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Original Research Article

Study the Physiological Effects of Chia (*Salvia hispanica*) Seeds Extract on Pancreatic Damage Induced in Male Rats

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Abstract: The present study was conducted to evaluate the protective and curative effects of chia (*Salvia hispanica L.*) seed extract (CSE) against STZ-induced pancreatic damage in male Wistar rats. Pancreatic injury was achieved by a single intraperitoneal administration of STZ at 55 mg/kg. Fifty male rats were randomly divided into five groups (n=10) including Normal Control group, Diabetic Control group and three treatment groups (CSE-Low; 250 mg/kg, CSE-High; 500 mg/g, Standard-treatment group; Metformin 150 mg/kg). Biochemical indicators of pancreatic activity, oxidative stress and histopathological alterations were analysed after 28 days of daily oral treatment. Results: CSE significantly ($P < 0.05$) decreased levels of fasting blood glucose whereas concentrations of serum insulin were found to increase after treatment in a dose dependent manner. In addition, CSE treatment also normalized the activity of pancreatic enzymes (serum amylase and lipase) which were significantly altered in diabetic control group. The pancreatic tissue oxidative stress indicator MDA was markedly decreased, and SOD and CAT were significantly increased. These results showed that Salvia hispanica seed extract has strong antioxidant and antidiabetic activity which provides a significant protection against pancreatic injury that exerted by chia seeds may be attributed to high contents of phenolic compounds, specifically alpha-linolenic acid.

Keywords: *Salvia hispanica L*, seed extract, STZ-induced, pancreatic damage, rats.

1. INTRODUCTION

The pancreas is a critical organ with endocrine and exocrine functions, which are central to metabolic homeostasis. The endocrine part, which mainly consists of the islets of Langerhans, is not only responsible for insulin and glucagon release maintaining blood glucose levels. In contrast, the exocrine portion of the pancreas releases digestive enzymes (i.e., amylase and lipase) to mix with contents in duodenum for carbohydrate and fat digestion. Pancreatic injury, be it due to inflammatory events such as pancreatitis or autoimmune destruction like in type 1 diabetes, results in dramatic metabolic derangement and systemic sequelae.

Diabetes mellitus is a common metabolic disorder characterized by prolonged hyperglycemia due to deficiency in insulin secretion, action, or both. The prevalence of diabetes worldwide has—the Transferable Character and Value of Identity: Implications for the Treatment of Diabetes International Diabetes Federation (2021) estimated that There are 537 million adults living with diabetes. Beta-cell destruction and/or dysfunction is a key pathological component of diabetes. Streptozotocin (STZ) is commonly used to develop an experimental model of pancreatic injury. STZ is a beta cell-selective glucosamine-nitrosourea that enters these cells through the GLUT2 carrier and causes DNA alkylation and then activates poly(ADP-ribose) polymerase (PARP)-1 for depletion of the cellular NAD+ pool (Szkudelski, 2001a), with resultant ATP depletion and necrosis. In addition, STZ-induced reactive oxygen species (ROS) may further enhance pancreas injury caused by its low levels of antioxidant enzymes.

Drugs in present use for the treatment of pancreatic injury and diabetes, insulin and oral hypoglycemic drugs, though effective also have

limitations like risk of hypoglycemia embarking on medications and bear weight gain and gastrointestinal complications. Accordingly, there is an increasing interest in the discovery of natural bioactive plant compounds with potential therapeutic usage and low toxicity. Herbal medicines from natural sources are effective in phytotherapy and have been widely used for centuries to treat different health disorders, which have recently been scientifically investigated.

Salvia hispanica L., also known as chia, is an annual herb of the family Lamiaceae. Originating in southern Mexico and Guatemala, the chia seed was a staple in ancient Aztec and Mayan diets. The seed is a superfood that has gained popularity in the US and throughout Europe in recent years. Seeds are an abundant source of omega-3 polyunsaturated fatty acid, alpha-linolenic acid (ALA), in addition to high-quality proteins, dietary fiber, vitamins and minerals (Khalid *et al.,* 2022). Apart from its essential nutrient content, chia seeds have a wide range of phytochemicals such as phenolic acids (caffeic acid, chlorogenic acid and rosmarinic acid) and flavonoids (quercetin, kaempferol and myricetin).

Recent investigations have reported the antioxidant, anti-inflammatory and cardioprotective effects of chia seeds. For example, Trisnadi (2023) discovered that the extract of chia seed can decrease blood sugar dearth and malondialdehyde in rats diabetes which may indicate protective functions against oxidative damage. It has also been proposed that the presence in chia of bioactive peptides and polyphenols may play a role in the altered activity of enzymes related to carbohydrate and lipid digestion, including amylase (alpha-amylose) and pancreas lipase (Mihafu, 2024). Although the beneficial health effects of chia have been reported, focused investigation on its effects over markers pancreatic architecture and exocrine function in a model of chemically induced damage is necessary.

The aim of the current study is to completely consider *Salvia hispanica* seed extract on pancreatic injury in male rats. Through the evaluation of markers of pancreatic function, oxidative stress and histopathological alterations, we intend to generate scientific evidence supporting the possible use of chia as a health food or therapeutic coadjuvant for both pancreatic disease and diabetes. The scientific rationale behind the study hypothesis The present study is based on the premise that antioxidant AC-extract of CS can protect STZ-induced oxidative stress, thus capable of preserving structural and functional integrity of pancreas.

2. MATERIALS AND METHODS

2.1. Plant Material and Extraction

Seeds of Salvia hispanica L. (black) were obtained from a well-reputed local commercial vendor and authenticated by a plant taxonomist at the Department of Botany. The voucher specimen of this plant (Garhwali No.123543) was deposited in the university herbarium for future reference. Seeds were cleaned from any kind of contamination and then powdered to the fineness with electric grinder.

The extraction was executed with a methanolic/ solvent system to achieve the optimum recovery of phenolics. In short, 500 g of ground chia seed was macerated in 2.5 L of 80% methanol for a period of 72 hours at room temperature with intermittent stirring. The solution was filtered through Whatman No. 1 filter paper. The filtrate was evaporated under vacuum with a rotary evaporator at 40 °C to remove the solvent. The crude methanolic extract (CSE) obtained was 174 dried under vacuum desiccator in the dark and then packed into airtight ambered container and stored at 4°C until use. The extract % yield was determined by the following equation:

Yield (%) = (Weight of extract / Weight of dry starting material) X 100.

2.2. Preliminary Phytochemical Screening

The crude and aqueous extracts were evaluated for the presence of secondary metabolites, such as alkaloids, flavonoids, tannins, saponins glycosides and terpenoids (Harborne 1998). The Total Phenolic Content (TPC) was also determined using the Folin-Ciocalteu method and the Total Flavonoid Content (TFC) was measured with aluminum chloride colorimetric method. Results for TPC were expressed in mg/g of GAE (gallic acid equivalents)/dry extract, whereas that of TFC was calculated as mg/g QE (quercetin equivalent)/dry extract.

2.3. Experimental Animals

Fifty healthy male adult Wistar rats weighing between 180 and 220 g constituted the material in this study. Animals were obtained from the Babylon university central animal house. The rats were kept in polycarp cages (furnished with five rats per cage) and maintained under standard conditions of temperature (25 ± 2°C), humidity (50-60%) as well as light/dark cycle of 12-hrs. The animals were allowed free access to a standard pellet diet and water. Animals were kept in the laboratory for 2 weeks before the experiment was started. All animal studies were approved by the Institutional Animal Ethics Committee (IAEC) guidelines for the care and use of laboratory animals.

2.4. Induction of Pancreatic Damage

Pancreatic injury and experimental diabetes were initiated by a single intraperitoneal (i.p.) injection of streptozotocin (STZ; Sigma-Aldrich, USA). STZ was dissolved in 0.1 M citrate buffer (pH 4.5) prior to use. Rats were fasted for 12 h overnight, and they were then administered with a dose of 55 mg/kg body weight. Rats of control groups were injected with citrate buffer with the same volume of 5 ml/kg alone. At 72 h after receiving the STZ injection, blood was obtained from tail vein, and fasting blood glucose levels were determined using a

glucometer. Diabetic rats were those with FBG level of more than 250 mg/dL which met the inclusion criteria.

2.5. Experimental Design

- The rats were assigned randomly to five groups, n=10:
- Group I (Normal Control): Non-diabetic rats treated with distilled water (5 mL/kg) orally.
- Group II (Diabetic Control): STZ-induced diabetic rats administered with distilled water (5 mL/kg) orally.
- Group III (CSE-Low): Diabetic rats were induced with STZ and they received chia seed extract orally at a dose of 250 mg/kg body weight.
- Group IV(CSE-High): Diabetic rats received chia seed extract at a dose of 500 mg/kg b.wt STZ.
- Group V: STZ-induced diabetic rats administered with the standard anti-diabetic drug Metformin (150 mg/kg).
- Extract and standard drug were given orally using a gastric gavage daily for 28 days. The body weight and oral food intake were recorded every weekly during the whole experimental period of treatment.

2.6. Collection of Samples

After 28 days of treatment, the rats were fasted overnight and anesthetized by intraperitoneal injection of ketamine (80 mg/kg) and xylazine (10 mg/kg). Cardiac puncture was performed and collected in 2 types of tubes, one without anticoagulant for serum separation and one with EDTA solubilized or liquid needed to analyze whole blood. Then, the serum was separated from blood by centrifugation at 3,000 rpm for 15 min at 4°C and then stored at –80°C for biochemical analysis.

The rats were then killed immediately after blood sample collection and the pancreas was removed. A part of pancreatic tissue was homogenized in ice-cold phosphate-buffered saline (PBS, pH 7.4) to produce a 10% uniformity (w/v). The homogenate was centrifuged at 10,000 rpm during 4°C for 10 min and the supernatant fluid was used for oxidative stress markers estimation. The rest of the pancreas was fixed in 10% neutral buffered formalin for histopathological analysis.

2.7. Biochemical Assays

2.7.1. Glycemic and Pancreatic Function Markers

Fasting blood glucose was determined by the method of Glucose oxidase - peroxidase GOD-POD. Serum insulin was measured with a rat enzyme-linked immunosorbent assay (ELISA) kit (Abcam, UK). Serum amylase and lipase activities as an indicator of exocrine pancreatic function were analyzed using kinetic colorimetric assay kits (Sigma-Aldrich, USA) following the supplier's instructions.

2.7.2. Oxidative Stress Markers

The pancreatic homogenate in the lipid peroxidation was estimated by malondialdehyde (MDA) using its thiobarbituric acid reactive substances (TBARS). The antioxidant enzymes (superoxide dismutase [SOD] and catalase [CAT]) activities were measured by spectrophotometric methods. Contents of the non-enzymatic antioxidant status were also determined by measurement of glutathione concentration (GSH).

2.9. Statistical Analysis

Values are presented as the mean ± SD. Data were statistically analyzed by one-way analysis of variance (ANOVA) and Tukey's post hoc test for multiple comparisons. Differences with $P<0.05$ were considered statistically significant. Statistical analysis was performed with GraphPad Prism software (version 9.0).

3. RESULTS

3.1. Phytochemical Analysis

Phytochemical profile In the present study, the methanolic extract of Salvia hispanica seeds contained wide range of bioactive compounds as described in Table.1. The extract was positive for flavonoids, phenolic acids, tannins and saponins whereas alkaloid was present in trace amount. The quantitative assay exhibited high of TPC and 185.4 ± 8.2 mg GAE/g of dry extract Total Flavonoid Content (TFC) on the levels of 42.1 ± 3.5 mg QE/g.These results indicate that the extraction step effectively enriched them in promising antioxidants rich compounds content from seed.

3.2. Effect on body weight and glycemic control

The induction of diabetes by STZ resulted in a marked ($P<0.05$) decrease in body weight of rats pertaining to diabetic control treatment compared to that normal control treatment. Nonetheless, weight was progressively restored from day 28 after treatment with CSE at doses of 250 mg/kg and 500 mg/kg.

From the view of glycemic status, diabetic controls remained hyperglycemic throughout the duration of study. Treatment with CSE reduced the FBG levels in a dose-dependent manner significantly ($P < 0.05$) (Figures 1 and 2). CSE, at the high dose of 500 mg/kg, was particularly effective and exhibited results similar to those of standard Metformin. In the diabetic control, serum insulin (I) was depleted but significantly improved in CSE-treated groups with some remaining beta cells protected or stimulation of insulin secretion.

Table 1: Effect of CSE on Body Weight, Fasting Blood Glucose (FBG), and Serum Insulin

Group	Initial Body Weight (g)	Final Body Weight (g)	FBG (mg/dL)	Serum Insulin (µIU/mL)
Normal Control	205.4 ± 5.2	248.6 ± 6.8	84.2 ± 5.1	18.4 ± 1.2
Diabetic Control	202.1 ± 6.1	162.4 ± 9.4*	312.5 ± 15.4*	4.2 ± 0.8*
CSE-Low (250 mg/kg)	204.8 ± 4.8	185.2 ± 7.2#	185.6 ± 12.8#	8.5 ± 1.1#
CSE-High (500 mg/kg)	201.5 ± 5.5	212.4 ± 8.5#	124.8 ± 9.6#	12.6 ± 1.4#
Standard (Metformin)	203.2 ± 5.9	225.8 ± 7.4#	110.4 ± 8.2#	14.2 ± 1.5#

Data are expressed as Mean ± SD (n=10). *P < 0.05 compared to Normal Control; #P < 0.05 compared to Diabetic Control.

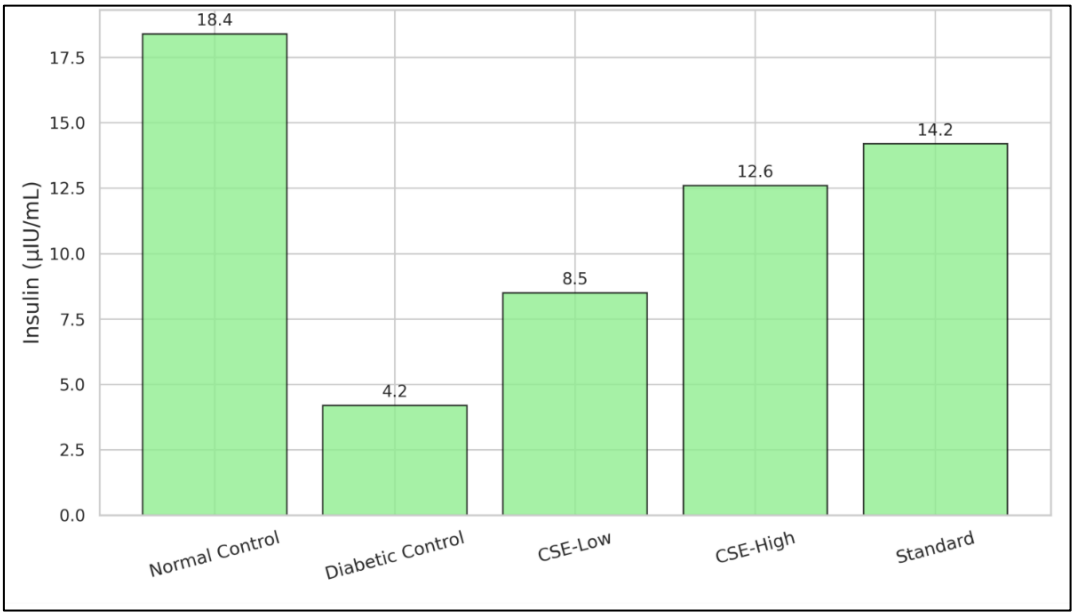

Figure 1: Serum insulin levels in study groups

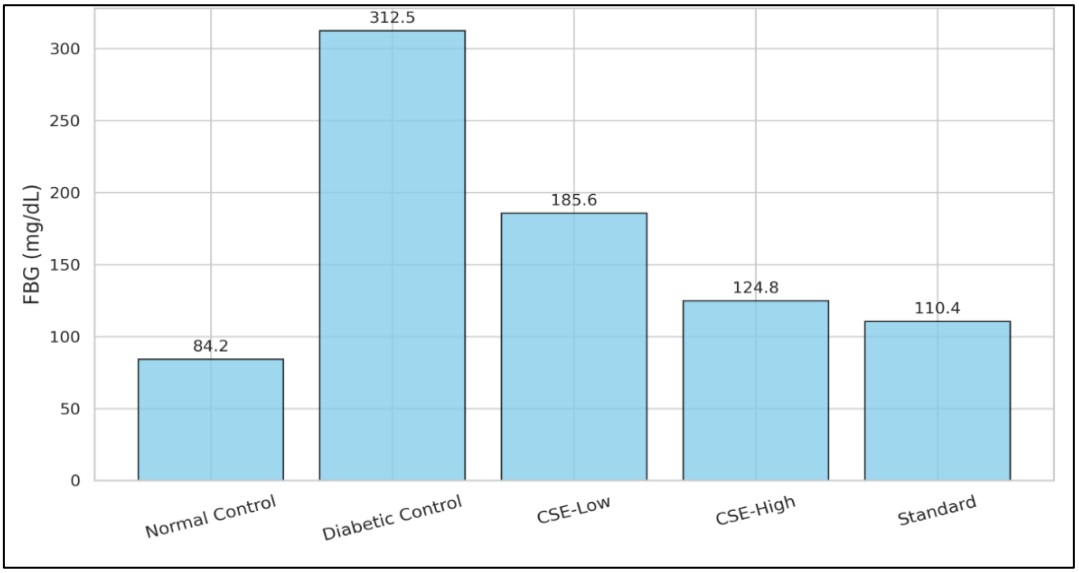

Figure 2: Blood glucose levels in study groups

3.3. Effect on Pancreatic Function Markers

Serum amylase and lipase were determined for assessment of exocrine pancreatic function. There was significant ($P < 0.05$) reduction in the activities of these two enzymes in diabetic control groups as compared to normal control group, showing significant exocrine inefficiency after STZ-induced injury. CSE treatment had the significantly ameliorative effects on this alteration, in particularly, 500 mg/kg dose was more effective to limit the activities of enzymes near normal control.

Table 2: Effect of CSE on Serum Amylase and Lipase Activities

Group	Serum Amylase (U/L)	Serum Lipase (U/L)
Normal Control	1450.4 ± 85.6	48.2 ± 4.5
Diabetic Control	620.5 ± 52.4*	18.6 ± 2.8*
CSE-Low (250 mg/kg)	895.2 ± 64.8#	29.4 ± 3.2#
CSE-High (500 mg/kg)	1180.6 ± 72.2#	38.5 ± 4.1#
Standard (Metformin)	1240.2 ± 68.5#	42.1 ± 3.8#

Data are expressed as Mean ± SD (n=10). *P < 0.05 compared to Normal Control; #P < 0.05 compared to Diabetic Control.

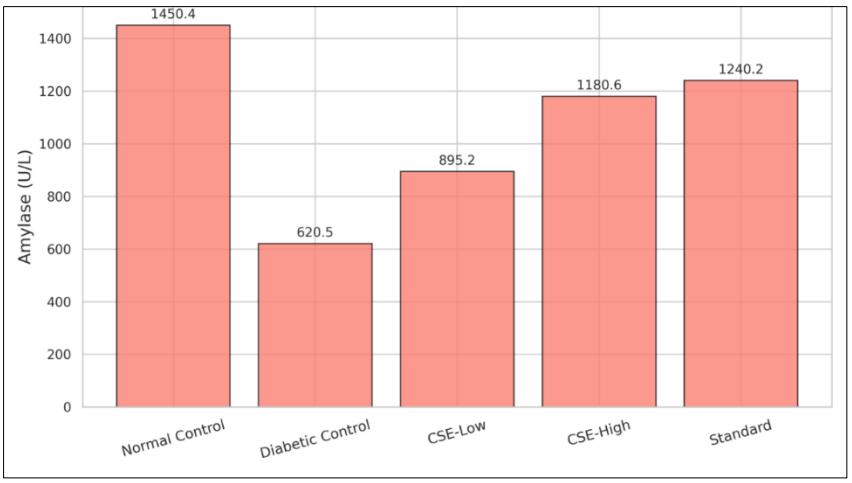

Figure 3: Serum amylase activity in study groups

3.4. Oxidative Stress in Pancreatic Tissue

The amounts of MDA in the pancreatic tissue in diabetic control group were noticeably elevated compared to normal control group, which suggested the increased lipid peroxidation. While, the levels of SOD and CAT activities as well as GSH content were reduced significantly. Treatment with CSE reversed these oxidative alterations by scavenging MDA as well as promoting the antioxidant defense system (SOD, CAT and GSH) in dose-dependent manner.

Table 3: Effect of CSE on Oxidative Stress Markers in Pancreatic Tissue

Group	MDA (nmol/mg protein)	SOD (U/mg protein)	CAT (U/mg protein)	GSH (µmol/mg protein)
Normal Control	1.24 ± 0.12	12.4 ± 1.1	24.8 ± 2.2	8.5 ± 0.6
Diabetic Control	4.85 ± 0.45*	4.2 ± 0.5*	8.6 ± 0.9*	2.1 ± 0.3*
CSE-Low (250 mg/kg)	3.12 ± 0.28#	7.5 ± 0.8#	14.2 ± 1.4#	4.6 ± 0.5#
CSE-High (500 mg/kg)	1.95 ± 0.18#	10.2 ± 0.9#	19.5 ± 1.8#	6.8 ± 0.7#
Standard (Metformin)	1.68 ± 0.15#	11.4 ± 1.0#	21.4 ± 1.9#	7.4 ± 0.8#

Data are expressed as Mean ± SD (n=10). *P < 0.05 compared to Normal Control; #P < 0.05 compared to Diabetic Control.

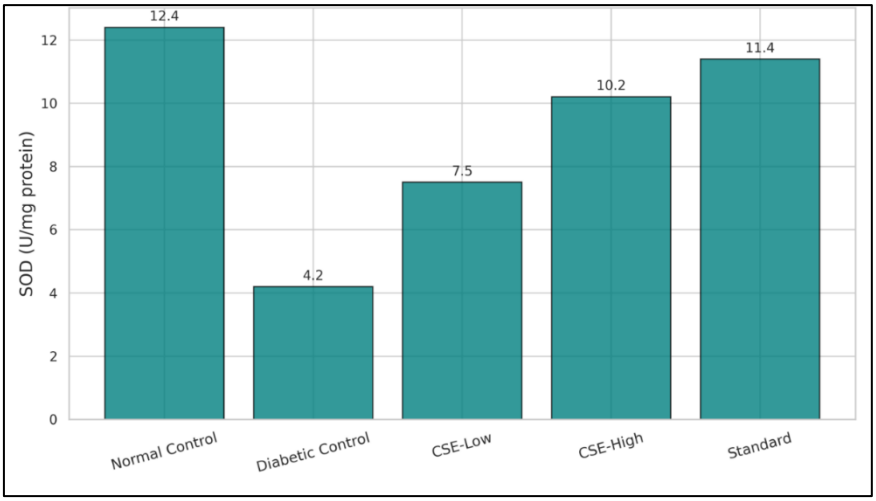

Figure 4: Pancreatic SOD activity

4. DISCUSSION

This study aimed to evaluate the protective effects of *Salvia hispanica* (chia) seed extract against streptozotocin-induced pancreatic damage in male Wistar rats. Results showed that CSE has significant anti-hyperglycemic and pancreatic-protective potential, as shown by the reversal of glycemic status, normalization of pancreatic enzyme activities, reduction in oxidative stress and histological preservation.

Streptozotocin, a widely used diabetogenic agent specifically kills pancreatic beta cells. Its mechanism consists in its intestinal absorption mediated by the GLUT2 transporter, DNA alkylation and ROS production that induce cellular stress and death (Szkudelski, 2001). In our experiment, the diabetic control rats showed classic features of pancreatic injury which included marked hyperglycemia, hypoinsulinemia and substantial body weight reduction. This weight loss observed in diabetic rats is usually due to exorbitant degradation of organic tissues, e.g., proteins and lipids by insulin deficiency. The treatment with CSE significantly inhibited weight loss and reduced blood glucose level, indicating that the extract either protects the beta cells from the STZ toxicity or causes a functional improvement of surviving cells.

An important finding in this study was the impact of CSE on exocrine pancreatic endpoints. Amylase and Lipase are necessary enzymes for digestion that may be elevated in case of injury to the pancreas. Although acute pancreatitis usually results in increase of activity of these enzymes, chronic injury or extensive beta cell destruction similar to advanced diabetes by STZ is frequently accompanied by a dramatic reduction in the activities reflecting exocrine pancreatic insufficiency (Madole *et al.,* 2016). Our findings are consistent with an apparent decrease of serum Amylase and Lipase levels in diabetic control group which was significantly restored by CSE intervention. These results suggest that the chia seed extract not only prevents radiation-induced damage in the endocrine cells of pancreas but also protects acinar cells functionally.

The beneficial effects of chia, may be related in part to its phytochemical composition. Chia is one of the best sources of alpha-linolenic (fatty acid), ALA that you can find, which boasts anti-inflammatory and antioxidant properties! ALA can also need to increase pancreatic and hepatic insulin sensitivity, and reduce lipids deposited in the pancreas and liver (Enes *et al.,* 2020). Also, our phytochemical screening showed the high content of both phenolic acids and flavonoids in CSE. Antioxidant activity may be due to phenolic compounds as caffeic acid, chlorogenic acid and quercetin; they are strong antioxidants that can remove free radicals and inhibit lipid peroxidation.

Oxidative stress is a central event in the pathogenesis of STZ-mediated damage to pancreas. The pancreas is susceptible to ROS damage because of its low level of antioxidant enzymes such as SOD, CAT, and GPx. This was confirmed in the present study by elevated levels of MDA (the product of lipid peroxidation) and decreased activities of SOD as well as CAT in pancreatic tissue from diabetic control rats. CSE treatment decreased MDA contents, but enhanced the antioxidant defense system to a large extent. This indicates that the action mechanism of CSE is to scavenge ROS produced by STZ, which prevents oxidative injury to cellular membranes and DNA in the pancreas.

The ameliorative impact of Salvia hispanica on the pancreas may be due to numerous interacting molecular pathways. One major one among them is through the regulation of Nrf2 (Nuclear factor erythroid 2-related factor 2) signaling. Nrf2, the master regulator of the antioxidant response, promotes a plethora of cytoprotective genes such as SOD, CAT and heme oxygenase-1 (HO-1) upon its activation. In view of the marked elevation in antioxidant enzyme activities reported here, it can be suggested that chronic exposure of chia phenolics (like caffeic acid and quercetin) are able to serve as Nrf2 activators leading to stimulation endogenous pancreas's defense against diabetogenic oxidative stress likely induced by STZ.

Additionally, the anti-inflammatory impact of chia seeds may be important in alleviating pancreatic injury. The STZ injection is known to induce an inflammatory process in the islets, involving macrophages infiltration and release of pro-inflammatory cytokines including TNF-α and IL-6. These cytokines can also intensify beta-cell apoptosis by activating the NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells) signaling pathway. This is particularly important considering the high alpha-linolenic acid (ALA) content in chia seeds, since omega-3 fatty acids block the NF-κB pathway and thereby decrease pro-inflammatory mediator production. By attenuating this inflammatory process, CSE could be protective against the chronic degradation of pancreatic structure.

An additional major mechanism includes the suppression of carbohydrate- digesting enzymes. Previous study showed that chia extracts can suppress α-amylase and α-glucosidase which would lead to delayed glucose absorbance from the GI tract (Mihafu, 2024). In this way, it not only aids the glycemic control but decreases secretory demand on already exhausted pancreatic beta cell and a concept of "beta-cell rest" is thereby facilitated. This decrease of task may favor recovery and regeneration of the endocrine tissue.

[column 3] Moreover, bioactive protein-derived peptides from chia have the ability of DPP-IV inhibition. DPP-IV - the enzyme that breaks down incretin hormones such as glucagon-like peptide-1

(GLP-1) which stimulate insulin release in a glucose-dependent manner. DPP-IV inhibition by chia fractions may be able to prolong GLP-1 half life and increase insulin secretion/beta cells survival. A such multi-approach targeting-effect (antioxidation, inflammation, and enzyme inhibition along with potential incretin modulation) renders Salvia hispanica an outstandingly powerful agent in holistic management of pancreatic disorders as well as diabetes.

5. CONCLUSION

In summary, the methanolic extract of *Salvia hispanica* seeds has a potential protective effect against STZ-induced pancreatic injury in male rats. The extract suppresses the hyperglycemic activity, enhances back the insulin levels, and normalises both amylase and lipase pancreatic activities. Mechanisms involved in these benefits are based on reducing of oxidative stress and improving antioxidant defense, as well as the maintenance of integrity in Langerhans islets. Due to an abundance of omega-3 fatty acids and phenolic antioxidants, the extraordinary nutritional-chia is offered as a functional food and may confer a protective effect with regard to prevention (even treatment) against pancreatic dysfunction; Chia also could potentially serve as adjuvant therapy associated chronic metabolic disorders. Additional clinical trials are needed to investigate the safety and efficacy of a chia seed extract in humans.

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