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Original Article

Antidiabetic and Antioxidant Potential of *Perunus dulcis* (Mill.)

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ABSTRACT

Background: Diabetes mellitus (DM) is a major public health ailment worldwide. The three main methods of treating diabetes are medication, diet therapy, and insulin therapy. However, these therapy not much significance due to their unavoidable disadvantages. Herbal drugs treatment is a noval strategy to treat DM (Type- II) due to their non-toxic effect. Fruit-producing herbal plants are crucial in the treatment of diabetes mellitus (DM). The main source of lipids, protein, dietary fibre, minerals, Vitamin E, mono-unsaturated fatty acids (MUFA), poly-unsaturated fatty acids (PUFA), arginine, and minerals is the oil of *Perunus dulcis* (almond). Carbohydrates, steroids, terpenoids, lipids, and other phenolic components were found in the oil upon phytochemical analysis. **Materials and Methods:** In Albino Wistar rats, a single intraperitoneal infusion of streptozotocin (50 mg/kg) caused overnight fasting rats to develop Non-insulin Dependent Diabetes Mellitus (NIDDM), after 15 min. of intraperitoneal administration of 100 mg/kg nicotinamide. If glucose level of rat is more than 200 mg/dL as Inclusion criteria in this study. In addition, enzymatic inhibition oxidative stress markers such as DPPH, H_2O_2 Radical Scavenging Activity Metal Ion Chelating Activity and Glucose Tolerance Test, Malondialdehyde (MDA), Reduced Glutathione (GSH), Catalase, and Superoxide dismutase (SOD) levels measured in liver tissue. Statistical analysis of carried out using one-way ANOVA was used to analyse the data, and then the Tukey post-hoc test. **Results:** Oil of kernals of *Perunus dulcis* (MILL) investigated against rats with Diabetes mellitus type II showed a significant reduction in (p<0.001) blood glucose level and inhibited key enzymes of carbohydrate metabolism i.e. α-amylase and α-glucosidase and also showed antioxidant activity. **Conclusion:** The findings of this study reflect that oil had antioxidant activities which showed the excellent potential of oil which was comparable to standard. Antioxidants may have a crucial role in the treatment of Diabetes Mellitus Type II (T2DM), since oxidative stress is linked to the pathophysiology of diabetes.

Keywords: Metformin, *Perunus dulcis*, Reactive Oxygen Species (ROS), Reduced Glutathione (GSH), Malondialdehyde (MDA), Streptozotocin (STZ), Super Oxide Dimustage (SOD).

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INTRODUCTION

A chronic metabolic condition involving the metabolism of carbohydrates, fats, and proteins is known as diabetes mellitus Type II (T2DM).[1] Reactive oxygen species (ROS) are produced at a higher rate during hyperglycemia, while antioxidant defences like MOD, SOD, etc. are suppressed. Additionally, deteriorating morphology in diabetic individuals and alterations in the enzymatic and functional markers of the liver and kidney were glaring signs of the loss in liver and kidney functionality.[2] The main treatments for managing diabetes include dietary therapy, pharmacology, and insulin therapy in addition to a wide range of glucose-lowering drugs that exert their hypoglycemic effects through various mechanisms.[3] Due to these therapies inherent drawbacks, such as medication resistance and toxicity, they are, nevertheless, of little consequence.

Due to the increased production of reactive oxygen species (ROS) and the inhibition of antioxidant defences as MOD, SOD, etc., hyperglycemia causes oxidative stress. Additionally, changes in liver and kidney enzymatic and functional indicators along with worsening morphology in diabetes subjects made the decline in their functionality clear.[2] Diet therapy, pharmacology, and insulin therapy are the main treatment choices available to control diabetes, in addition to a large range of glucose lowering medications that exert their hypoglycemic effects through various mechanisms.[3] These are only a few of the many therapeutic tactics. However, these therapies are of little consequence because of their inherent drawbacks, such as medication resistance and toxicity. Due to the fact that herbal-based medications are safe and non-toxic, supplementing these hypoglycemic drug to treat DM (Type II) is now a novel therapy method. Due to the fact that herbal-based medications are safe and non-toxic, supplementing

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these hypoglycemic pharmaceuticals to treat DM (Type II) is now a novel therapy method.[4] Many plants having fruits has antioxidant properties. From last few decades, natural antioxidants have widely used in all over the world instead of synthetic antioxidants due to their lower toxicity.[5] Several Indian fruits and their parts show low hypoglycaemic index.[6] The administration of insulin is to produce hypoglycaemia in subjects. This ensuing in widen demand for herbal products with effective antidiabetic effects and with minimal facet effect.[7] Oil of kernels of *Perunus dulcis (P. dulcis)* (Almond) are a nutrient-rich source of lipids, protein, dietary fibre, minerals,[8] vitamin E, monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), arginine, and potassium.[9-11] Almonds contain a variety of phenolic compounds, including flavonols (such as isorhamnetin, kaempferol, quercetin, catechin, and epicatechin), flavanones (such as naringenin), anthocyanins (such as cyanidins and delohinidin), procyanidins, and phenolic acids (such as caffeic acid, ferulic acid, p-coumaric acid, and vanillic acid).[12]

This research was aimed to determining the antidiabetic and antioxidant potential of *P. dulcis* oil effect on the destruction of liver β cells of diabetic rats.

MATERIALS AND METHODS

Plant Material and Preparation of the Extracts

The oil from kernels of *P. dulcis* were obtained by compressed method at room temperature and stored at same temperature.

Preliminary Phytochemical Screening

The extracts were screened for phytochemicals to identify the phyto constituents like alkaloids, steroids, terpenoids, fats, phenolic compounds, tannins, flavonoids and saponins: according to the prescribed method.[13]

Animals

Healthy, Albino Wistar rats (150-220g) of either sex were procured from Disease Free Small Animal House, Lala Lajpat Rai University of Veterinary and Animal Sciences, Hisar (Haryana). The animals were housed in cages at 25±1°C under standard laboratory conditions of humidity and temperature with light and dark cycle in every 24 hr. All the animals were acclimatized to laboratory environment for a week prior to experiment. The rats were kept on free access to food and water. The experimental protocol was duly approved by Institutional Animals Ethics Committee (IAEC) and animals care were strictly according with guidelines prescribed by Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Govt. of India (Registration No. 0436). The albino Wistar rats, who were in good health, were split into the following groups, each consisting of six rats. DM II was induced in rats of all groups excluding normal group.

Induction of Diabetes

Healthy, Albino Wistar rats (150-220g) of either sex were taken for study with free ingress to food and water. A single intraperitoneal infusion of streptozotocin (50 mg/kg) caused overnight fasting rats to develop Non-insulin Dependent Diabetes Mellitus (NIDDM), after 15 min. of intraperitoneal administration of 100 mg/kg nicotinamide.[14] The Inclusion criteria in this study was restricted to rats having blood sugar levels more than 200 mg/dL.

Drugs and chemicals

Ammonium molybedate and α amylase [S.D. Fine Chem., Ltd.,-Mumbai], butanol, carboxy methyl cellulose, citric acid, petroleum ether, disodium phosphate, 1,1-diphenyl-β-picrylhydrazyl (DPPH), ethylenediaminetetraacetic acid (EDTA), streptozotocin (STZ), (SRL, India), Ellman's reagent, ferric chloride, ferrozine, ferrous chloride, glacial acetic acid glucosidase, and acarobose (Siga Aldrich), hydrochloroacetic acid, malondialdehyde, thiobarbituric acid (CDH, India), metformin (Met.) (Cepla, Mumbai), methanol, monosodium phosphate, NADPH, napthylethlenediamine, nitrophenyl-α-Dglucopyranoside (PNGP), pyridine, potassium dihydrogen phosphate, soluble starch, iodine, nicotinamide (Hi-media Lab Pvt. Ltd., Mumbai), Sulphanilamide and Ethanol (SRL, India), sulphuric acid, trichloroacetic acid, tris buffer (CDH, India).

Acute Toxicity Studies

The oil extract was administered orally to the rats in four groups (n=2) at doses of 100, 120, 150, and 180 mg/kg body weight, respectively. They were monitored for behavioural changes over 24 hr and for any lethality for 48 and 72 hr. The oil test sample dose was determined to be efficacious at 120 mg/kg and was chosen for the further study.[15,16]

Experimental Design

Group 1: Normal rats were received vehicle (distilled water).

Group 2 (Diabetic control): Rats were administered vehicle only.

Group 3: Diabetic rats were administered Metformin (50 mg/kg; orally).

Group 4: Diabetic rats were administered orally with oil 120mg/kg.

The drug treatment was carried out every day morning with the help of intragastric tube (oral needle) orally for 4 weeks. After treatment of 28 days, the blood samples were taken from all groups in centrifuged tubes and Centrifugation at 3000-5000 rpm for 5-7 min was used to extract the serum and different tests like blood glucose and lipid profile were assessed in serum on semi autoanalyzer.[22]

In vitro Activity

α –Amylase inhibition activity

The published approach, with a few minor adjustments, was used to calculate the activity. Acarbose used as standard. In order to prepare the substrate, 500 mg of starch were dissolved in 25 mL of 0.4 M HCl, heated for 5 min at 100°C, and afterwards allowed to cool in an ice bath. With the help of 2M HCl the pH was adjusted to 7.4 and used distilled water to made up the volume upto 100 mL. In Phosphate buffer was used to prepare test samples at various concentrations (pH 7.4). Each microplate well received. The substrate and sample solutions were added to each microplate well in the quantities of 40 µl and 20 µl, respectively, and incubated at 37°C for 15 min. The plate was then incubated for a further 15 min with 20 µl of 50µg/mL α -amylase solution added to each well. The reaction was stopped by adding 200 µl of 1 mM iodine solutions and 80 µl of 0.1 M HCl. Utilizing the following equation, the blend's absorbance at 650 nm was measured to calculate its percentage inhibitory activity.

$$\%\text{inhibition} = \left[1 - \frac{\text{(absorbance of sample and substratse-absorbance of sample,substratse and amylase)}}{\text{(absorbance of substratse-absorbance of substratse and amylase)}}\right] \times 100$$

α- Glucosidase inhibition activity

The evaluation of α-glucosidase inhibition on a microplate was conducted using the standard method with a modest modification. 20 µL of enzyme solution (α-glucosidase, 0.2 U/mL), 170 µL of 0.1 M phosphate buffer (pH 7.0), and 20 µL of concentrates were combined. Following a 15-min incubation period at 37°C, the response was initiated by adding 20 µL of the substrate (2.5 mM p-nitrophenyl- α D-glucopyranoside). After this, 80 µL of 0.2 M Sodium carbonate was added to stop the process. Use 405 nm to measure the absorbance. The control contained methanol rather than test sample. As a control, a different set of blends was created with the same amount of phosphate cradle rather than enzyme. The formula below was used to calculate the enzyme inhibitory rates for the samples.

$$\text{Inhibition \%} = \frac{\text{absorbance of control-absorbance of sample}}{\text{absorbance of control}} \times 100$$

Antioxidant Assays

The antioxidant potential of oil of kernels of *P. dulcis* was analyzed by *in vitro* assays i.e., Metal chelating, hydrogen peroxide radical scavenging, and DPPH radical scavenging activities.

DPPH radical scavenging activity

1,1-diphenyl-β-picryl-hydrazyl radical scavenging (DPPH) assay was utilized for the evaluations of the radical scavenge capability of different antioxidant compounds.[20] Methanol stock solution (50mL), in various concentrations, was made in test tubes and 2 mL of 0.06 mm methanol solution of DPPH was added to each of the test tubes containing the samples. Using methanol as a blank, the sample solutions' absorbance was measured at 517 nm (Harborne, 1998). As a control, the absorption of DPPH radicals without antioxidants was employed. The standard was ascorbic acid. The formula was used to compute the % of inhibition.

$$\%\ \text{Inhibition} = \frac{\text{(absorbance of control-absorbance of test)}}{\text{(absorbance of control)}} \times 100$$

Hydrogen Peroxide (H_2O_2) Radical Scavenging Activity

Oil's reducing ability was assessed using the Hydrogen Peroxide (H_2O_2) assay 14. With 2.5 mL of 0.2 M phosphate buffer (pH 6.6) and 2.5 mL of 1% potassium ferricyanide [$K_3Fe(CN)_6$], various amounts of oil were added and incubated at 50°C for 20 min. The combination was given a dose (2.5 mL) of 10% trichloroacetic acid (TCA). The mixture was then centrifuged for 10 min at 3000 rpm. In order to evaluate absorbance at 700 nm in a UV-spectrophotometer, 2.5 mL of the supernatant, 2.5 mL of distilled water, and 0.5 mL of freshly made 0.1% $FeCl_3$ solution were added. An rise in the reaction mixture's absorbance served as confirmation of the improved reducing power. The standard was ascorbic acid. The following formula was used to calculate the percentage of inhibition.

$$\%\ \text{Increase in reducing power} \left[\frac{\text{absobance of test}}{\text{absorbance of sample}} - 1\right] \times 100$$

Metal Ion Chelating Activity

The extract was estimated to chelate ferrous and ferric ions21. Samples in DMSO were prepared at various concentrations, and the volume was increased to 1 mL using methanol. 50 µl of $FeCl_3$ (2 mM) and an additional 3.7 mL of methanol were mixed. 5mM Ferrozine (0.2 mL) was added to the mixture to begin the reaction. The mixture was then vigorously agitated and allowed to stand at room temperature for 10 min. The pink violet solution's absorbance was then calculated spectrophotometrically at 562 nm. The following formula was used to determine the barrier percent for the production of the ferrozine-Fe^{2+} complex.

$$\text{Inhibition \%} = \frac{\text{(absorbance of control- absorbance of sample)}}{\text{(absorbance of control)}} \times 100$$

Glucose Tolerance Test

After treatment of 27 days, the blood samples were collected from all groups at varying intervals of time i.e., 0, 30, 60, 90 and 120

min from tail vein on day 28 after the administration of 2g/kg glucose. Then blood glucose level was estimated *via* glucometer.

Estimation of Serum Glucose

By using the Erba Mannhein Germany Chem 5 Plus-V2 Auto-analyser and a commercially available biological kit called the Erba Glucose Kit (GOD-POD Method), the precise level of glucose in the serum sample was determined. The glucose readings were calculated as a blood sample of mg/dL.[23]

Glucose Estimation

Measurement of glucose in blood serum is essential for diabetes, hypoglycaemia and several other conditions. Blood glucose level is measured by GOD-POD method[24] using Standard Glucose Kit (Erba). Glucose is stable in serum for 24 hr at temp 2-8°C.

RESULTS

Phytochemical Screening

Phytochemical screening carried out on the oil (test sample) of kernels of *Prunus dulcis (*Mill.) D.A. Webb, (*Prunus dulcis).* The Oil of kernels of *Prunus dulcis* (Mill) D.A. WEBB possess various phyto-constituents like carbohydrates, sterols, saponins, phenolic compounds, flavonoids, resins and terpenoids (Table 1). Terpenoids have various therapeutic effects against cancer, malaria, inflammation and many infectious diseases.[25] Phenolic compound have shown antibacterial activities.[26] Sterols, saponins, have antifungal and antiviral properties.[27] Flavonoids showed various actions against cancer, strock and heart disease.[28]

α-amylase and α-glucosidase Inhibition Activity

The IC_{50} value of Acarbose and Oil of *P. dulcis* were determined and a significant (p<0.001) inhibition of α-amylase and α-glucosidase when compared to Acarbose (standard). The IC_{50} value of Acarbose and Oil were found to be 60.29±1.79, 120.6±2.63 for α-amylase and 39.50±1.24; 121.85±2.21 µg/mL for α-glucosidase, respectively.

Antioxidant Assays

DPPH Radical Scavenging Activity

The action of DPPH radical scavengers of oil of kernels of *P. dulcis* was measured and compared with ascorbic acid (standard). The Oil expressed significant (p<0.001) scavenging of DPPH radicals as compared to diabetic control. The IC_{50} values of ascorbic acid and oil were 28.28±1.04 and 101.01±1.47 µg/mL, respectively.

Hydrogen peroxide (H_2O_2) radical scavenging activity

Excellent hydrogen peroxide radical scavenging was achieved by the oil, and oil from *P. dulcis* kernels significantly (p<0.001) decreased the H_2O_2 radicals when compared to ascorbic acid. The IC_{50} values of ascorbic acid and oil were 44.93 ±0.61 and 104.69±0.92µg/mL, respectively.

Metal chelating activity

In comparison to ascorbic acid, which was used as the reference, *P. dulcis* oil demonstrated metal chelating properties and caused significant (p<0.001) chelation. IC_{50} (µg/mL) values were found to be 77.06±1.41 and 201.89±1.38 of ascorbic acid and oil, respectively.

In vivo activity

Every value is presented as Mean ± SEM. A one-way ANOVA was used to analyse the data, and then the Tukey *post hoc* test.

Blood Glucose Level

The antidiabetic potential was evaluated by checking the blood glucose level at day 14 and day 28 after induction of diabetes mellitus (Table 2).

Antioxidant Activity

Malondialdehyde (MDA), Reduced Glutathione (GSH), Catalase, and Superoxide dismutase (SOD) levels in liver tissue at day 28th are measures of antioxidant activity (Table 3).

The repeated oral administration of different treatments i.e., Metformin and oil for 28 days caused a significant (p<0.001) increase in total protein levels compared to the group of diabetics. Oil has shown profound action. The total protein content in liver is appeared in Table 3. Rats with diabetes had considerably (p<0.001) higher MDA concentration in their liver tissue (6.16±0.045 nmoles/mg protein) in contrast to normal (2.09±0.030 nmoles/mg protein). The recurrent administration of oil of *P. dulcis* for 28 days were caused decline in the level of MDA as compared to diabetic control. The MDA content of oil was found to be 5.31±0.033 nanomoles/mg protein. The decrement in the level of GSH is directly proportional to the increment in oxidative stress. The results of the present investigation showed that a considerable (p<0.001) lower in GSH content in diabetic rats (5.57±0.16 µg/mg protein) as compared to normal (11.68±0.34 µg/mg protein). A rise in GSH levels in treated groups compared to diabetic controls shows that the harm done by free radicals has been lessened. Superoxide dismutase (SOD) and catalase, two antioxidant enzymes, are essential in protecting cells and tissues from reactive oxidative radical damage.

DISCUSSION

The present study was to investigate antidiabetic and antioxidant potential of oil of kernels of *Prunus dulcis* in diabetic rats. The carbohydrate hydrolyzing enzymes i.e., α-glucosidase and α-amylase plays the key role in the genesis of hyperglycaemia. α-glucosidase is a key enzyme in the digestion of carbohydrates.[29,30]

Table I : Preliminary Phytochemical Screening of *Prunus dulcis*

Sr. No.	Plant constituents Test reagents	Test Sample (Oil)
1.	**Alkaloids**	
	Mayer's reagent	-
	Dragendroff's Reagents	-
	Hager's reagent	-
	Wagner's reagent	-
2.	**Carbohydrates**	
	Fehling's test	+
	Benedict's test	-
3.	**Sterols**	
	Liebermann-Burchard test	+
	Salkowski test	+
4.	**Saponins**	
	Faom test	+
5.	**Phenolic compounds and tannins**	
	Ferric chloride test	-
	Lead test	+
	Potassium dichromate test	+
	Gelatin solution test	+
	Potassium permagenate test	+
	Iodine test	+
	Silver mirror test	+
6.	**Flavonoids**	
	Shinoda/Pew test	+
	Ammonia test	+
7.	**Resins**	
	Saponification Value	+
	Iodine Value	+
8.	**Terpenoids**	
	Sulphur powder test	+

+ : indicates the presence of constituents;
- : indicates the absence of constituents

Thus, the suppression of the activity of both enzymes is being considered to be efficacious in T2DM. In the present experimental design, *in vitro* enzymatic studies showed that oil of kernels of *P. dulcis* caused significant (p<0.001) inhibition of α-amylase and α-glucosidase when compared to acarbose which was taken as standard. The IC_{50} values of acarbose and oil of *P. dulcis* were 60.29±1.79 and 400.96±3.95 for α-amylase and 39.50±1.24; 397.38±3.45 for α-glucosidase respectively. Earlier studies suggested that medicinal plants having good content of polyphenols responsible for the inhibition of α-amylase and α-glucosidase inhibitory activities.[31,32] The phytochemical screening showed the presence of polyphenols in *P. dulcis* was

Table 2: Effect of oil on blood glucose level in experimental groups.

Experimental Groups	Blood glucose level (mg/dl)	
	Day 14	Day 28
Normal	81.83±1.16	82.51±1.10
Diabetic Control (DC)	241±0.60	277.5±1.91
Metformin (Met.)	93±0.98	120.5±1.33
Oil	137.5±2.63	182.16±1.19

Values are mean±SD (n=6). Statistical comparison between values of different extracts on 28th day was done using by one way ANOVA followed by Tukey *post hoc* test (p<0.001).

responsible for the remarkable inhibition of both carbohydrate hydrolyzing enzymes. The increase in blood glucose level (277.5±1.91 mg/dL) might be due to decrement in the secretion of insulin as a result of destruction of pancreatic β-cells by STZ action. The oil of *P. dulcis* caused significant (p<0.001) reduction in blood glucose level (182.16±1.19 mg/dL) as compared to diabetic control. Previous studies reported that several medicinal plants contains polyphenolic compounds were found to significantly inhibit the sodium dependent transport of in intestinal epithelial cells.[33] It is widely published that the good antioxidant activity could be one of the mechanisms responsible in the prevention of diabetic complications.[34]

Metformin was used as a standard (reference) drug. It inhibits gluconeogenesis, by enters into liver cell via OCT1 and suppresses electron transport chain in mitochondria resulted into insufficient energy production which is maintained by decreasing the utilization of energy.[35]

Excess blood glucose level leads to stimulate the production of reactive oxidative radicals by different pathways leads to cellular damage via destruction of DNA, lipids and proteins.[36] Because oxidative stress is associated with the pathogenesis of diabetes, therefore, it can be suggested that the antioxidants may exert a key role in the management of T2DM.[37]

Earlier studies exhibited the strong correlation between the polyphenols and DPPH radical scavenging activity. The oil of kernels of *P. dulcis* showed significant (p<0.001) scavenging of DPPH radicals as compared to diabetic control. The IC_{50} values of acarbose and oil of *Prunus dulcis* were 28.28±1.04; 101.01±1.47 µg/mL respectively.

The oil of *P. dulcis* exhibited metal chelating properties and caused significant quenching of Iron as compared to diabetic control group.[38] IC_{50} (µg/mL) values were found to be 77.06±1.41 and 201.89±1.38 of ascorbic acid and oil respectively.

Increased oxidative glucose metabolism increases mitochondrial production of the superoxide anion, which is converted to the hydroxyl radical and hydrogen peroxide. The scavenging of hydrogen peroxide radicals by the oil of *P. dulcis* produced more profound action as compared to standard as IC_{50} (µg/mL) were

Table 3: Effect of plant extracts of Total Protein, MDA, Reduced Glutathione (GSH), Superoxide Dismutase (SOD) and Catalase level on liver in experimental groups.

Experimental groups	Total protein (mg/dl)	MDA (nmoles/mg Protein)	GSH (µg/mg protein)	SOD (µg/mg protein)	Catalase (µmoles/ mg protein)
Normal	63.04±0.83	2.09±0.030	11.68±0.34	3.3±0.08	9.06±0.05
DC	28.56±0.43	6.16±0.045	5.57±0.16	0.9±0.02	3.87±0.03
Met.	54.45±0.65	2.90±0.030	10.16±0.17	2.4±0.06	7.87±0.03
Oil	43.31±0.51	5.31±0.033	6.83±0.039	1.5±0.03	5.58±0.02

Values are mean±SD (n=6). Statistical comparison between values of extracts was done using by one way ANOVA followed by Tukey *post hoc* test (p<0.01).
nmoles/mg = nanomoles per miligram
µg/mg = Microgram per miligram
µmoles/ mg = Micromoles per miligram

found 44.93 ±0.61 and 104.69±0.92 for ascorbic acid and Oil, respectively. It was observed that a lot of differences were found when *in vitro* data correlated to *in vivo* studies.[39] Therefore, *in vivo* antioxidant studies were also performed for evidence of antioxidant potential of oil of kernels of *P. dulcis*. Malondialdehyde (MDA) is a biomarker of lipid peroxidation, produced from the degradation of polyunsaturated lipids by reactive oxygen radicals. The level of MDA was increased in hyperglycaemic condition, indication of an oxidative stress condition. Results showed that MDA content significantly (p<0.001) rise in liver tissue in diabetic rats (6.16±0.045 nanomoles/mg protein) as compared to normal (2.09±0.030 nanomoles/mg protein). The oil of *P. dulcis* had shown remarkable effect as oil was found to be 5.31±0.033 nanomoles/mg protein respectively.

The decrease in the level of GSH is directly proportional to the increment in oxidative stress. The results of the present investigation showed that a significant (p<0.001) decrease in GSH content in diabetic rats. A marked fall (p<0.001) was observed in SOD and catalase content in diabetic rats as compared to normal. The repeated administration of oil of *P. dulcis* for 28 days resulted into a significant rise in SOD and catalase as compared to diabetic control. Further study may also be needed to investigate the involvement of other factor which contributing to the antidiabetic potential of plant extract. The said effect produced by the plant extract may be attributed to potential action.

CONCLUSION

The antidiabetic and antioxidant property of Oil of kernels of *P. dulcis* were evaluated by giving chronic treatment to rats. The present study was done for the assessment of *Prunus dulcis* on STZ-Nicotinamide induced type II diabetic rats. The Oil of *P. dulcis* contained alkaloids, carbohydrates, sterols, saponins, phenolic compounds, tannins, and flavonoids, based on the preliminary phytochemical screening investigation. Antioxidant activities such as DPPH radical scavenging activity, hydrogen per-oxide method and metal ions chelating activity showed the excellent potential of oil which was comparable to standard. The antidiabetic potential was evaluated by biochemical parameters like blood glucose estimation, and total protein, malondialdehyde, glutathione, catalase, and super oxide dismutase levels.

Oil has a great potential to protect and regenerate β-cells of islets of Langerhans, blood vessels, vacuoles and septae of the diabetic rats. Overall it is concluded that, Oil was found to be having excellent potential to protect the animals from the destructive effect of the STZ. This study has found that the oil has antioxidant and anti-diabetic properties.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVATIONS

CPCSEA: Committee for the Purpose of Control and Supervision of Experiments on Animals; **DM:** Diabetes mellitus; **DMSO:** Dimethyl Sulfoxide; **DPPH:** 1,1-diphenyl-β-picrylhydrazyl; **EDTA:** Ethylenediaminetetraacetic acid; **GSH:** Reduced Glutathione; **H_2O_2:** Hydrogen Peroxide; **IAEC:** Institutional Animals Ethics Committee; **IC_{50}:** Half-maximal inhibitory concentration; **T2DM:** Diabetes mellitus Type II; **MDA:** Malondialdehyde; **Met:** Metformin; **MUFA:** mono-unsaturated fatty acids; **NADPH:** Napthylethlenediamine; **NIDDM:** Non-insulin Dependent Diabetes Mellitus; ***P. dulcis***: *Perunus dulcis;* ***Perunus dulcis:*** Almond; **PNGP:** Nitrophenyl-α-Dglucopyranoside; **PUFA:** poly-unsaturated fatty acids; **ROS:** Reactive Oxygen Species; **SOD:** Superoxide dismutase; **STZ:** Streptozotocin; **TCA:** Trichloroacetic acid.

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