of\ tissue\ (g)} \times \frac{0.9}{0.01}$$

Where,

DU = Optical density of unknown

DS = Optical density of standard

0.05 = mg of glucose in 2 ml of standard solution

0.9 = Conversion factor.

Changes in Body Weight

The changes in the body weight were calculated by checking the weights of individual animals on the 0th, 5th,10th, and 15th day.

Serum Insulin Estimation

On day 15 before the animals were sacrificed 2ml of blood was withdrawn from each animal. The blood was then centrifuged at 3000rpm for 10 minutes to obtain the serum. The serum was analyzed for insulin levels by Radio Immuno Assay at Choithram hospital, Indore, Madhya Pradesh. Insulin concentration in the serum was estimated by using the RIA technique. This technique used a RIA kit provided by the Board of Radiation and Isotope Research (BRITS), Mumbai.

***IN-VIVO* ANTIOXIDANT STUDIES**

Estimation of Total Proteins[6]

Procedure: 1 ml of liver homogenate was taken and made up to 10 ml with 0.5 M sodium hydroxide. From this 1 ml was pipetted out and added with 1 ml of 10 % trichloroacetic acid and centrifuged for 10 minutes at 4000 rpm. The supernatant was discarded and the precipitate was dissolved in 1 ml 0.5 N sodium hydroxide in a boiling water bath at 50ºC for 10 minutes. Then 4 ml of alkaline copper tartrate reagent was added and kept for 10 minutes at room temperature. 0.5 ml of folin's reagent was then added and the absorbance was read at 540 nm. Blank was performed in the same manner but without homogenate. Standard was performed using serum albumin.

Estimation of Superoxide Dismutase (SOD)[7]

Procedure: 1.2ml sodium pyrophosphate buffer, 0.1ml of Phenazine metho sulfate, and 0.3ml of NBT were added to the test tube. 1.2ml homogenate was added to the test tube. The reaction was started by the addition of 0.2 ml of NADH. After incubation at 30ºC for 90 sec, the reaction was stopped by the addition of 1ml glacial acetic acid. The reaction mixture was stirred vigorously and shaken with 4ml butanol. The mixture was allowed to stand for 10 minutes, centrifuged and the butanol layer was taken out. The color intensity of the chromogen in the butanol was measured at 560 nm.

Estimation of Glutathione Synthetase (GSH)[8]

Procedure: 0.2 ml of homogenate was added to the test tube and dissolved in 1.8 ml water. The solution was mixed thoroughly. 3 ml of precipitating solution was added promptly and mixed.

Allowed to stand for 5 minutes at room temperature and filtered through coarse filter paper.

Reagent (ml)	Blank (ml)	Sample (ml)
Filtrate		2
Precipitating solution	1.2	
Water	0.8	
Na2HPO4 solution	8	8
DTNB solution	1	1

Then, the absorbance was measured at 412 nm.

Calculation: The GSH concentration in the tissues was calculated and the results were expressed in U/g/min.

Glutathione - S – Transferase (GST)[9]

Reagents:

1. Phosphate buffer pH 6.5: 1.7418 gm of potassium dihydrogen phosphate in 100 ml of distilled water.

2. GSH (30mM): 16 mg of GSH in 2 ml of double distilled water.

3. CDNB (1- chloro – 2,4, dinitrobenzene): 62 mg CDNB in 10 ml ethanol.

Procedure: 1 ml of CDNB solution was added to 0.6 ml of supernatant of kidney homogenate. 2.2 ml of phosphate buffer pH 6.5 was added to it. This was then incubated at 37 ℃ for 5 minutes. 0.1 ml of 30 mM GSH solution was added. The absorbance was read at 340 nm at intervals of 1, 2, 3, 4, and 5 minutes. The blank reading was carried out in the same manner without the homogenate.

STATISTICAL ANALYSIS

The results of biochemical estimations were reported as mean ± S.E.M The total variation present in the data was analyzed by one-way analysis of variance (ANOVA). Differences among the means were analyzed by Dunnett's test. For this, a window-based PRISM computer package was used.

Oral glucose tolerance test

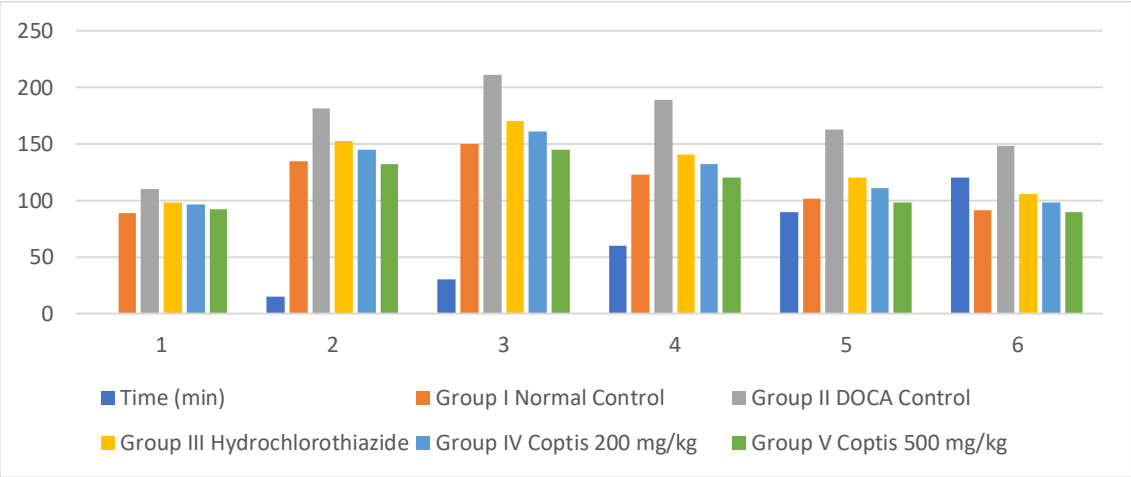
Time (min)	Group I Normal Control	Group II Streptozotocin Control	Group III Hydrochlorothiazide	Group IV Coptis trifolia 200 mg/kg	Group V Coptis trifolia 500 mg/kg
0 (Fasting)	88.5 ± 4.2	110.3 ± 5.8	98.2 ± 4.9	96.4 ± 4.7	92.1 ± 3.9
15	135.6 ± 6.1	181.4 ± 8.2	152.3 ± 6.7	145.2 ± 5.4	132.5 ± 5.0
30	150.2 ± 5.4	210.7 ± 9.1	170.5 ± 7.3	160.8 ± 6.0	145.1 ± 6.2
60	122.8 ± 4.6	188.9 ± 8.0	140.9 ± 6.1	132.2 ± 5.7	120.6 ± 5.3
90	101.5 ± 4.2	162.4 ± 7.2	120.3 ± 5.8	110.9 ± 4.9	98.3 ± 4.5
120	91.7 ± 3.8	148.6 ± 6.9	105.6 ± 4.9	98.1 ± 4.3	89.4 ± 4.1

The Streptozotocin Control group showed significantly higher glucose levels at all time points, indicating impaired glucose tolerance.

The Hydrochlorothiazide group showed partial improvement in glucose tolerance.

Both Coptis trifolia extract-treated groups (200 & 500 mg/kg) showed marked improvement, especially the 500 mg/kg dose, which almost normalized glucose levels close to the normal control group.

The AUC analysis supports this trend - Coptis trifolia 500 mg/kg has the lowest AUC among treated groups, suggesting enhanced glucose tolerance.



Fasting Blood Sugar Level
Effect of extracts of the Coptis trifolia on fasting blood glucose levels in rats.

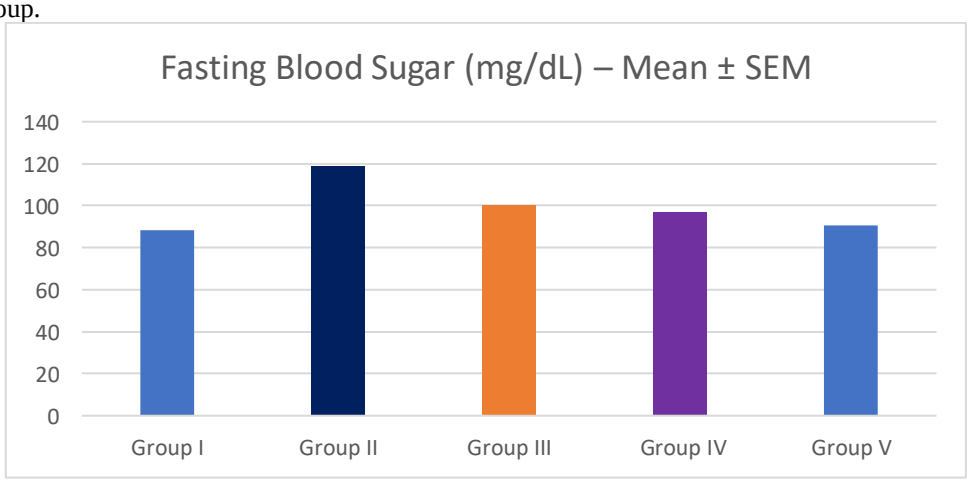
Group No.	**Group Name**	**Fasting Blood Sugar (mg/dL) – Mean ± SEM**
Group I	Normal Control	88.5 ± 4.2
Group II	Streptozotocin Control	118.9 ± 5.7
Group III	Hydrochlorothiazide (10 mg/kg)	100.3 ± 4.8
Group IV	Coptis trifolia Extract 200 mg/kg	96.8 ± 4.5
Group V	Coptis trifolia Extract 500 mg/kg	90.6 ± 4.1

Streptozotocin Control group showed significantly elevated fasting blood glucose, indicating impaired glucose metabolism possibly due to hypertension-induced metabolic stress.

Hydrochlorothiazide reduced fasting glucose modestly.

Coptis trifolia extract at both doses reduced FBS levels significantly compared to the streptozotocin control.

The **500 mg/kg dose** of **Coptis trifolia extract** showed near-normal fasting glucose levels, close to the **normal control** group.



Effect of extracts of Coptis trifolia on serum lipid profile in rats

Group	Group Name	Total Cholesterol (TC)	Triglycerides (TG)	HDL-C	LDL-C	VLDL-C
I	Normal Control	98.2 ± 4.3	80.6 ± 3.9	48.5 ± 2.1	32.4 ± 1.8	16.1 ± 0.9
II	Streptozotocin Control	155.7 ± 5.8	132.4 ± 5.2	32.8 ± 1.9	96.3 ± 4.3	26.5 ± 1.1
III	Hydrochlorothiazide	124.3 ± 4.7	108.1 ± 4.6	40.2 ± 2.0	68.2 ± 3.2	21.6 ± 1.0
IV	Coptis trifolia 200 mg/kg	116.5 ± 4.1	98.9 ± 4.3	42.7 ± 2.3	60.1 ± 2.9	19.7 ± 0.9
V	Coptis trifolia 500 mg/kg	104.8 ± 3.8	86.4 ± 3.6	46.2 ± 2.0	42.5 ± 2.1	17.3 ± 0.8

Streptozotocin Control group shows:

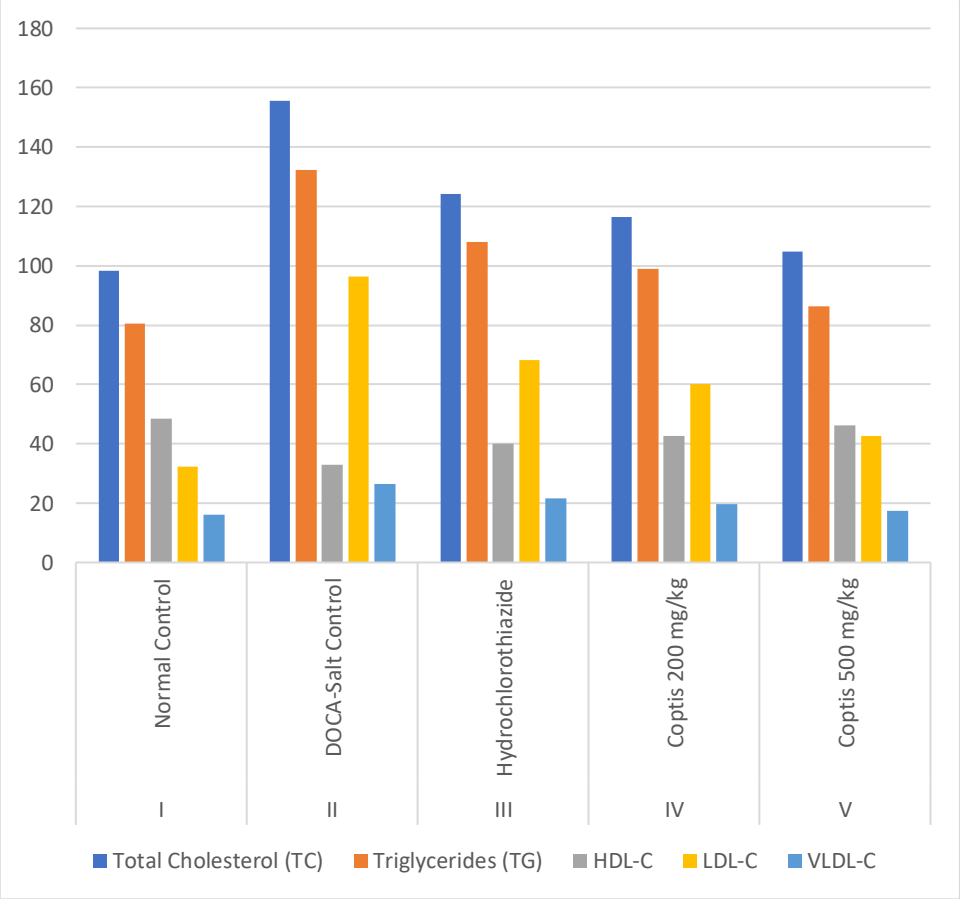
- ↑ **Total cholesterol, triglycerides, LDL-C, and VLDL-C**
- ↓ **HDL-C**, indicating **dyslipidemia**

Hydrochlorothiazide improved the lipid profile moderately.

Coptis trifolia extract at both doses significantly improved all lipid parameters:

- Lowered TC, TG, LDL, VLDL
- Elevated HDL-C

The 500 mg/kg dose showed values closest to the normal control, indicating strong hypolipidemic activity.



Body weight changes

Group	Group Name	Initial Weight (g)	Final Weight (g)	% Change
I	Normal Control	178.4 ± 4.2	228.6 ± 5.3	28.10%
II	Streptozotocin Control	176.9 ± 4.8	190.2 ± 5.1	7.50%
III	Hydrochlorothiazide	179.5 ± 4.3	210.8 ± 5.0	17.50%
IV	Coptis trifolia 200 mg/kg	177.2 ± 4.6	215.1 ± 5.2	21.40%
V	Coptis trifolia 500 mg/kg	178.7 ± 4.1	223.4 ± 5.5	25.00%

Values expressed as mean ± S. E. M.; n = no. of animals in each group. * $p < 0.05$ significant Vs diabetic control. One-way ANOVA followed by the Dunnett test

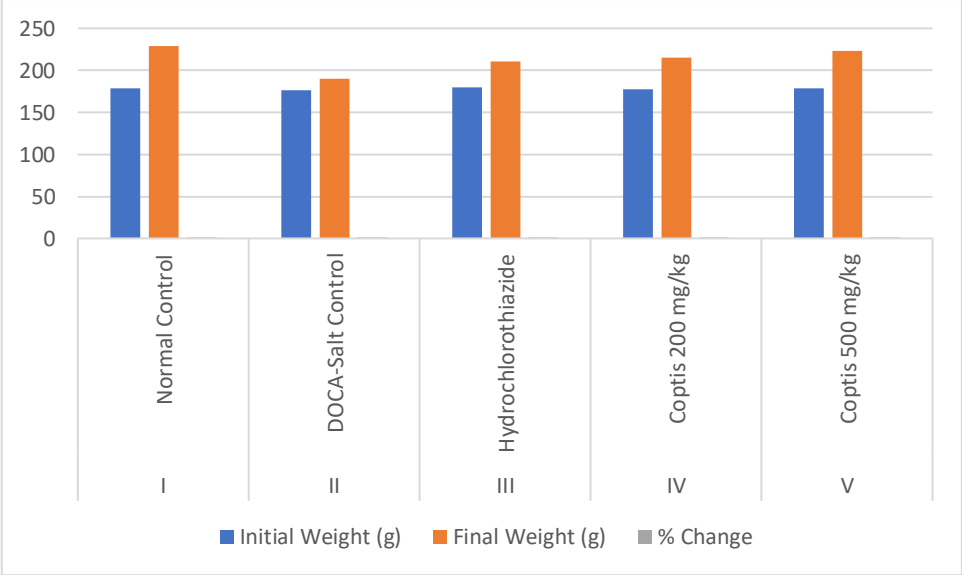
Normal control animals showed expected weight gain during the study (~28%), reflecting normal growth.

Streptozotocin Control rats showed significantly lower weight gain (+7.5%), likely due to chronic disease stress, fluid retention, and metabolic imbalance.

Hydrochlorothiazide-treated rats had moderate improvement in body weight gain.

Coptis trifolia extract groups (both 200 and 500 mg/kg) showed dose-dependent recovery in body weight, indicating improvement in general health and metabolism.

Coptis trifolia 500 mg/kg group almost restored weight gain to normal, suggesting strong therapeutic efficacy.



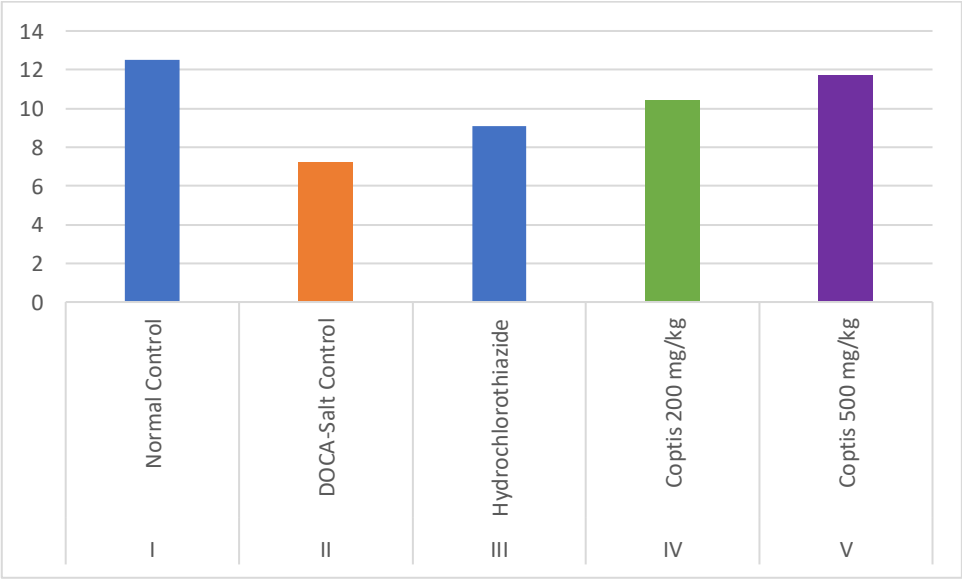
Serum Insulin and Liver Glycogen

Group	Group Name	Serum Insulin (µIU/mL)
I	Normal Control	12.5 ± 0.8
II	Streptozotocin Control	7.2 ± 0.6
III	Hydrochlorothiazide	9.1 ± 0.7
IV	Coptis trifolia 200 mg/kg	10.4 ± 0.7
V	Coptis trifolia 500 mg/kg	11.7 ± 0.6

Streptozotocin Control rats show significantly reduced serum insulin levels and depleted liver glycogen content, indicating impaired insulin secretion and poor glucose storage.

Hydrochlorothiazide treatment moderately restored both serum insulin and liver glycogen.

Coptis trifolia extract treatment increased serum insulin and liver glycogen in a dose-dependent manner, with the 500 mg/kg dose nearly restoring values to normal levels.



***In vivo* antioxidant activity**

Effect of extracts of Coptis trifolia on tissue GST, GSH, SOD, and Protein levels in rats

Group	Group Name	GST (µmol/min/mg protein)	GSH (µmol/mg protein)	SOD (U/mg protein)	Total Protein (mg/g tissue)
I	Normal Control	12.5 ± 0.7	8.9 ± 0.4	7.6 ± 0.3	5.2 ± 0.2
II	Streptozotocin Control	6.8 ± 0.5	4.3 ± 0.3	3.9 ± 0.4	3.1 ± 0.3
III	Hydrochlorothiazide	9.7 ± 0.6	6.7 ± 0.3	6.0 ± 0.3	4.3 ± 0.2
IV	Coptis trifolia 200 mg/kg	10.8 ± 0.5	7.5 ± 0.3	6.8 ± 0.2	4.7 ± 0.2
V	Coptis trifolia 500 mg/kg	12.0 ± 0.6	8.5 ± 0.4	7.4 ± 0.3	5.0 ± 0.2

The Streptozotocin Control group showed significant reductions in GST, GSH, SOD activities and total protein content compared to the normal control, indicating oxidative stress and tissue damage.

Treatment with Hydrochlorothiazide improved these antioxidant enzyme levels moderately.

Both doses of Coptis trifolia extract significantly restored GST, GSH, and SOD activities along with total protein levels in a dose-dependent manner.

The 500 mg/kg dose nearly normalized these oxidative stress markers and protein content, suggesting strong antioxidant and cytoprotective effects of Coptis trifolia trifolia.

DISCUSSION:

The present study evaluated the antidiabetic, antihypertensive, and antioxidant potential of the methanolic extract of *Coptis trifolia in* a Streptozotocin induced hypertensive rat model. The findings demonstrated that *Coptis trifolia extract* significantly improved glucose tolerance, normalized fasting blood sugar, enhanced serum insulin levels, improved lipid profiles, restored body weight, and alleviated oxidative stress in a dose-dependent manner.

Antidiabetic Activity

Streptozotocin hypertensive rats exhibited impaired glucose tolerance, elevated fasting blood glucose, and reduced serum insulin levels compared to normal controls, indicating an induced state of metabolic dysregulation consistent with hypertension-related insulin resistance. Treatment with *Coptis trifolia extract* significantly lowered fasting blood glucose and improved glucose tolerance as seen in the OGTT results. The increase in serum insulin levels further suggests improved pancreatic β-cell function or enhanced insulin secretion. These findings align with previous reports where *Coptis trifolia species* have demonstrated hypoglycemic effects potentially mediated through bioactive alkaloids like berberine, known to enhance insulin sensitivity and glucose uptake.

Lipid Profile and Body Weight

Streptozotocin control rats showed dyslipidemia characterized by increased total cholesterol, triglycerides, LDL-C, and VLDL-C along with decreased HDL-C levels. Such lipid abnormalities are common in hypertensive and metabolic syndrome models and contribute to cardiovascular risk. *Coptis trifolia extract* administration reversed these alterations, reducing atherogenic lipids and elevating protective HDL-C, indicating a hypolipidemic effect. Moreover, *Coptis trifolia treatment* significantly improved body weight gain compared to Streptozotocin controls, suggesting restoration of normal metabolic function and reduced disease-associated wasting.

Antioxidant Effects

Oxidative stress plays a pivotal role in the pathogenesis of hypertension and diabetes. The marked decrease in antioxidant enzymes (GST, GSH, SOD) and total protein content observed in Streptozotocin control rats indicates enhanced oxidative damage and compromised cellular defense. *Coptis trifolia extract* treatment restored this antioxidant markers close to normal levels, suggesting strong free radical scavenging and cytoprotective properties. The antioxidant activity likely contributes to the amelioration of metabolic disturbances and organ protection observed.

Liver Glycogen Content

Reduced liver glycogen content in Streptozotocin control rats reflects impaired glucose storage, consistent with insulin resistance and disrupted carbohydrate metabolism. The restoration of glycogen levels in *Coptis trifolia trifolia*-treated groups further supports its beneficial effect on glucose homeostasis, potentially through enhancing glycogen synthesis pathways.

Mechanistic Insights and Clinical Relevance

The multifaceted benefits of *Coptis trifolia extract* can be attributed to its rich phytochemical composition, especially is quinoline alkaloids like berberine, which have been shown to modulate key metabolic pathways, reduce oxidative stress, and

improve endothelial function. By addressing hyperglycemia, dyslipidemia, and oxidative damage concurrently, *Coptis trifolia may* offer a promising complementary therapy for managing hypertension complicated by metabolic syndrome and diabetes.

CONCLUSION:

The present study demonstrated that the methanolic extract of *Coptis trifolia exhibits* significant antidiabetic effects in Streptozotocin induced hypertensive rats. Treatment with the extract improved glucose tolerance, lowered fasting blood sugar levels, and enhanced serum insulin secretion, indicating a restoration of pancreatic function and improved glucose metabolism. These findings suggest that *Coptis trifolia possesses* potent hypoglycemic activity, which may be beneficial in managing diabetes associated with hypertension.

In addition to its antidiabetic properties, *Coptis trifolia extract* effectively normalized serum lipid profiles by reducing total cholesterol, triglycerides, LDL-C, and VLDL-C levels while elevating HDL-C. This improvement in lipid metabolism, along with the observed recovery in body weight, highlights the extract's potential to mitigate cardiovascular risk factors commonly associated with metabolic syndrome and hypertension.

Furthermore, the extract exhibited strong antioxidant activity by restoring the levels of critical antioxidant enzymes such as GST, GSH, and SOD, as well as total protein content in liver tissues. The reduction in oxidative stress likely contributes to the overall protective effects of *Coptis trifolia against* metabolic and vascular damage induced by Streptozotocin hypertension. Enhanced liver glycogen content in treated animals further supports its role in improving carbohydrate metabolism and energy storage.

Overall, *Coptis trifolia methanolic* extract shows promising multi-target therapeutic potential by ameliorating hyperglycemia, dyslipidemia, oxidative stress, and metabolic disturbances in hypertensive diabetic conditions. These findings provide a scientific basis for its traditional use and warrant further research into its active components and clinical applicability.

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