IJPSR (2021), Volume 12, Issue 5 (Research Article)

INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH

Received on 30 April 2020; received in revised form, 05 September 2020; accepted, 15 September 2020; published 01 May 2021

ANTIHYPERGLYCAEMIC AND ANTIOXIDANT ACTIVITY OF METHANOLIC EXTRACT OF *CAPPARIS DECIDUA* (FORSSK.) EDGEW. IN STREPTOZOTOCIN-INDUCED DIABETIC RATS

C. Mathur [*1], M. Soni [2] and R. S. Gupta [1]

Reproductive Physiology Section [1], Department of Zoology, University of Rajasthan, Jaipur - 302004, Rajasthan, India.
School of Engineering and Technology [2], Department of Biotechnology, SADTM Campus, Jaipur National University, Jagatpura, Jaipur - 302017, Rajasthan, India.

Keywords:

Capparis decidua, Antidiabetic, Antioxidant, Streptozotocin

Correspondence to Author:
Charuta Mathur

Research Scholar,
Department of Zoology,
University of Rajasthan, Jaipur - 302004, Rajasthan, India.

E-mail: mathur.charu88@gmail.com

ABSTRACT: The present study evaluates the antihyperglycaemic and antioxidant activity of the methanolic extract of *Capparis decidua* (CDMtE) fruits in streptozotocin (STZ) induced diabetic rats. Streptozotocin (50mg/kg body weight) was injected intraperitoneally in rats to induce diabetes. The STZ-induced diabetic rats were treated with CDMtE (200 mg/kg body weight/day) for seven days. Biochemical analysis, toxicity studies, and histopathological studies were carried out to assess the antihyperglycaemic and antioxidant effects. Treatment with CDMtE (200 mg/kg b.wt/day) caused a significant decrease in fasting blood glucose and improvement in the serum insulin level of the diabetic rats. Antioxidant enzymes SOD and GSH activity were increased, and LPO levels of diabetic rats decreased after oral administration of CDMtE (200 mg/kg body weight/day). Histopathological studies showed significant recovery in liver architecture after CDMtE administration. Histopathological results suggest the antihyperglycaemic and antioxidant ability of CDMtE. This study supports the therapeutic potential of *C.decidua* fruits in the treatment of diabetes.

INTRODUCTION: Diabetes mellitus is a complex metabolic disease in which there may be either a defect in insulin secretion or the body is not able to respond to insulin, resulting in hyperglycemia [1]. According to IDF (International Diabetes Federation) currently, there are 415 million people suffering from diabetes mellitus globally and are estimated to rise by 225 million by 2040 [2].

QUICK RESPONSE CODE

DOI: 10.13040/IJPSR.0975-8232.12(5).2700-06

This article can be accessed online on www.ijpsr.com

DOI link: http://dx.doi.org/10.13040/IJPSR.0975-8232.12(5).2700-06

Hyperglycaemia is the major factor for the onset and progress of diabetic complications, mainly by producing oxidative stress [3]. In diabetes, besides hyperglycaemia, abnormalities in lipid level, which is directly correlated with accelerated atherosclerosis and subsequent cardiovascular diseases are one of the major causes of death in the world. It has been found that chronic hyperglycaemia is associated with the increased production of free radicals. Several hypotheses have been proposed for this genesis that includes oxidation of glucose, a constant increase in the formation of glucose-derived AGEs, and degradation of glycated protein.

In low-income countries where the resources are limited, traditional medicines are the best source

for the management of diabetes mellitus [4]. Despite the availability of novel antidiabetic drugs, diabetes continues to be one of the major medical problems. All oral hyperglycaemic agents are accompanied by several serious, undesirable side effects like hypoglycaemia, gain in body weight, gastro-intestinal disorders, lactic acidosis, and body fluid retention [5]. Therefore, the management and treatment of diabetes remain a big challenge for both scientific fraternity and society. Hyperglycaemia-induced oxidative stress can be treated and managed by natural products of medicinal plants [6].

Capparis decidua (family Capparaceae) commonly known as Kair, folk plant is cultivated abundantly in the "Thar desert" of India [7]. Flavanoids, alkaloids, phenols, sterols, and glycosides are abundant in this folk plant. This plant has been used in the treatment of several diseases, as reported in the Unani medicine system. The plant used in the treatment of various conditions like flatulence, tumor, respiratory disorders, inflammation, skin diseases, and as a diuretic, and antihelminthic, antifungal [8]. Though the effect of *Capparis decidua* on blood sugar, hemoglobin, and lipids are well-known among traditional healers, there is a lack of well-conducted study to weigh up of its medicinal properties. Therefore, this research study was designed to assess the effect, efficacy, and potential benefits of the methanolic extract of the plant *Capparis decidua* as a potent anti-hyperglycaemic agent.

MATERIALS AND METHODS:

Drugs and Chemicals: Streptozotocin (STZ) was purchased from Sigma-Aldrich, St Louis, MO. Glucose kits based on the glucose oxidase method were purchased from Ecopak, Accurex Biomedical Pvt. Ltd, Mumbai. Other chemicals and kits were obtained from HiMedia (Mumbai, India) and SD Fine Chemicals Limited (Mumbai, India).

Preparation of Plant Extract: Fruits of *C. decidua* were collected freshly from the local market of Jaipur, India. The authentication of the species was done at the Department of Botany, University of Rajasthan, Jaipur, Rajasthan. A voucher specimen has been provided by the Department of Botany, the University of Rajasthan with reference no. RUBL 211632. The collected fruits were shed dried for four weeks at room temperature. The dried sample was pulverized using a manual grinder. Plant powder (250 g) was soaked in 500 ml of 100% methanol and kept at room temperature for 24 h. The mixture was extracted in a soxhlet apparatus with methanol for 72 h [9]. The extract was filtered, and methanol was distilled off to obtain a dark reddish mass.

Experimental Model: Healthy adult male albino Wistar rats (*Rattus norvegicus*) were used as a model organism for all the experiments. Animals weighed 160g to 180g were used in the experiment and housed in polypropylene cages in 60-70% relative humidity and 12 h light/12 h dark condition, temperature maintained at 24-28^{o}C. Water *ad-libitum* and standard pellet diet were provided to the animals. The experiments were performed as per the ethical guidelines proposed by the Committee for the Purpose of Control and Supervision of experiments on animals (CPCSEA), Ministry of Environment and Forest, Government of India (1678/GO/a/12/CPCSEA Dated 09-01-2013).

Experimental Design: Rats used for the experiments were divided into four groups. Dose 200 mg/kg body weight/day was given for 7 days.

Group I: served as normal control orally received 0.5ml distilled water.

Group II (Diabetic control): STZ-induced untreated diabetic rats (50 mg/kg body weight).

Group III: This group included STZ-induced diabetic rats that were administered with *C. decidua* fruits methanolic extract at 200mg/kg body weight /day.

Group IV: This group included STZ-induced diabetic rats that were administered with glibenclamide at 0.3 mg/kg body weight/day.

Experimental Diabetes Induction: Streptozotocin (50mg/kg. body weight) was prepared in citrate buffer (0.1M, pH 4.5) to induce diabetes in an overnight fasted rat by injecting it intraperitoneally [10]. Glucose solution (5%) was supplied for 24 h to avoid early drug-induced hypoglycaemic mortality to STZ-induced rats [11]. Fasting blood glucose was measured to confirm diabetes in STZ-injected rats 72 h after injection with STZ.

Administration of Crude Plant Extract: Methanolic extract of *C. decidua* (CDMtE) and standard drug glibenclamide were suspended in-vehicle solution (distilled water, 0.5ml) and administered via oral route by force-feeding for 7 days daily to the rats belonging to respective groups. Feeding to the rats was stopped 30 min before and after the treatment. Only vehicle solution was provided to control (group I) and diabetic control groups (group II). Glibenclamide (dissolved in 0.5ml distilled water) at a dose of 0.3 mg/kg per day was used as a standard drug based on reports of a previous study[12].

Biochemical Analysis: An autopsy was performed at the end of the experiment by giving mild ether anesthesia to overnight fasted rats. Blood was collected from the heart via cardiac puncture and allowed to clot at room temperature, further centrifuged at 4 °C at 3000 rpm for separation of serum. The liver was dissected, rinsed with ice-cold saline, and stored at −20 °C for further studies. The activities of all the antioxidant enzymes were calculated in liver tissue homogenates. Reduced glutathione (GSH) was estimated using standard method [12]. Alteration in the activity of Superoxide Dismutase (SOD) was measured by the procedure given by Marklund and Marklund [13]. Liver glycogen was calculated by Montogmery's method [14]. Lipid peroxides were estimated in liver tissue homogenates using standard method [15]. The concentrations of serum insulin were assessed according to the standard protocol using radioimmunoassay (RIA).

Acute Oral Toxicity Study: Acute oral toxicity test was performed for *C. decidua* fruit methanolic extract [16]. The male Wistar rats were treated with graded dose of methanolic extracts of fruit of *C. decidua* (5, 50, 300, 2000 mg/kg b.wt./rats/day) were kept under close observation to find out any possible toxic effects or changes in behavioural pattern. Based on, the biological dose was fixed at a maximum of 200 mg/kg body weight of the extract for the treatment.

Histopathological Studies: Hepatic tissue was cut into small pieces and fixed in Bouin's solution. Ethanol-xylene series was used to process the fixed tissues. Harris hematoxylin and eosin staining was preceded by section cutting and paraffin embedding of the processed tissues. Microscopy was performed using Leitz-diaplan microscope (Leica, Germany) for micrographs of H&E stained sections **Fig. 3a-3d**.

Statistical Analysis: Values are mentioned as mean ± SEM (standard error of the mean). One-way ANOVA with Tukey-Kramer multiple comparison tests was used to calculate variations in groups [17]. Values of $p<0.05$ were considered statistically significant.

RESULTS: The fruits of *C. decidua* were extracted with methanol, and different identification tests were conducted for evaluation of chemical constituents present in the extract as per the standard methods [18]. The phytochemical screening indicated presence of proteins, alkaloids, carbohydrates, saponins phenols, flavonoids, and glycosides, in methanolic extract of fruits of *Capparis decidua* (Forssk.) Edgew **Table 1**.

TABLE 1: PHYTOCHEMICAL INVESTIGATION OF METHANOLIC EXTRACT OF FRUITS OF *CAPPARIS DECIDUA* (FORSSK.) EDGEW.

Chemical category	Name of test	Methanolic extract
Carbohydrates	Molish	+
Proteins and Amino acids	Biuret test	+
Alkaloids	Dragondorff's	+
Glycosides	Liebermann's Test	+
Phenolics/Tannins	Ferric chloride test	+
Flavonoids	Shinoda's Test	+
Saponins	Drug + water + shaking	+
Steriods	Libermann-Buchard test	+

(+ = present)

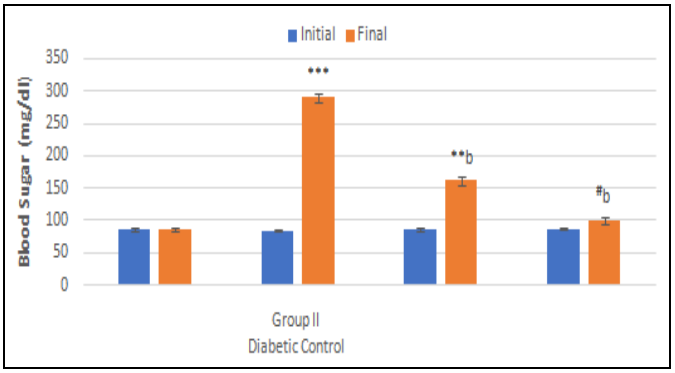

FIG. 1: EFFECT OF CDMtE ON BLOOD GLUCOSE. Values are expressed as mean ± SEM (n=6). Level of significance: - #-non significant, *p<0.05, ** p<0.01, ***p<0.001 when compared with Group I (Normal Control), [a]p<0.01; [b]p<0.01 when compared with Group II (Diabetic Control)

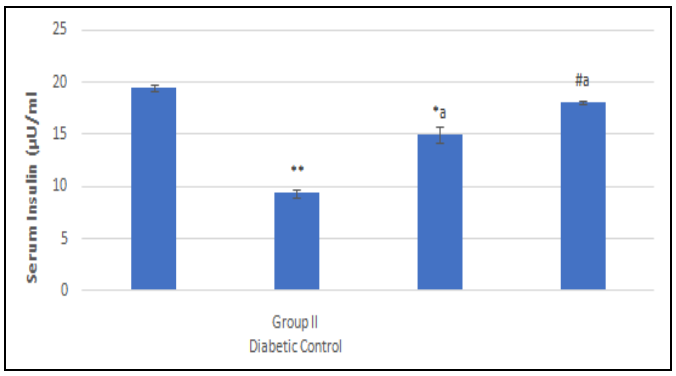

FIG. 2: EFFECT OF CDMtE ON SERUM INSULIN. Values are expressed as mean ± SEM (n=6). Level of significance:- #-non significant, *p<0.05, ** p<0.01,***p<0.001 when compared with Group I (Normal Control), [a]p<0.01; [b]p<0.01 when compared with Group II (Diabetic Control)

A significant increase (p<0.001) in blood glucose level was observed in STZ treated rats (diabetic control rats). A significant antihyperglycaemic effect was marked after oral treatment with CDMtE for 7 days **Fig. 1**. The results of the current study revealed that the CDMtE exhibited significant antihyperglycaemic activity in STZ-diabetic rats.

A significant decrease (p<0.01) in serum insulin level was also noticed in STZ treated rats. Oral treatment with CDMtE at a dosage of 200 mg/kg for 7 days boosted insulin level in diabetic rats **Fig. 2**.

Table 2 shows that there is a significant elevation in LPO levels and reduction in GSH, SOD, and Glycogen levels in the liver of diabetic rats compared to control rats. A significant change in biochemical parameters- LPO, SOD, GSH, and liver glycogen was observed after administration of *Capparis decidua* fruits methanolic extract at a dosage of 200 mg/kg in comparison to diabetic rats of group II **Table 2**.

TABLE 2: EFFECT OF CDMtE ON LIVER BIOCHEMISTRY

Treatment Groups	Glycogen (mg/g)	LPO (nmoleMDA/mg protein)	GSH (nmole/g tissue)	SOD (µmole/mg protein)
Group I - Control (Vehicle Treated)	5.83±0.17	2.02±0.19	4.79±0.12	9.08±0.20
Group II - Diabetic Control	3.10±0.28**	8.27±0.22***	2.40±0.50**	3.21±0.51***
Group III - Diabetic + *C. decidua* (200mg/kg b.wt/day)	4.58±0.32[#a]	6.97±0.50[*a]	3.40±0.18[#a]	6.49±0.13[*b]
Group IV - Diabetic + Glibenclamide (0.3mg/kg b.wt/day)	5.02±0.26[#b]	4.09±0.29 [#a]	4.11±0.31[#a]	7.05±0.30 [#b]

Values are expressed as mean ± SEM (n=6). Level of significance: - #-non significant, *p<0.05, ** p<0.01, ***p<0.001 when compared with Group I (Normal Control), [a]p<0.05; [b]p<0.01 when compared with Group II (Diabetic Control)

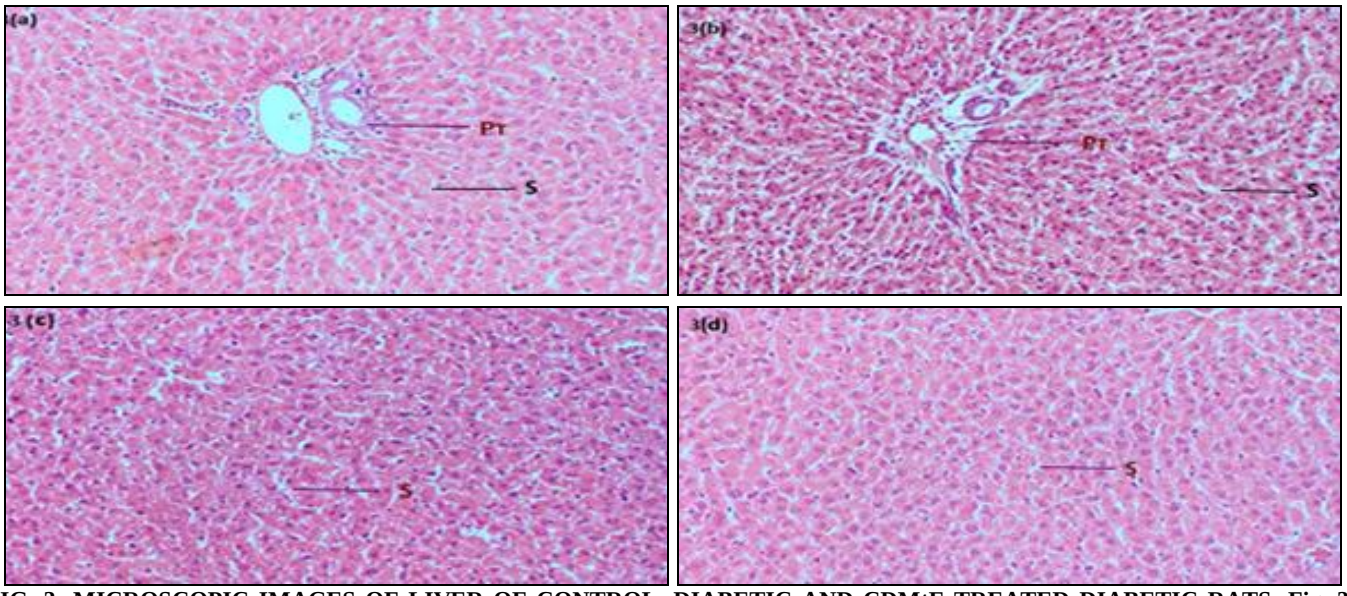

FIG. 3: MICROSCOPIC IMAGES OF LIVER OF CONTROL, DIABETIC AND CDMtE TREATED DIABETIC RATS. Fig. 3a shows the normal architecture of liver tissue, normal portal triad, and sinusoids of normal control rats. (Hematoxylin and eosin stain; original magnification X 200). **Fig. 3b** is showing degeneration in the normal structure of the liver showing dilation of sinusoids of STZ-diabetic untreated rats. (Hematoxylin and eosin stain; original magnification X 200).In **Fig. 3c**, CDMtE (200 mg/kg b.wt./day) treatment to diabetic control rats showing the reversion of ruptured liver to near-normal structure. (Hematoxylin and eosin stain; original magnification X 200). In **Fig. 3d**, glibenclamide (0.3 mg/kg b.wt./day) treatment to diabetic control rats showing the normal structure of liver. (Hematoxylin and eosin stain; original magnification X 200).

Fig. 3a shows the histological results of hepatic tissues. The structure of hepatic tissue, portal triad and blood sinusoids were normal in the control rats **Fig. 3B**. Induction of diabetes in animals caused severe pathological changes, including dilation of hepatic sinusoids and degeneration of hepatic tissue **Fig. 3c-d**. However, liver of rats treated with CDMtE extract significantly reduced the histological alterations and reversed the structure near to normal **Fig. 3c** and **3d**.

DISCUSSION: Oxidative stress plays a vital role in the development of complications of diabetes such as dysfunction of β-cells, insulin resistance, and impaired glucose tolerance. Oxidative stress refers to overproduction of reactive oxygen species or ROS. Reactive oxygen species (ROS) are oxygen-containing chemically reactive species such as peroxides, superoxide, hydroxyl radical. Under the diabetic state, excessive glucose reacts with plasma protein and forms advanced glycation end products (AGEs). These AGEs trigger the over-production of ROS, resulting in the generation of oxidative stress. ROS are responsible for hepato-cyte cytotoxicity [19]. Streptozotocin is also responsible for the increase of ROS/RNS generation [20].

To balance the increased ROS, cells have their own defense system- the antioxidant protection system. This system includes enzymatic and non-enzymatic components. Antioxidants are stabilizing agents that regulate the overproduction of ROS by scavenging activity. Antioxidants donate their electrons to stabilize free radicals or ROS and minimize their harmful effects. Organ damage due to oxidative stress can be prevented by various antioxidants (both enzymatic and non-enzymatic) [21].

SOD is an enzymatic antioxidant that removes the superoxide radicals and repairs cells from the damage caused by these superoxides [22]. In contrast, GSH is a non-enzymatic antioxidant and also an intracellular non-protein thiol which reduced H_2O_2 and acts against oxidative stress by removing over produced free radicals.

Many plants also contain compounds that possess antioxidant activity. *Capparis decidua* is one of them. Phytochemical investigations of the plant also confirmed the presence of flavonoids, tannins, phenols, saponins, and sterols. These phytochemicals exhibit antioxidant activities [23].

In this study, hyperglycaemia was noticed in STZ–induced diabetic rats due to overproduction of ROS. Administration of *C. decidua* fruits methanolic extract to diabetic rats significantly improved the blood sugar level. The possible mechanism by which CDMtE at a dose of 200 mg/kg significantly decreased the blood glucose in diabetic rats is probably due to antioxidants present in the extract. These antioxidants stimulate surviving β-cells or regenerate β-cells, resulting in improved insulin secretion. It has been reported that antioxidants play a major role in β-cells regeneration [24].

In the present study, a reduction in the activity of antioxidants such as SOD and GSH in hepatic tissue of diabetic control rats was observed. This finding was in accordance with a study conducted on male albino wistar rats [25]. This study explained that a reduction in SOD indicates free radical-induced damage. Additionally, in diabetes, glycosylation of SOD and loss of copper, which act as an essential cofactor in SOD activity, can be another factor responsible for a reduction in SOD activity [26].

In CDMtE treated diabetic rats, an elevation in GSH and SOD level was observed. Antioxidants present in *Capparis decidua* extract reduce oxidative stress, resulting in an increase in GSH levels and SOD activity. The increased activity of SOD shows the potential effect of CDMtE on the scavenging of free radicals. The activity of many enzymes, glutathione S-transferase, γ-glutamyl transferase, glucose 6-phosphate dehydrogenase, *etc*. decide the levels of GSH [27]. The CDMtE extract might be responsible for the activation of these enzymes, which further increase the glutathione pool in hyperglycaemic rats.

Overproduction of free radicals is also associated with an increase in Malondialdehyde production (MDA). MDA is a byproduct of polyunsaturated fatty acid peroxidation [28]. An elevation in the level of MDA in diabetic rats has been reported in previous researches [29, 30]. The finding of our study confirms these observations. A low amount of

insulin in diabetes also increases fatty acyl coenzyme-A oxidase enzyme activity which is responsible for the initiation of beta-oxidation, resulting in peroxidation of lipids [31].

Treatment of diabetic rats with CDMtE reduced peroxidation of lipids which is probably due to the presence of active components in *C. decidua* methanolic extract (CDMtE). The antioxidants present in *C. decidua* are responsible for free radicals scavenging activity and hepatoprotective properties of the extract.

In our study, a decrease in glycogen content in hepatic tissue was noticed in STZ-induced diabetic rats. These results are in agreement with the effect of *Calanthe fimbriata* on streptozotocin-induced diabetic mice [32]. STZ is a toxic compound that induces diabetes by destroying pancreatic β-cells, which results in a decrease in insulin level [33]. When CDMtE at a dosage of 200 mg/kg was given orally to diabetic rats, glycogen content in hepatic tissue increased significantly which may be due to an increase in insulin level. Insulin stimulates glucose uptake and activates glycogen synthase in hepato-cytes [34]. Insulin mediates activation of phospho-inositide-dependent serine-threonine protein kinase (AKT), which plays a major role in glucose homeostasis [34]. Another serine-threonine protein kinase GSK-3, acts as a key component in the regulation of glucose metabolism [35]. In insulin-stimulated conditions, AKT phosphorylates and inactivate GSK-3. Inactivation of GSK-3 inhibits phosphorylation of enzyme glycogen synthase (GS) [34]. Hence, glycogen synthesis increases. This mechanism is responsible for the conversion of glucose into glycogen.

The observations of this study clearly indicate that the CDMtE decreases oxidative stress and effective in preventing oxidative damage, which is considered as one of the major factors responsible for liver damage in the diabetic state.

CONCLUSION: It can be concluded that CDMtE is potentially effective in protecting the liver tissue from the damage caused by ROS. Phytochemical investigations suggest that CDMtE consists of many phytoconstituents, which elevate cellular antioxidant defense activities and reduction of hyperglycemia in STZ-treated diabetic control rats. Hence, the findings of this research can be used to develop a potent antidiabetic drug from the plant extract used. Though further research is required to isolate and characterize the potent molecules, which probably play an important role in the treatment of diabetic mellitus and associated hepatotoxicity and more pharmacological investigations are also required to elucidate the exact mechanism and to identify and isolate active principle(s).

ACKNOWLEDGEMENT: The authors are very thankful to the Head and Coordinator (CAS) of the Department of Zoology, University of Rajasthan, Jaipur, India, for providing the necessary facilities for the above investigation.

CONFLICTS OF INTEREST: The authors declare that they have no conflict of interest.

REFERENCES:

1. Schwartz SS, Epstein S, Corkey BE, Grant SFA, Gavin JR and Aguilar RB: The time is right for a new classification system for diabetes: rationale and implications of the β-cell-centric classification scheme. Diabetes Care 2016; 39(2):179-86.
2. International Diabetes Federation: IDF Diabetes, International Diabetes Federation, Brussels, Belgium 2015; 7th edition.
3. Lu S, Liao Z, Lu X, Katschinski DM, Mercola M, Chen J, Brown JH, Molkentin JD, Bossuyt J and Ber DM: Hyperglycemia acutely increases cytosolic reactive oxygen species *via* O-linked GlcNAcylation and CaMKII activation in mouse ventricular myocytes. Circulation Research 2020; 126(10): 80-96.
4. Kasole R, Martin HD and Kimiywe J: Traditional Medicine and its role in the management of diabetes mellitus: "Patients' and Herbalists' Perspectives". Evidence-Based Complementary and Alternative Medicine 2019; 1-12.
5. Shrestha JTM, Shrestha H, Prajapati, M, Karkee A and Maharjan A: Adverse effects of oral hypoglycemic agents and adherence to them among patients with type 2 diabetes mellitus in Nepal. Journal of Lumbini Medical College 2017; 5(1): 34-40.
6. Li J, Zhang Y, Jiao L, Olatunji OJ and He B: Preventive effect of crude polysaccharide extract from Chinese wolfberry against hyperglycemia-induced oxidative stress and inflammation in streptozotocin-induced diabetic rats. Pharmacognosy Magazine 2019; 15(65): 638-44.
7. Mathur C and Gupta RS: A Review on medicinal plants of Rajasthan having antidiabetic activity. Asian Journal of Pharmaceutical and Clinical Research 2018; 11(12): 33-39.
8. Nazara S, Hussain MA, Khan A, Muhammad G and Tahir MN: *Capparis decidua* Edgew (Forssk.): A comprehensive review of its traditional uses, phytochemistry, pharmacology and nutrapharmaceutical potential. Arabian Journal of Chemistry 2020; 13(1): 1901-16.

9. Soni LK, Dobhal MP, Arya D, Bhagour K, Parasher P and Gupta, RS: *In-vitro* and *in-vivo* antidiabetic activity of isolated fraction of **Prosopis cineraria** against streptozotocin-induced experimental diabetes: A mechanistic study. Biomedicine and Pharmacotherapy 2018; 108: 1015-21.
10. Jaishree V and Narsimha S: Swertiamarin and quercetin combination ameliorates hyperglycemia, hyperlipidemia and oxidative stress in streptozotocin-induced type 2 diabetes mellitus in wistar rats. Biomedicine and Pharmacotherapy 2020; 130: 1-8.
11. Gupta R, Mathur M, Bajaj VK, Katariya P, Yadav S, Kamal R and Gupta RS: Evaluation of antidiabetic and antioxidant activity of *Moringa oleifera* in experimental diabetes. Journal of Diabetes 2012; 4: 164-71.
12. Moron MJ, Depierre JW and Mannivik B: Levels of glutathione, glutathione reductase and glutathione S-transferease activities in rat lung and liver. Biochemica et Biophysica Acta 1979; 582: 67-68.
13. Marklund S and Marklund G: Involvement of the superoxide anion radical in the autoxidation of pyrogallol and a convenient assay for superoxide dismutase. European Journal of Biochemistry 1974; 47: 469-74.
14. Montgomery R: Determination of glycogen. Archives of Biochemistry and Biophysics 1957; 67: 378-81.
15. Ohkawa H, Ohishi N and Yagi K: Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. Analytical Biochemistry 1979; 95: 351-58.
16. Walum E: Acute oral toxicity. Environmental Health Perspectives 1998; 106: 497-503.
17. Tukey JW: One degree of freedom for non-additivity. Biometrics 1949; 5: 232-42.
18. Banu KS and Cathrine L: General techniques involved in phytochemical analysis. International Journal of Advanced Research in Chemical Science 2015; 2(4): 25-32.
19. Eftekhari A, Ahmadian E, Panahi-Azar V, Hosseini H, Tabibiazar M and Dizaj SM: Hepatoprotective and free radical scavenging actions of quercetin nanoparticles on aflatoxin B1-induced liver damage: *in-vitro*/*in-vivo* studies. Artificial Cells, Nanomedicine, and Biotechnology 2018; 46(2): 411-20.
20. Al-Nahdi AMT, John A and Raza H: Cytoprotective Effects of N-Acetylcysteine on Streptozotocin- Induced Oxidative Stress and Apoptosis in RIN-5F Pancreatic β-Cells. Cellular Physiology and Biochemistry 2018; 51: 201-16.
21. Bamagous GA, Al Ghamdi SS, Aziz Ibrahim IA, Mahfoz AM, Afify MA, Alsugoor MH, Shammah AA, Arulselvan Pand Rengaraja T: Antidiabetic and antioxidant activity of ethyl acetate extract fraction of *Moringa oleifera* leaves in streptozotocin-induced diabetes rats *via* inhibition of inflammatory mediators. Asian Pacific Journal of Tropical Biomedicine 2018; 8(6): 320-27.
22. Nair A and Nair BJ: Comparative analysis of the oxidative stress and antioxidant status in type II diabetics and nondiabetics: A biochemical study. Journal of Oral Maxillofacial Pathology 2017; 21(3): 394-401.
23. Forni C, Facchiano F, Bartoli M, Pieretti S, Facchiano A, D'Arcangelo D, Norelli S, Valle G, Nisini R, Beninati S, Tabolacci C, and Jadeja RN: Beneficial role of phytochemicals on oxidative stress and age-related diseases. BioMed Research International 2019; 1-16.
24. Wang J and Wang H: Oxidative stress in pancreatic beta cell regeneration. Oxidative Medicine and Cellular Longevity 2017; 1930261: 1-9.
25. Sugumar M, Doss DVA and Maddisetty PNP: Hepato-renal protective effects of hydroethanolic extract of *Senna alata* on enzymatic and non-enzymatic antioxidant systems in streptozotocin-induced diabetic rats. Integrative Medicine Research 2016; 5(4): 276-83.
26. Haddad N, Nori E and Ali S: The effect of type two diabetes mellitus on superoxide dismutase (SOD) activity and its correlation with HbA1c in Iraqi patients. International Journal of Engineering Research and Science 2016; 2(4): 7-15.
27. Subramani C, Rajakannu A, Gaidhani S, Raju I, Vaidya D and Singh K: Glutathione-redox status on hydro-alcoholic root bark extract of *Premna integrifolia* Linn. in high fat diet induced atherosclerosis model. Journal of Ayurveda and Integrative Medicine 2018; 1-7.
28. Baliga S, Chaudhary M, Bhat S, Bhansali P, Agrawal A and Gundawar S: Estimation of malondialdehyde levels in serum and saliva of children affected with sickle cell anemia. Journal of Indian Society of Pedodontics and Preventive Dentistry 2018; 36(1): 43-47.
29. Konda PY, Dasari S, Konanki S and Nagarajan P: *In-vivo* antihyperglycemic, antihyperlipidemic, antioxidative stress and potential antioxidant activities of *Syzygium paniculatum* Gaertn. in streptozotocin-induced diabetic rats. Heliyon 2019; 5(3): e01373.
30. Ghanbari E, Nejati V and Khazaei M: Improvement in serum biochemical alterations and oxidative stress of liver and pancreas following use of royal jelly in streptozotocin-induced diabetic rats. Cell Journal 2016; 18(3): 362-70.
31. Jackowski S and Leonardi R: Deregulated coenzyme A, loss of metabolic flexibility and diabetes. Biochemical Society Transactions 2014; 42(4): 1118-22.
32. Peng Y, Gao Y, Zhang X, Zhang C, Wang X, Zhang H, Wang Z, Liu Y and Zhang H: Antidiabetic and hepatoprotective activity of the roots of *Calanthe fimbriata* Franch. Biomedicine and Pharmacotherapy 2019; 111: 60-67.
33. Wu J and Yan L: Streptozotocin-induced type 1 diabetes in rodents as a model for studying mitochondrial mechanisms of diabetic β cell glucotoxicity. Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy 2015; 8: 181-88.
34. Li Q, Zhao Q, Zhang J, Zhou L,Zhang W, Chua BT, Chen Y, Xu L and Li P: The protein phosphatase 1 complex is a direct target of AKT that links insulin signaling to hepatic glycogen deposition. Cell Reports 2019; 28(13): 3406-22.
35. Nabben M and Neumann D: GSK-3 Inhibitors: Anti-Diabetic treatment associated with cardiac risk? Cardiovascular Drugs and Therapy 2016; 30: 233-35.

How to cite this article:
Mathur C, Soni M and Gupta RS: Antihyperglycaemic and antioxidant activity of methanolic extract of *Capparis decidua* (Forssk.) Edgew. in streptozotocin-induced diabetic rats. Int J Pharm Sci & Res 2021; 12(5): 2700-06. doi: 10.13040/IJPSR.0975-8232.12(5).2700-06.