Ind. J. Pharm. Edu. Res., 2026; 60(3s):s1231-s1239.
https://www.ijper.org

Original Article

Antidiabetic and Hepatoprotective Effects of *Dichrostachys cinerea* Bark Extract in Streptozotocin-Induced Diabetic Rats

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ABSTRACT

Background: Diabetes mellitus is a complex metabolic disorder characterized by chronic hyperglycemia resulting from impaired insulin secretion, insulin action, or both. Persistent hyperglycemia is often accompanied by secondary complications, including hepatic dysfunction. Plant-based therapies continue to attract interest as potential adjuncts for managing diabetes and its associated complications. **Objectives:** The present study aimed to evaluate the antidiabetic and hepatoprotective effects of *Dichrostachys cinerea* bark extract in a Streptozotocin (STZ)-induced diabetic rat model. **Materials and Methods:** Diabetes was induced in male Wistar rats using streptozotocin (60 mg/kg, intraperitoneally). Animals were divided into five groups (n = 6): normal control, diabetic control, standard drug–treated (glibenclamide, 600 µg/kg), and two *D. cinerea* extract-treated groups receiving different oral doses. Following treatment, blood glucose levels and hepatic function markers-including Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Alkaline Phosphatase (ALP), Lactate Dehydrogenase (LDH), and total protein-were assessed to evaluate antidiabetic activity and hepatic status. **Results:** Administration of *D. cinerea* bark extract resulted in a significant reduction in blood glucose levels compared with the diabetic control group. Diabetic rats exhibited elevated hepatic enzyme levels, indicating liver dysfunction, which were markedly ameliorated following extract treatment. The observed biochemical improvements suggest a protective effect of the extract against diabetes-associated hepatic alterations. **Conclusion:** The findings demonstrate that *D. cinerea* bark extract exhibits antidiabetic activity and mitigates diabetes-associated hepatic enzyme disturbances in STZ-induced diabetic rats. These effects support its potential as a complementary therapeutic agent for diabetes-related hepatic complications. However, further studies involving phytochemical characterization, mechanistic investigations, and long-term safety evaluation are required before clinical application.

Keywords: Diabetes, Hepatotoxicity, Streptozotocin, *Dichrostachys cinerea* Bark Extract (DCBE).

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Received: 21-12-2025;
Revised: 03-03-2026;
Accepted: 16-04-2026.

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia and abnormal glucose excretion, resulting from impaired insulin secretion, reduced insulin sensitivity, or both. The global prevalence of diabetes continues to rise due to factors such as population growth, aging, urbanization, obesity, and sedentary lifestyles.[1] Poor glycemic control is frequently complicated by hypoglycemia, which remains a

DOI: 10.5530/ijper.20263929

major challenge in diabetes management, as patient awareness, perception of symptoms, and self-management strategies vary widely across populations and cultural settings.[2] The complex clinical nature of diabetes, often accompanied by multiple comorbidities and polypharmacy, necessitates disease-specific evidence to guide individualized therapeutic decisions, as treatment recommendations may differ significantly from those for non-diabetic populations.[3] Experimental and preclinical studies further suggest that obesity-associated metabolic and inflammatory alterations can influence glucose homeostasis, organ function, and hypoglycemia risk in susceptible subgroups.[4] Uncontrolled diabetes substantially impairs quality of life and leads to progressive damage of vital organs, including the liver, kidneys, heart, nerves, and blood vessels. The incidence of hypoglycemia among diabetic individuals shows wide variability depending on diabetes type and population, with markedly higher rates reported in type 1 diabetes compared to type 2 diabetes.[5] Epidemiological data indicate no significant sex-based differences in diabetes prevalence among adults aged 20-49 years, and recent estimates report that diabetes affected approximately 463 million adults worldwide in 2019, with projections reaching 700 million by 2045, underscoring its growing public health burden.[6,7]

Diabetes mellitus represents a major global epidemic, accounting for substantial adult mortality and affecting nearly 10% of the world's population, with complications that markedly reduce quality of life and impose a significant burden on healthcare systems. Advancing biomedical research to improve diabetes management is therefore essential, and due to ethical and practical limitations in human studies, experimental animal models play a crucial role in elucidating disease mechanisms and evaluating potential therapeutic agents.[8] The Wistar rat is widely employed in diabetes research because chemically induced diabetes in this model closely resembles key aspects of human diabetic pathophysiology, making it suitable for testing antidiabetic interventions.[9] Streptozotocin (STZ) is one of the most commonly used diabetogenic agents for inducing experimental diabetes, as it selectively targets pancreatic β-cells through its cytotoxic nitrosourea moiety, leading to impaired insulin secretion and irreversible β-cell damage.[10,11] Beyond its diabetogenic action, STZ has been reported to induce multisystem toxicity, including hepatic and renal injury, highlighting its relevance for studying diabetes-associated organ dysfunction.[8,11]

Improving existing antidiabetic therapies to achieve safer, more affordable, and widely accessible treatment options remains a major priority.[12] In this context, increasing attention has been directed toward medicinal plants, whose therapeutic potential in diabetes management is widely recognized. The World Health Organization encourages the investigation and use of traditional herbal remedies, citing their effectiveness, lower toxicity, and reduced adverse effects compared with synthetic drugs.[13] Numerous plant-derived products have demonstrated antidiabetic activity, largely due to their rich content of bioactive compounds such as flavonoids, alkaloids, terpenoids, glycosides, and polyphenols, which may contribute to glycemic regulation, lipid metabolism, and oxidative stress reduction.[7,14] These phytochemicals are known to act through multiple mechanisms, including enhancement of insulin sensitivity, inhibition of carbohydrate-digesting enzymes, and antioxidant activity.[7] *Dichrostachys cinerea* (family: Fabaceae), commonly known as sickle bush or Chinese lantern tree, is a medicinal plant traditionally used in indigenous systems of medicine and reported to possess diverse biological activities, including antioxidant and antimicrobial properties.[15] However, despite its broad ethnomedicinal relevance, scientific evidence supporting its antidiabetic and organ-protective effects remains limited, warranting further investigation.

The medium-sized tree *D. cinerea*, also called Mimosae, is found in forests in Africa, Australia, India, and some regions of Southeast Asia. In addition to their aphrodisiac qualities, the bark and leaves of *D. cinerea* are traditionally thought to be the main sources of compounds used in medicine for a variety of conditions, such as jaundice, rheumatism, inflammation, fever, body aches, asthma, chest issues, ulcers, toothaches, wounds, and eye diseases. The plant includes flavonoids, tannins, triterpenes, saponins, and steroids, according to preliminary phytochemical investigations of the plant, especially of its leaves and bark extracts. Furthermore, aliphatics and triterpenoids make up *D. cinerea's* heartwood. It is also widely known that the tannins extracted from *D. cinerea* have antimicrobial properties.[16] The bark of the plant is used to cure a variety of conditions, including arthritis, syphilis, gonorrhoea, leprosy, toothache, elephantiasis, vermifuge, snakebite, and dysentery. abortifacients, which are also used as pain relievers and for pulmonary issues.[15]

Thus, the present study investigates the effect of *D. cinerea* bark extract in streptozotocin-induced diabetic rats, with particular emphasis on its influence on hepatic function. The study specifically evaluates changes in hepatic enzyme markers and total protein levels associated with diabetes-related liver alterations. The findings may contribute to the growing evidence supporting the potential of plant-derived agents as complementary approaches for the management of diabetes and its associated hepatic complications.

MATERIALS AND METHODS

Materials

Prior to the start of the study, all chemicals, kits, equipment, and reagents of a high analytical category were prepared and made available by purchasing them from reliable commercial sources.

Animals

Following the approval of the Institutional Animal Ethical Committee, mature and healthy Wistar rats were procured, each weighing between 180 and 200 g, from the Animal Facility. The rats were acclimated in a meticulously sanitized laboratory setting, where ambient conditions were carefully regulated to maintain a temperature of 25°C and a relative humidity of 55% over a period of one week. A 12-hr light and dark cycle was implemented during this adaptation phase. Throughout the acclimatization process, the rats were provided with a standard diet and had continuous access to fresh water. Bedding was replaced daily, while the cages were cleaned and replaced every three days. All experimental procedures conducted in the study received prior approval from the ethics committee, and every effort was made to handle the animals with the highest level of care and respect.[17] The experimental protocol entitled "Investigating the potential of *D. cinerea* bark extract on diabetes and diabetes-related complications in a chemically induced diabetic animal model" was reviewed and approved by the Institutional Animal Ethics Committee (IAEC) of Muthayammal Centre for Advanced Research, Muthayammal College of Arts and Science, Rasipuram, Namakkal, Tamil Nadu, India (Approval No: MCAS/IAEC/03/05, dated 04.04.2024). All procedures were conducted in accordance with the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India.

Diabetes Induction

Diabetes was induced in experimental animals using the medication Streptozotocin (STZ). A dose of 60 mg/kg of STZ was administered intraperitoneally in a 0.1M citrate buffer at pH 4.4. To confirm the onset of diabetes, blood glucose levels were measured after 7 days of STZ treatment. In this study, rats with blood glucose levels exceeding 11 mmol/L were selected for further investigation.[17,18]

Experimental Design

In this study, thirty male Wistar rats were randomly assigned into five groups (n = 6 per group) as follows: Group I (Normal Control): Received 0.5% Dimethyl Sulfoxide (DMSO) orally once daily. Group II (Diabetic Control): Diabetes was induced by a single intraperitoneal (i.p.) injection of streptozotocin (STZ; 60 mg/kg body weight), followed by oral administration of 0.5% DMSO once daily. Group III (DCBE 150 mg/kg): Diabetes was induced by STZ (60 mg/kg body weight, i.p.), and rats were treated with *D. cinerea* Bark Extract (DCBE) at 150 mg/kg body weight, administered orally once daily. Group IV (DCBE 300 mg/kg): Diabetes was induced by STZ (60 mg/kg body weight, i.p.), and rats were treated with DCBE at 300 mg/kg body weight, administered orally once daily. Group V (Standard Control): Diabetes was induced by STZ (60 mg/kg body weight, i.p.), and rats were treated with Glibenclamide at 600 µg/kg body weight, administered orally once daily. DCBE and Glibenclamide were prepared in 0.5% DMSO and administered in the morning for 60 consecutive days. At the end of the experimental period, animals were euthanized, blood samples were collected for biochemical analysis, and liver tissues were excised for histopathological examination.[19]

Estimation of fasting blood glucose and body weight of the animals

Body weight of the experimental animals was recorded at baseline and at the end of the treatment period. Fasting Blood Glucose (FBG) levels were measured after an overnight fast at baseline (day 0) and on 60 days of the experimental period. Blood samples were collected from the orbital sinus, and glucose levels were estimated using a commercial glucose estimation kit according to the manufacturer's instructions. The glucose measurement kit was used to estimate blood glucose levels from carefully collected orbital sinus blood samples.[20,21]

Analysis of food intake, and water intake of STZ-provoked animals

During the course of the study, for each group of rats, routine assessments of their food and water intake were carried out. Following a fasting period of 60 days, all rats were humanely euthanized under appropriate anesthesia, utilizing 24 mg/kg body weight of ketamine administered intramuscularly. Blood samples were collected in both anticoagulated and non-anticoagulated tubes. Subsequently, the liver was weighed and meticulously cleaned with ice-cold saline to eliminate any residual blood. A 10% homogenate was prepared using a 0.1 M Tris-HCl buffer, which was then subjected to centrifugation at 1000 g for 10 min. The isolated supernatants were utilized for tests on various biochemical parameters.[22]

Biochemical analysis

At the conclusion of the experimental period, the animals were euthanized under appropriate anesthesia in accordance with CPCSEA guidelines. Sterile syringes were used to puncture the heart and draw blood, which was then allowed to coagulate. For biochemical studies, the serum was kept at -20°C after being separated by centrifugation at 3000 rpm for 10 min. Total protein and albumin were measured using the biuret and bromocresol green dye-binding methods, respectively.[17]

Relative liver weight

Animals were sacrificed via cervical dislocation after blood was drawn. The liver was carefully removed, cleaned with ice cold saline, blotted dry, and weighed. The following formula was used to compute the relative liver weight:

$$\text{Relative Liver Weight (\%)} = (\text{Liver Weight (g)} / \text{Final Body Weight (g)}) * 100$$

Estimation of Total Serum Protein

1 mL of the Biuret reagent was combined with 0.1 mL of the serum sample. For 15 min, the mixture was allowed to incubate at room temperature. The absorbance at 540 nm was determined using a spectrophotometer. A standard calibration curve based on Bovine Serum Albumin (BSA) was used to estimate the concentration of protein.

Estimation of Serum Albumin

1 mL of BCG reagent was blended with 0.1 mL of serum. At room temperature, the absorbance was determined at 630 nm after 10 min of incubation. A standard curve was used to calculate albumin concentration.

Estimation of Serum Globulin and A/G Ratio

The globulin level was estimated using the below formula:

$$\text{Globulin (g/dL)} = \text{Total Protein (g/dL)} - \text{Albumin (g/dL)}$$

Once globulin is estimated, the ratio of Albumin to Globulin is estimated using the below given formula:

$$\text{A/G Ratio} = \text{Albumin level / Globulin level}$$

Estimation of Hepatic Enzymes

At the conclusion of the experimental period, the animals were carefully anesthetized, and their livers were swiftly extracted to ensure optimal integrity. Each liver was meticulously rinsed in chilled normal saline, blotted dry, and weighed to guarantee precise measurements. Subsequently, a premium glass-Teflon homogenizer was used to homogenize the liver tissue in ice-cold 0.1 M phosphate buffer (pH 7.4), resulting in a concentrated 10% (w/v) liver homogenate. The homogenate was subjected to centrifugation at 10,000 rpm for a duration of 15 min at a temperature of 4°C. This procedure facilitated the collection of a clear supernatant, which is essential for the accurate estimation of liver enzyme levels.

Effect of DCBE on Alanine Aminotransferase (ALT)

ALT was calculated using the Reitman and Frankel technique. 0.5 mL of liver supernatant was added to the substrate solution. Incubated at 37°C for 30 min. After adding the DNPH reagent, the mixture was incubated for 20 min. The absorbance was determined at 505 nm following the addition of NaOH.[23]

Effect of DCBE on Aspartate Aminotransferase (AST)

AST was estimated using the Reitman and Frankel method. A 0.5 mL sample of liver supernatant was mixed with an AST-specific substrate solution and incubated at 37°C for 30 min. Next, DNPH reagent was added, and the mixture was incubated for an additional 20 min. After adding NaOH, the absorbance was determined at 505 nm.[23]

Effect of DCBE on Alkaline Phosphatase (ALP)

ALP was calculated using the Kind and King method. Substrate buffer was mixed with 0.1 mL of liver supernatant. Further, it was incubated at 37°C for 30 min. Color intensity was determined at 405 nm.[24]

Effect of DCBE on Lactate Dehydrogenase (LDH)

The LDH was estimated using King's approach. The liver homogenate was treated with lactate and NAD^+. The change in absorbance owing to NADH production was measured at 340 nm.[25]

Statistical Analysis

The statistical analysis was performed using GraphPad Prism version 6.01. Results are expressed as the Mean ± Standard Deviation (SD). To assess differences among the groups, ANOVA was utilized alongside the Tukey *post hoc* test. A *p*-value of less than 0.05 was considered statistically significant for the differences observed between the means.[17]

RESULTS

Impact of DCBE on the Fasting Blood Glucose (FBG) levels in the experimental rats

Figure 2 demonstrates the effect of DCBE on fasting blood glucose levels in the experimental animals on the 7th and 30th days. With the exception of the control group, all experimental groups' blood glucose levels were nearly identical on the seventh day. By the 30th day, all groups' blood glucose levels were compared to that of the STZ-treated control rats (group II). Therefore, it is noteworthy that animals receiving dose-based DCBE treatment (groups III and IV) showed a drop in blood glucose levels. The fasting blood glucose levels of the animals treated with DCBE and those treated with Glibenclamide did not differ significantly at the end of the research.

Impact of DCBE on the bodyweight of experimental rats

Figure 1 illustrates the effects of DCBE on body weight in STZ-treated rats. On the 0th day, all experimental groups exhibited equivalent body weights. Group II, which consisted of animals subjected solely to STZ treatment, demonstrated a substantial decrease in body weight. In comparison, the animals in the STZ-induced groups that received DCBE extract (Groups III and IV) displayed a notable effect on their body weight, corresponding to the dose administered.

Impact of DCBE on food consumption, HOMA-IR, Water intake and insulin level in experimental animals

The impact of DCBE on several key indicators, including insulin levels, food intake, and water consumption, was systematically

assessed in rats with diabetes induced by STZ. It was observed that, in comparison to the control group, the STZ-induced rats exhibited significantly elevated levels of average food intake, and water consumption (Figure 1). However, the administration of DCBE resulted in a marked reduction in food intake, and water consumption among the STZ-induced rats. Furthermore, the substance increased the insulin level in animals treated with DCBE as opposed to rats exposed to STZ (Figure 2). Glibenclamide treatment had results that were similar to DCBE.

Impact of DCBE on the liver weight (g), and serum protein levels in the experimental rats

Figure 3 illustrates that diabetic rats exhibited significantly lower levels of total protein, albumin, globulin, and the albumin/globulin ratio in comparison to control rats, alongside an increase in liver weight. Following treatment with DCBE, the measured levels of total protein, albumin, globulin, and the albumin/globulin ratio showed a notable return towards normal values. The protective effect of DCBE was particularly prominent regarding total protein levels when contrasted with the positive control, GB. Furthermore, the administration of DCBE led to a significant reduction in liver weight, and at a dosage of 300 mg/kg, measurements approached those of the positive Control Group (GB). In summary, the administration of DCBE in diabetic rats effectively reversed these biochemical alterations.

Impact of DCBE on the hepatic markers level in the experimental rats

Figure 4 depicts the effect of DCBE on liver enzymes like ALT, AST, ALP, and LDH in STZ-treated rats. When compared to control rats, diabetic rats had substantially greater hepatic marker activity. Biomarkers like ALT, AST, ALP, and LDH levels were significantly lower in the DCBE-treated diabetic group, and these levels varied more with larger doses of the DCBE extract. This was similar to the positive control GB, in which the rats' liver enzyme levels were significantly lowered. Consequently, when compared to diabetic control rats, DCBE treatment significantly restored the aforementioned abnormalities in diabetic rats.

DISCUSSION

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia resulting from impaired insulin secretion and/or insulin action, leading to disturbances in carbohydrate, lipid, and protein metabolism. It is one of the most prevalent endocrine disorders worldwide and represents a major global health challenge.[26,27] In the present study, Streptozotocin (STZ) administration successfully induced diabetes in rats, as evidenced by persistent hyperglycemia, reduced body weight, altered insulin levels, and impaired liver function markers. These findings are consistent with previous reports describing the diabetogenic action of STZ through selective destruction of pancreatic β-cells.[11,28]

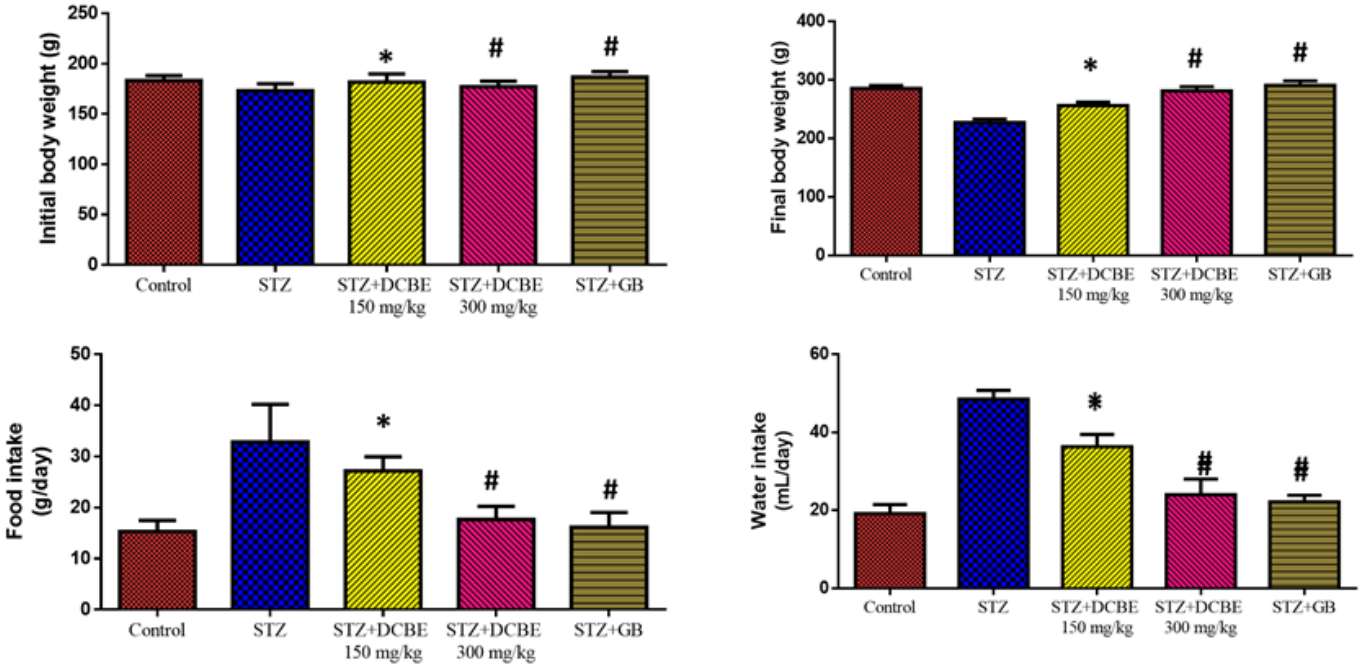

Figure 1: Effect of DCBE on the initial and final body weight of experimental rats. (a) Initial body weight, (b) Final body weight, (c) Food intake, (d) Water Intake. Data was provided as the Mean ± SD of three distinct values. Tukey's *post hoc* test and one-way ANOVA were used to statistically evaluate all of the test results. '*' denotes $p < 0.05$ in comparison to control. "#" means that the difference with DN-initiated rats is less than 0.01.

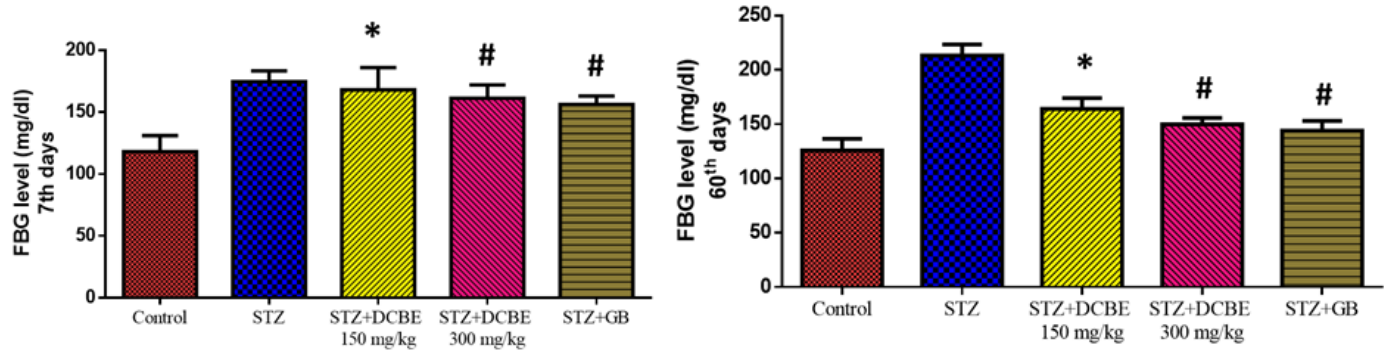

Figure 2: Effect of DCBE on the Fasting Blood Glucose (FBG) level in the experimental rats. (a) 7th days, (b) 60th days. The data are presented as the Mean ± Standard Deviation (SD) of three individual measurements. All results were statistically analyzed using one-way ANOVA followed by Tukey's *post hoc* test. '*' indicates a *p*-value of less than 0.05 compared to the control group, while '#' indicates a *p*-value of less than 0.01 compared to the rats initiated with diabetes.

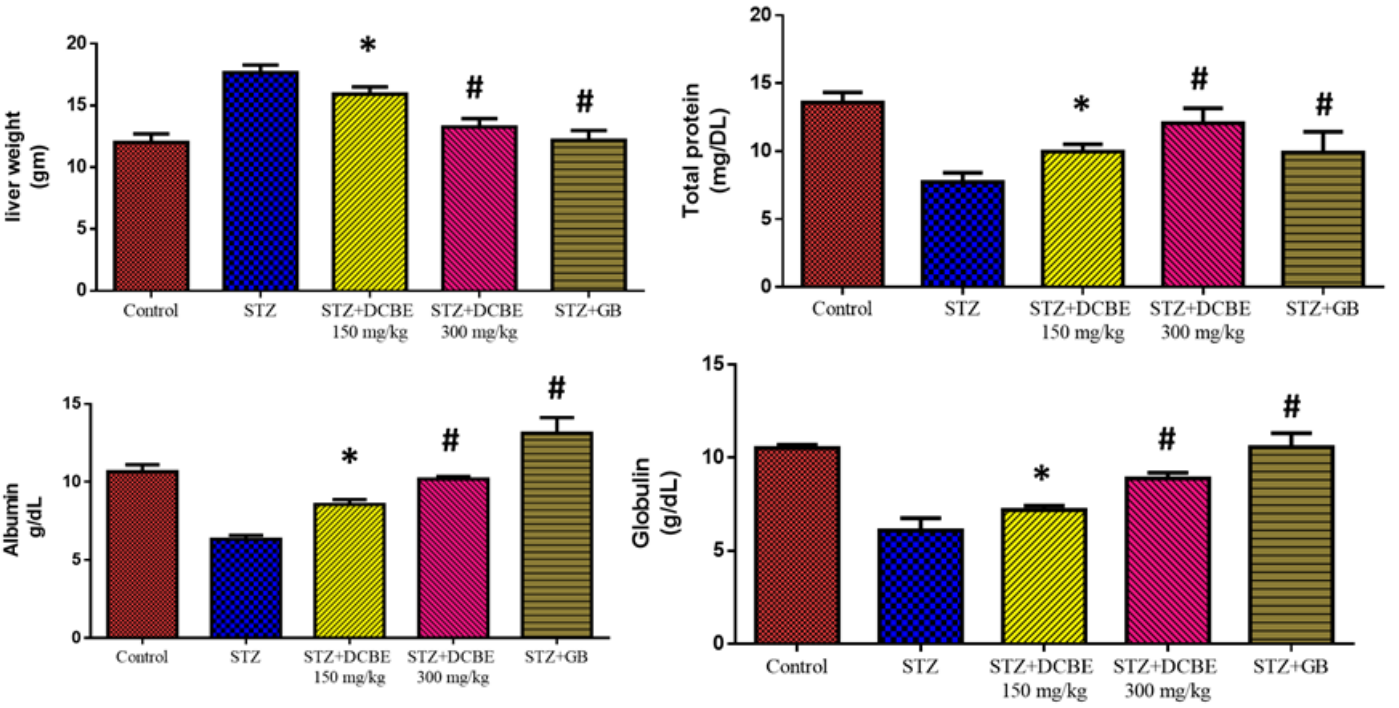

Figure 3: Effect of DCBE on the liver weight (g), Total protein, Total albumin, and Total globulin level in the experimental rats.Three distinct values were presented as the Mean ± SD. A one-way ANOVA and Tukey's *post hoc* test were used to statistically evaluate each result. $p < 0.05$ when compared to control is indicated by a "*." '#' denotes a *p*-value < 0.01 in comparison to rats that were DN-initiated.

STZ-induced diabetic rats exhibited a significant elevation in fasting blood glucose levels compared with normal controls. This hyperglycemic state is attributed to β-cell damage and reduced insulin secretion following STZ entry into pancreatic cells via the GLUT2 transporter.[6,28] Treatment with *D. cinerea* Bark Extract (DCBE), particularly at a dose of 300 mg/kg body weight, significantly reduced fasting blood glucose levels over the treatment period. These results indicate that DCBE possesses notable antihyperglycemic activity, in agreement with earlier studies reporting antidiabetic effects of plant-derived extracts.[29]

Loss of body weight observed in diabetic control rats is a well-documented consequence of insulin deficiency and increased protein and lipid catabolism driven by gluconeogenesis and glycogenolysis.[30] In the present study, DCBE-treated diabetic rats showed a significant improvement in body weight compared to untreated diabetic rats. This improvement may be associated with better glycemic control and enhanced metabolic utilization of nutrients. Similarly, diabetic rats exhibited increased food and water intake, reflecting polyphagia and polydipsia due to impaired glucose utilization and excessive urinary glucose loss. DCBE treatment significantly normalized these parameters in a dose-dependent manner, indicating improved metabolic regulation.[31]

Insulin levels were markedly reduced in STZ-induced diabetic rats, confirming pancreatic β-cell dysfunction. DCBE administration significantly increased insulin levels, particularly at the higher

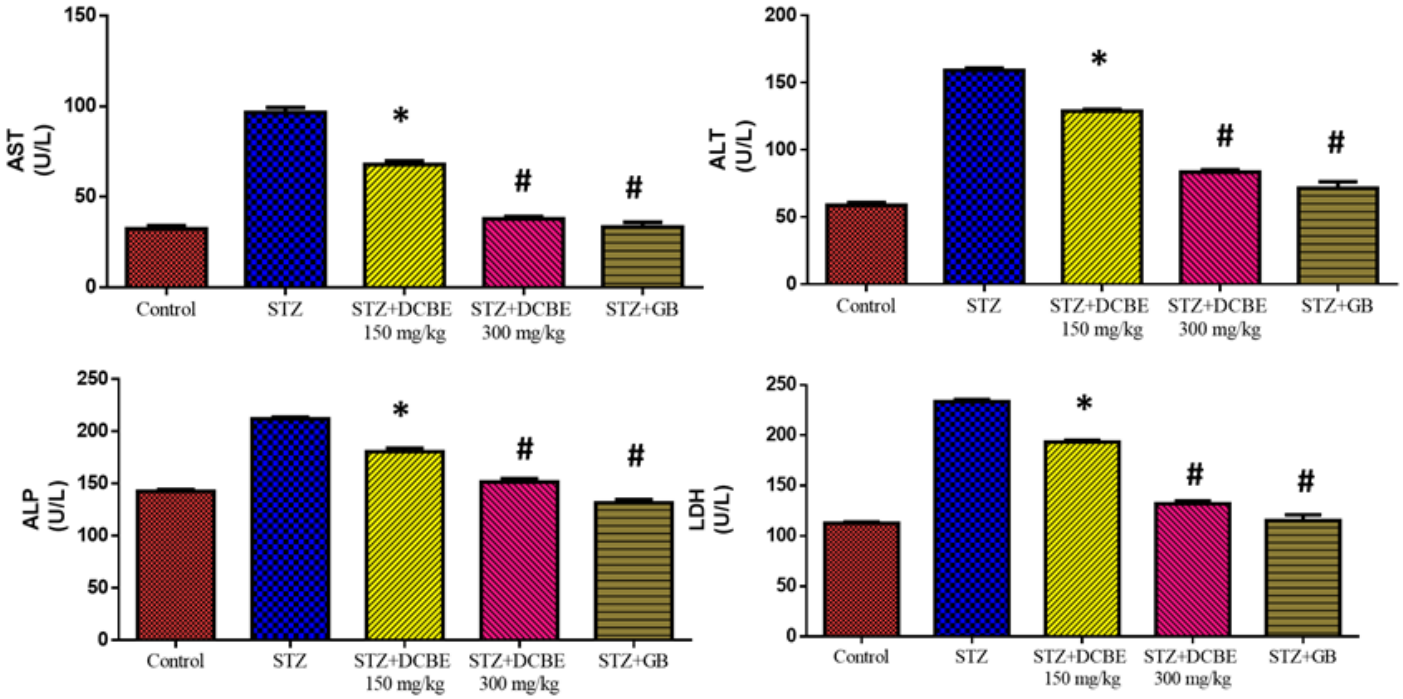

Figure 4: Effect of DCBE on the hepatic marker (Alanine Transaminase (ALT), AspartateTransaminase (AST), ALP, and LDH) levels in the liver tissue homogenates of the experimental rats. The data were presented as three separate values' Mean ± SD. All findings were statistically evaluated using Tukey's *post hoc* test and one-way ANOVA. In comparison to control, a "*" denotes a *p*-value < 0.05. Rats that were DN-initiated showed a *p*-value of less than 0.01 and indicated as #.

dose, suggesting partial restoration of insulin secretion or protection of residual β-cell function. The improvement in insulin levels observed in DCBE-treated groups is consistent with reports that certain phytoconstituents may support pancreatic function.[32] Diabetes is frequently associated with hepatic dysfunction due to altered carbohydrate and protein metabolism.[33] In the present study, STZ-induced diabetic rats showed significant elevations in serum liver enzymes (AST, ALT, ALP, and LDH), along with reduced total protein levels, indicating hepatocellular damage and impaired protein synthesis.[34] Treatment with DCBE significantly restored liver enzyme activities and protein levels toward normal values, suggesting a hepatoprotective effect. The normalization of these biochemical markers indicates reduced hepatic injury and improved liver function in DCBE-treated diabetic rats.[14,17]

Overall, the findings of this study demonstrate that DCBE exerts significant antidiabetic and hepatoprotective effects in STZ-induced diabetic rats, as evidenced by improved glycemic control, enhanced insulin levels, restoration of body weight, normalization of liver enzymes, and recovery of protein metabolism. While these results are promising, further mechanistic and molecular studies are required to confirm the pathways involved and to establish the therapeutic potential of DCBE in diabetes-associated hepatic dysfunction.

CONCLUSION

The findings of this study strongly indicate that DCBE possesses remarkable anti-diabetic properties in STZ-induced rats. Moreover, its potential as a hepatoprotective agent highlights its promise as a compelling alternative to conventional diabetes treatments. With its glycemic benefits and a significantly lower risk of side effects compared to synthetic medications, DCBE stands out as a safer option that positively influences liver enzymes. In addition, delving into the molecular aspects of *D. cinerea* through advanced biotechnological methods offers a unique opportunity to illuminate the intricate interactions between its bioactive compounds and human biological systems. This exploration could unlock groundbreaking therapeutic agents, revealing innovative drug candidates that are both powerful and safe. Such discoveries have the potential to transform diabetes treatment, leading to more precise therapies with minimized side effects, ultimately enhancing patient well-being and quality of life.

ACKNOWLEDGEMENT

DST-FIST No.SR/FST/College-2017/12(C), Ministry of Science and Technology, India for financial support.

ABBREVIATIONS

ALP: Alkaline Phosphatase; **ALT:** Alanine Aminotransferase; **ANOVA:** Analysis of Variance; **AST:** Aspartate Aminotransferase; **DCBE:** Dichrostachys cinerea bark extract;

DNPH: 2,4-Dinitrophenylhydrazine; **ELISA:** Enzyme-Linked Immunosorbent Assay; **GB:** Glibenclamide; **GLUT2:** Glucose Transporter 2; **HOMA-IR:** Homeostasis Model Assessment of Insulin Resistance; LDH**:** Lactate Dehydrogenase; NAD**:** Nicotinamide Adenine Dinucleotide; NADH**:** Nicotinamide Adenine Dinucleotide, reduced form; NaOH**:** Sodium Hydroxide; ROS**:** Reactive Oxygen Species; **SD:** Standard Deviation; STZ**:** Streptozotocin; T1D**:** Type 1 Diabetes; T2D**:** Type 2 Diabetes; WHO**:** World Health Organization.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICAL APPROVAL

The experimental protocol entitled "Investigating the potential of *Dichrostachys cinerea* bark extract on diabetes and diabetes-related complications in a chemically induced diabetic animal model" was reviewed and approved by the Institutional Animal Ethics Committee (IAEC) of Muthayammal Centre for Advanced Research, Muthayammal College of Arts and Science, Rasipuram, Namakkal, Tamil Nadu, India (Approval No: MCAS/IAEC/03/05, dated 04.04.2024). All procedures were conducted in accordance with the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India.

SUMMARY

Current evidence suggests that DCBE has the ability to improve diabetes and related complications, particularly the accompanying hepatotoxicity. Clinical investigations are needed to corroborate this.

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Cite this article: Abudoleh SM, Abualassal Q, Abudayeh ZH, Ahmed ZAGA, Hussein-Al-Ali SH, Ramalingam K, *et al*. Antidiabetic and Hepatoprotective Effects of *Dichrostachys cinerea* Bark Extract in Streptozotocin-Induced Diabetic Rats. Indian J of Pharmaceutical Education and Research. 2026;60(3s):s1231-s1239.