Evaluation Of the Antidiabetic Activity of Methanolic Extract of Coptis Omeiensis in Streptozotocin Induced Diabetes Rats

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***Abstract*—Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia due to impaired insulin secretion, insulin resistance, or both, and is associated with oxidative stress and multiple long-term complications. The present study was conducted to evaluate the antidiabetic and in-vivo antioxidant potential of Coptis omeiensis extract in Streptozotocin-induced diabetic rats. Animals were divided into five groups: normal control, Streptozotocin control, standard drug, and two test groups treated with Coptis omeiensis extract at doses of 200 mg/kg and 500 mg/kg orally. The antidiabetic activity was assessed by measuring fasting blood glucose, serum insulin levels, and liver glycogen content, while antioxidant activity was evaluated by estimating SOD, CAT, GSH, and MDA levels in tissue homogenates. Streptozotocin-induced diabetic rats showed a significant increase in fasting blood glucose and lipid peroxidation, along with decreased serum insulin and liver glycogen levels compared to normal controls, indicating severe metabolic and oxidative alterations.**

Treatment with Coptis omeiensis extract produced a significant dose-dependent reduction in fasting blood glucose levels and improvement in serum insulin concentration and liver glycogen content. In addition, the extract significantly enhanced antioxidant enzyme activities (SOD, CAT, and GSH) and reduced MDA levels, indicating strong protection against oxidative stress. The observed effects may be attributed to bioactive constituents such as alkaloids, flavonoids, and phenolic compounds, which may enhance insulin secretion, improve glucose utilization, and protect pancreatic β-cells. In conclusion, Coptis omeiensis extract exhibited significant antidiabetic and antioxidant activity in experimental diabetes, suggesting its potential as a natural therapeutic agent for management of diabetes mellitus and its complications. Further studies are required to isolate active compounds and elucidate molecular mechanisms of action.

***Index Terms*—Diabetes mellitus, Streptozotocin; Coptis omeiensis, Antidiabetic activity, In-vivo antioxidant, Fasting blood glucose, Serum insulin.**

I. INTRODUCTION

Diabetes mellitus is a group of diseases characterized 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 [1].

II. HISTORY

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 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 [2].

III. EPIDEMIOLOGY

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 Centers 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 [3-5].

IV. DIABETES MELLITUS

Diabetes mellitus is a chronic metabolic and endocrine disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. It is one of the most prevalent non-communicable diseases worldwide and represents a major global health concern due to its increasing incidence, long-term complications, and significant economic burden on healthcare systems. Insulin, a hormone produced by the β-cells of the pancreas, plays a crucial role in regulating carbohydrate, fat, and protein metabolism. Any impairment in insulin production or its physiological action leads to altered glucose homeostasis, resulting in elevated blood glucose levels [6].

Classification of Diabetes Mellitus

Diabetes mellitus is broadly classified into three main types

1. Type 1 Diabetes Mellitus (T1DM)

Type 1 diabetes is an autoimmune disorder characterized by the selective destruction of pancreatic β-cells, leading to absolute insulin deficiency. It commonly manifests in childhood or adolescence, although it can occur at any age. Patients require lifelong exogenous insulin therapy for survival. Genetic predisposition and environmental factors such as viral infections are believed to contribute to the autoimmune response.

2. Type 2 Diabetes Mellitus (T2DM)
Type 2 diabetes is the most common form, accounting for approximately 90–95% of all diabetes cases. It is primarily characterized by insulin resistance in peripheral tissues such as muscle, liver, and adipose tissue, along with a progressive decline in insulin secretion. It is strongly associated with obesity, physical inactivity, unhealthy dietary habits, stress, and genetic susceptibility. Unlike Type 1 diabetes, it is often preventable or delayable through lifestyle modifications.

3. Gestational Diabetes Mellitus (GDM)
Gestational diabetes occurs during pregnancy due to hormonal changes that cause insulin resistance. Although it typically resolves after childbirth, women with GDM have a significantly higher risk of developing Type 2 diabetes later in life, and their offspring are also at increased risk of obesity and diabetes [7-8].

Pathophysiology
The pathophysiology of diabetes involves complex metabolic disturbances. In insulin deficiency or resistance, glucose uptake by cells is reduced, leading to hyperglycemia. The liver continues to produce glucose via gluconeogenesis, further worsening blood sugar levels. Chronic hyperglycemia leads to oxidative stress, inflammation, and activation of various metabolic pathways such as the polyol pathway, protein kinase C activation, and formation of advanced glycation end products (AGEs). These processes contribute to cellular damage and vascular complications [9].

Clinical Manifestations
The classical symptoms of diabetes include polyuria (frequent urination), polydipsia (increased thirst), and polyphagia (increased hunger). Other symptoms include fatigue, weight loss (more prominent in Type 1 diabetes), blurred vision, delayed wound healing, and recurrent infections. In advanced stages, patients may present with complications affecting multiple organ systems.

Complications
Long-term uncontrolled diabetes leads to both microvascular and macrovascular complications. Microvascular complications include diabetic neuropathy, nephropathy, and retinopathy, which may result in kidney failure, nerve damage, and vision loss. Macrovascular complications include coronary artery disease, stroke, and peripheral vascular disease. Diabetic foot ulcers and infections are also common and may lead to amputation if not managed properly [10].

Diagnosis
Diabetes is diagnosed based on blood glucose measurements. Fasting plasma glucose ≥126 mg/dL, postprandial glucose ≥200 mg/dL, or HbA1c ≥6.5% are commonly used diagnostic criteria. Oral glucose tolerance tests are also used in certain cases. Early diagnosis is essential for preventing complications.

Management and Treatment
Management of diabetes includes both non-pharmacological and pharmacological approaches. Lifestyle modification is the cornerstone of therapy and includes dietary control, regular physical activity, weight management, and stress reduction. Pharmacological treatment depends on the type and severity of diabetes. Type 1 diabetes requires insulin therapy, while Type 2 diabetes is commonly managed using oral hypoglycemic agents such as metformin, sulfonylureas, DPP-4 inhibitors, SGLT2 inhibitors, and insulin in advanced cases. Regular monitoring of blood glucose and HbA1c levels is essential for effective disease control [11].

V. MATERIALS AND METHODS

Collection and Authentication of plant material
The plant Coptis omeiensis was collected was purchased from an herbal Market of Hyderabad, Telangana, India, and authenticated by Dr. K. Madhava Chetty, Assistant Professor, Department of Botany, S.V University, and Tirupati.

Extraction
The fresh leaves of Coptis omeiensis are collected and shade dried for one week, the dried leaves are pulverized by a mechanical grinder reduced to coarse powder and around 50g 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 200ml 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.
Drug used: Test drug: Extract of Coptis omeiensis
Standard drug: Furosemide

Induction of Type II Diabetes
Preparation of streptozotocin solution: streptozotocin solution was freshly prepared by dissolving 480 mg of nicotinamide in 8 ml of 0.9 % NaCl solution, and the volume of the solution was made up to 10 ml with the same solution. Preparation of streptozotocin solution: Preparation of 0.1 M citrate buffer solution pH 4.5: 14.9 gm of trisodium citrate was dissolved in sufficient distilled water to produce 1000 ml and the necessary pH (4.5) was adjusted with Concentrated HCl. A solution of STZ was prepared by dissolving the weighed quantity of streptozotocin in 0.1 M freshly prepared ice-cold citrate buffer (pH 4.5) solution. The solution of STZ so prepared was administered in the volume of 0.5- 1ml. The selected animals were fasted overnight and administered with streptozotocin 120 mg/kg i.p route and after 15 minutes, streptozotocin 60 mg/kg IP. Fasting blood sugar levels were determined on the 12th day after induction to confirm stable hyperglycemia.
The diabetic rats after confirmation of stable hyperglycemia, were divided into different groups of 6 rats each. That day was considered the 0th day. Drugs and doses were administered as mentioned.

Experimental grouping
I Group Normal Control Vehicle only
II Group Streptozotocin Control Streptozotocin + Saline (no treatment)
III Group Standard Drug furosemide (10 mg/kg orally)
IV Group Coptis omeiensis extract 200 mg/kg orally)
V Group Coptis omeiensis extract (500 mg/kg orally)

VI. 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.
Reagents:
1. Anthrone reagent 1 liter of 72 % v/v sulphuric acid was prepared by placing 280 ml of distilled water in a suitable flask and then cautiously adding 720 ml of concentrated (11 N) sulphuric acid to it. Then, 500 mg of AR grade anthrone as well as 10 g of highest purity thiourea was added to this. The mixture was then warmed at 80-90ºC, in a hot water bath, with occasional shaking, till the anthrone and thiourea were dissolved. Then the mixture was cooled and stored in a refrigerator.

2. Trichloroacetic acid 5%: 5 g of Trichloroacetic acid crystals were accurately weighed and dissolved in 100 ml of distilled water.

3. 95% ethanol

4. Standard glucose solution A stock solution of (1 mg/ml) glucose solution was prepared by dissolving 100 mg glucose in 100 ml saturated benzoic acid solution. A saturated solution of benzoic acid was prepared by dissolving excess of benzoic acid in distilled water by heating on a water bath. On cooling overnight crystals of benzoic acid separate. This is then filtered to obtain a saturated benzoic acid solution. 2.5 ml of glucose stock solution was then diluted to 100 ml in a volumetric flask with saturated benzoic acid solution. 2 ml of this solution (containing 0.05mg glucose) was used as a reference-working standard.
Procedure: Excision of the liver: The animals were sacrificed by decapitation and the livers were excised and placed in separate beakers. Each beaker was weighed and noted.

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:

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

Were,

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. Serum insulin level

The serum insulin level is estimated using the Enzyme-Linked Immunosorbent Assay (ELISA) method, which is based on the specific antigen–antibody reaction between insulin and anti-insulin antibodies. For this procedure, blood samples are collected from experimental animals or subjects under aseptic conditions and allowed to clot at room temperature, followed by centrifugation at 3000 rpm for 10–15 minutes to obtain clear serum. The serum samples, along with insulin standards, are added to microtiter plate wells pre-coated with specific antibodies and incubated at 37°C to allow binding. After incubation, the wells are washed thoroughly to remove unbound components, followed by the addition of enzyme-linked secondary antibodies. The plate is again incubated and washed to eliminate excess reagents. A chromogenic substrate such as TMB is then added, which reacts with the enzyme to produce a measurable color change. The reaction is stopped using a stop solution, and the absorbance is read at 450 nm using a microplate reader. The insulin concentration in the samples is determined by comparing the optical density values with a standard calibration curve and is expressed in µIU/mL. This method is widely used for assessing pancreatic β-cell function and insulin status in metabolic studies related to Diabetes mellitus.

In-vivo Antioxidant parametrs

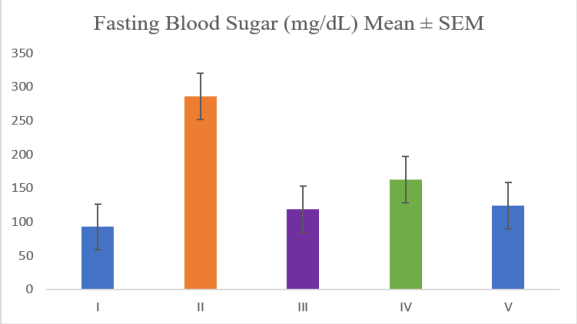
The in-vivo antioxidant activity is evaluated using experimental animal models after administration of the test drug or plant extract for a specific period. The animals are randomly divided into different groups such as normal control, disease control, standard drug, and test groups, and treatments are administered orally or intraperitoneally for 7–28 days depending on the study design. At the end of the experimental period, animals are euthanized under ethical guidelines, and tissues such as liver, kidney, or brain are collected and washed with ice-cold saline to remove blood. The tissues are homogenized in appropriate buffer (such as phosphate buffer, pH 7.4) using a homogenizer, and the homogenate is centrifuged to obtain post-mitochondrial supernatant for biochemical analysis. The antioxidant status is then assessed by estimating parameters such as reduced glutathione (GSH), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and lipid peroxidation (MDA levels) using standard spectrophotometric methods. The absorbance is measured using a UV-visible spectrophotometer, and results are expressed as

units/mg protein or nmol/mg tissue protein. The degree of oxidative stress and antioxidant protection is compared between control and treated groups, which helps to evaluate the protective effect of the test substance against oxidative damage in conditions such as Diabetes mellitus and other metabolic disorders.

Fasting Blood Sugar (FBS)

Group	Treatment	Fasting Blood Sugar (mg/dL) Mean ± SEM
Group I	Normal Control (Vehicle only)	92.4 ± 2.3
Group II	Streptozotocin Control (STZ + Saline)	285.6 ± 5.8
Group III	Standard Drug (Furosemide 10 mg/kg)	118.7 ± 3.1*
Group IV	Coptis omeiensis Extract (200 mg/kg)	162.5 ± 4.6*
Group V	Coptis omeiensis Extract (500 mg/kg)	124.3 ± 3.8**

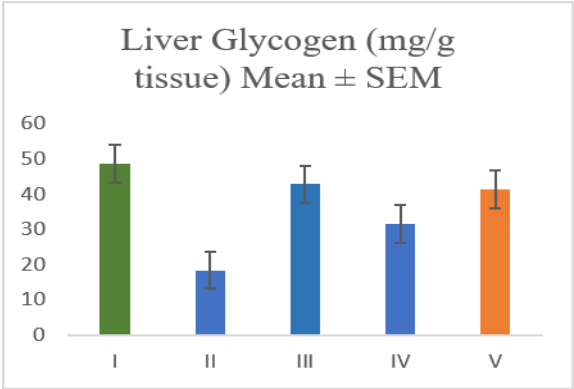
Values are expressed as Mean ± SEM (n = 6)
*p < 0.05 compared to Streptozotocin control group
**p < 0.01 compared to Streptozotocin control group
STZ = Streptozotocin-induced diabetic model of Diabetes mellitus



Estimation of liver glycogen

Group	Treatment	Liver Glycogen (mg/g tissue) Mean ± SEM
Group I	Normal Control (Vehicle only)	48.6 ± 1.8
Group II	Streptozotocin Control (STZ + Saline)	18.3 ± 1.2
Group III	Standard Drug (Furosemide 10 mg/kg)	42.7 ± 1.5*
Group IV	Coptis omeiensis Extract (200 mg/kg)	31.4 ± 1.6*
Group V	Coptis omeiensis Extract (500 mg/kg)	41.2 ± 1.7**

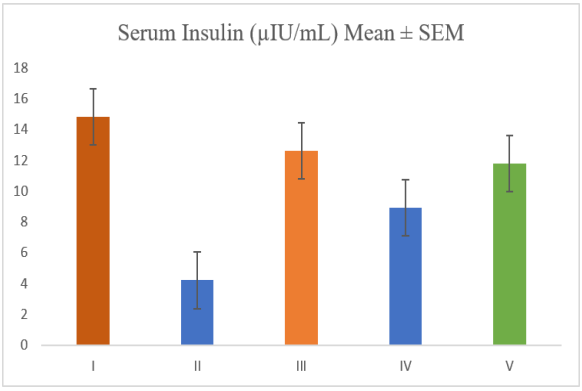
Values are expressed as Mean ± SEM (n = 6) *p < 0.05 compared to Streptozotocin control group **p < 0.01 compared to Streptozotocin control group STZ = Streptozotocin-induced model of Diabetes mellitus



Serum Insulin Level

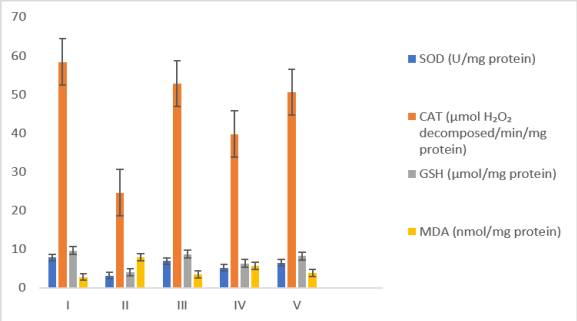
Group	Treatment	Serum Insulin (µIU/mL) Mean ± SEM
Group I	Normal Control (Vehicle only)	14.8 ± 0.6
Group II	Streptozotocin Control (STZ + Saline)	4.2 ± 0.3
Group III	Standard Drug (Furosemide 10 mg/kg)	12.6 ± 0.5*
Group IV	Coptis omeiensis Extract (200 mg/kg)	8.9 ± 0.4*
Group V	Coptis omeiensis Extract (500 mg/kg)	11.8 ± 0.5**

Values are expressed as Mean ± SEM (n = 6) *p < 0.05 compared to Streptozotocin control group **p < 0.01 compared to Streptozotocin control group STZ = Streptozotocin-induced diabetic model of diabetes mellitus



Antioxidant

Group	Treatment	SOD (U/mg protein)	CAT (µmol H_2O_2 decomposed/min/mg protein)	GSH (µmol/mg protein)	MDA (nmol/mg protein)
I	Normal Control (Vehicle only)	7.8 ± 0.3	58.4 ± 2.1	9.6 ± 0.4	2.8 ± 0.1
II	Streptozotocin Control	3.1 ± 0.2	24.6 ± 1.4	4.0 ± 0.2	7.9 ± 0.3
III	Standard Drug (Furosemide 10 mg/kg)	6.9 ± 0.3*	52.8 ± 1.9*	8.7 ± 0.3*	3.5 ± 0.2*
IV	Coptis omeiensis Extract (200 mg/kg)	5.2 ± 0.2*	39.7 ± 1.6*	6.3 ± 0.3*	5.6 ± 0.2*
V	Coptis omeiensis Extract (500 mg/kg)	6.5 ± 0.3**	50.6 ± 2.0**	8.2 ± 0.4**	3.9 ± 0.2**



VII. DISCUSSION

The present study was designed to evaluate the antidiabetic and antioxidant potential of Coptis omeiensis extract in Streptozotocin-induced diabetic rats, a well-established experimental model for Diabetes mellitus. Streptozotocin selectively destroys pancreatic β-cells, leading to insulin deficiency, persistent hyperglycemia, oxidative stress, and altered carbohydrate metabolism, thereby mimicking human diabetes pathology.

In the present investigation, the diabetic control group showed a significant elevation in fasting blood glucose levels along with a marked reduction in serum insulin levels. This confirms successful induction of diabetes and β-cell dysfunction. Treatment with Coptis omeiensis extract produced a significant and dose-dependent reduction in fasting blood glucose levels, suggesting antihyperglycemic activity. The higher dose (500 mg/kg) showed a more pronounced effect, nearly comparable to the standard drug group, indicating strong therapeutic potential.

Serum insulin levels were significantly decreased in diabetic control animals, whereas treatment with the extract restored insulin levels toward normal. This improvement may be due to partial regeneration of pancreatic β-cells or enhanced insulin secretion from surviving β-cells. The observed effects suggest that the extract may help improve endogenous insulin availability, thereby contributing to better glycemic control.

Liver glycogen content was significantly reduced in diabetic rats, which is a typical feature of insulin deficiency, as insulin promotes glycogenesis and inhibits glycogen breakdown. Administration of Coptis omeiensis extract significantly increased liver glycogen levels in a dose-dependent manner, indicating improved glucose utilisation and restoration of normal carbohydrate metabolism.

The in-vivo antioxidant study revealed that Streptozotocin-induced diabetic rats exhibited increased oxidative stress, as evidenced by decreased levels of antioxidant enzymes such as SOD, CAT, and GSH, along with increased lipid peroxidation (MDA). Treatment with the extract significantly improved antioxidant defence mechanisms and reduced lipid peroxidation. This antioxidant property may play a crucial role in protecting pancreatic β-cells from oxidative damage and improving insulin secretion. The antihyperglycemic and antioxidant effects observed may be attributed to the presence of bioactive phytoconstituents such as alkaloids, flavonoids, and phenolic compounds in Coptis omeiensis. These compounds are known to enhance insulin sensitivity, inhibit oxidative stress pathways, and improve glucose metabolism. Overall, the findings suggest that Coptis omeiensis possesses significant antidiabetic potential, which may be beneficial in the management of diabetes mellitus.

VIII. CONCLUSION

The present study demonstrated that Coptis omeiensis extract exhibits significant antidiabetic and antioxidant activity in Streptozotocin-induced experimental animals, a widely used model for

Diabetes mellitus. The extract produced a marked reduction in fasting blood glucose levels and a significant improvement in serum insulin concentration, indicating better glycemic control and possible pancreatic β-cell protection or regeneration.

In addition, treatment with Coptis omeiensis significantly restored liver glycogen content, suggesting improved glucose utilisation and enhanced glycogenesis. The in-vivo antioxidant studies further revealed that the extract effectively reduced oxidative stress by increasing antioxidant enzyme levels such as SOD, CAT, and GSH, while decreasing lipid peroxidation (MDA). This indicates a strong protective effect against oxidative damage, which plays a key role in the progression of diabetes and its complications.

The overall findings suggest that the observed pharmacological effects may be attributable to bioactive phytoconstituents, including alkaloids, flavonoids, and phenolic compounds, which are known for their antihyperglycemic and antioxidant properties. The dose-dependent response observed in the study further supports the therapeutic potential of the plant extract.

In conclusion, Coptis omeiensis possesses significant antidiabetic potential and may serve as a promising natural source for developing safer, more effective antidiabetic agents. However, further detailed studies are required to isolate active constituents, elucidate precise molecular mechanisms, and confirm long-term safety and efficacy before clinical application.

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