Antioxidant effect of *Lagerstroemia speciosa* Pers (Banaba) leaf extract in streptozotocin-induced diabetic mice

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Aqueous leaf extract of *L. speciosa* (banaba) effectively decreased the blood glucose in streptozotocin-induced diabetic mice after 15th day of banaba exposure. Further, banaba leaf extract have the potential to inhibit lipid peroxidation and effectively intercept/neutralize reactive oxygen species such as super oxide, $H_2O_2$ and NO based free radicals. The aqueous banaba leaf extract (150 mg/kg bodyweight) duly reduced STZ generated reactive intermediates and radical species helping to regulate normal levels of antioxidative markers like superoxide dismutase, catalase, glutathione-S-transferase and reduced glutathione.

Keywords: Diabetes, Free radicals, *Lagerstroemia*, Oxidative stress

Diabetes mellitus (DM) is characterized by abnormal insulin secretion, derangements in carbohydrate/lipid metabolism and is diagnosed by hyperglycemia. The prevalence of diabetes is increasing worldwide, with an approximate doubling of new cases predicted to occur by the year 2025\(^1,2\). This multi-factorial disease is hypothesized to damage cell membranes resulting in elevated production of reactive oxygen species (ROS) creating oxidative stress. An increase in ROS, an impairment of antioxidant defence systems or an insufficient capacity to repair oxidative damage, the three factors contributing to oxidative stress, appears to play a critical role in pathogenesis. The major concern with diabetes clearly relates to marked increase of neuropathy and series morbidity and mortality related to the development of other complications\(^3\).

Tropical plant *Lagerstroemia speciosa* Pers., (Lythraceae) also called banaba and found in India, Philippines, southern China, Malaya and tropical Australia, has been used as a folk medicine for the treatment of diabetes and kidney diseases\(^5\). Hypoglycemic activity of banaba extract was studied in genetically induced diabetic type II KK-Ay mice\(^6\). Originally, because of its hypoglycemic activity, banaba leaf was thought to contain “insulin-like principle”, a type of plant – derived peptide hormone. However, there is a dearth of literature supporting the antioxidant potential and oxidative status on consumption of the banaba leaf extract. In light of this, the study is designed to evaluate the *in vitro* antioxidant activity of banaba leaf extract as well as *in vivo* antioxidant status in the liver and brain tissue samples following the administration of graded doses of banaba extract in STZ-diabetic mice.

Materials and Methods

**Plant material**—Standardized aqueous leaf extract of *Lagerstroemia speciosa* (banaba), having 1% Corosolic acid fraction, was procured from Changsha Botaniex Inc, China.

**Chemicals**—Streptozotocin (STZ), 2, 2′-azinobis-[3-ethylbenzthiazoline-6-sulfonic acid] (ABTS), 6-hydroxy-2, 5, 7, 8-tetramethylchroman-2-carbonic acid (TROLOX) were obtained from Sigma Aldrich. Butylated hydroxy toluene (BHT) was purchased from Merck India Ltd. All other chemicals were of analytical grade.

**Animals**—Adult (3- months-old) albino mice, *Mus musculus*, weighing 30±5g were procured from Sri Raghavendra Enterprises, Bangalore and acclimatized to laboratory conditions (12:12h dark/light, 25° ± 2°C). Standard mice pellet diet was given *ad libitum*; the animals were maintained in accordance with the guidelines of National Institute of Nutrition, ICMR Hyderabad and approved under Institutional Animal Ethical Committee Bangalore University, Bangalore.
**Induction of experimental diabetes**—A freshly prepared solution of streptozotocin (50 mg/kg body weight) in 0.1 mol/L citrate buffer, pH 4.5 was injected intraperitoneally in a volume of 1 mL/kg body weight. Five days after streptozotocin administration, mice with moderate diabetes having hyperglycemia (110-120 mg/dL) were used for the experiment.

**Experimental design**—To check the dose response on hyperglycemia, five grades of aqueous banaba leaf extract (BLE) in 0.1 mL having 50, 100, 150, 250, 500 mg/kg body weight/day for two months were given in the pilot study to diabetic animals using intra-gastric tube on 5th day of STZ administration respectively. To assess the anti-hyperglycemic effect of BLE, blood was collected by tail vein puncture from overnight fasted animals and the blood glucose levels were measured on 5th, 10th, 15th, 30th, 45th and 60th day of banaba exposures.

In the second set, to check the dose response efficacy of grades of BLE (50-500 mg/kg body weight/day) on oxidative stress indices in liver and brain tissues, the animals were sacrificed after 15 days of BLE exposure as the extract has shown antihyperglycemic effect on the 15th day of BLE exposure. Dissected liver and brain tissues were washed in ice-cold saline, patted dry and used for biochemical assays.

**In vitro antioxidant activity**

Trolox equivalent antioxidant capacity (TEAC assay)—The antioxidant activity by the TEAC assay was determined as per Re et al., using the radical cation ABTS**. The ABTS** radical cation was pregenerated by mixing 7 mM ABTS stock solution with 2.45 mM potassium persulfate (final concentration) while incubating for 12–16 h in dark at room temperature until the reaction was complete and the absorbance was stable. The working solution of ABTS** was obtained by diluting the stock solution in phosphate buffer saline (PBS, pH 7.4) to give an absorption of 0.70±0.02 at \(\lambda = 734\) nm. ABTS working solution (about 3 mL) was mixed with 10 µL of the Trolox standard (50 mM) or the test samples (0.05-10 mg/mL) and the absorbance was measured at every 10 sec for 6 min at 734 nm. All experiments were repeated six times.

The TEAC value was calculated by measuring the area under curve, derived from plotting the percentage inhibition of the absorbance as a function of time. The calculation of the area under the curve was performed for one sample dilution which had a final percentage inhibition between 20 and 80%. Each extract was analysed in triplicate. The antioxidant activity of the plant extracts was expressed as µmol of TE/g weight of the extract.

Nitric oxide scavenging assay—The activity was measured according to the modified method of Sreejayan and Rao. To 4 mL of the extract having different concentrations of banaba (1-50 µg/mL) were added 1 mL of sodium nitroprusside (SNP) solution (5 mM) and incubated for 2 h at 27°C. An aliquot (2 mL) of the incubation solution was removed and diluted with 1.2 mL of Griess reagent (1% sulphanilamide in 5% H3PO4 and 0.1% naphthylethylene diamine dihydrochloride). The absorbance of the chromophore was read immediately at 550 nm and compared with standard, BHT.

Scavenging activity (%) = \(\frac{[\text{Abs (control)} - \text{Abs(test)}]}{\text{Abs(control)}}\)×100

where, Abs (control): Absorbance of the control and Abs (test) : Absorbance of the extract/standard.

Superoxide scavenging assay—The activity was evaluated using nitro blue tetrazolium (NBT) reduction method.

**H2O2 radical scavenging assay**—The ability of the extract to scavenge \(\text{H}_2\text{O}_2\) was determined according to the method by Ruch et al.

Reducing power assay—The reducing power of the BLE was determined according to the method of Oyaizu et al.

Determination of plasma glucose level—Fasting blood glucose was estimated by O-toluidine method.

**In vivo antioxidant assays**—Oxidative stress markers viz lipid peroxidation (LPO), superoxide dismutase (SOD), catalase (CAT), reduced glutathione (GSH) and glutathione-S-transferase (GST) were assayed. Protein was estimated by the method of Lowry et al., using bovine serum albumin as standard.

**Statistical analysis**—Results are expressed as mean ± SD of 6 observations. Data compilation was carried out using SPSS 15.0 software. Under in vitro assays, linear regression analysis was used to calculate the IC50 values. Data from dose-response study of BLE on hyperglycemia were analysed using two way univariate ANOVA by Duncan multiple range test (DMRT) post hoc to examine.
the effective day and dose of BLE exposure as well as One way Analysis of Variance (ANOVA) to examine effective dose on each day examined and the data from in vivo biochemical studies by employing one way ANOVA followed by Bonferroni post hoc.

Results

The total antioxidant activity of the extract was calculated from the decolorization of ABTS** which was measured spectrophotometrically at 734 nm. The TEAC assay results, using the standard curve of Trolox standard on ABTS (R² = 0.865), showed the total antioxidant activity of BLE (1% corosolic acid) was 0.012 µmol of TE/g wt extract.

Percentage inhibition on superoxide radical generation by the BLE was found increasing in a dose dependent manner, showing the IC₅₀ value of 1.10 mg/mL, when compared to the IC₅₀ value 3.35 µg/mL of ascorbic acid. With regard to scavenging of H₂O₂ and NO, the different concentrations of BLE showed decrease in percentage inhibition while increase in the concentrations with IC₅₀ value of 14.95 mg/mL (IC₅₀ of ascorbic acid -1.23µg/mL) and 0.23 g/mL (IC₅₀ of BHT 0.15 µg/mL) respectively, and in case of the total reduction capability, IC₅₀ value 2.49 mg/mL, exhibited an increase in a dose dependent manner (Fig. 1).

On post administration of banaba extract to diabetic models, it initially showed no effect up to 10 days exposure whereas the serum glucose levels decreased significantly (*P < 0.05) on 15th day of extract administration and further remained constant in 30th, 45th, and 60th days of BLE exposure, indicating the hypoglycemic properties of the leaf extract of *L. speciosa*, as the substances with hypoglycemic properties would be effective in the management of diabetes (Table 1). Statistical analysis using two way univariate analysis demonstrated that the serum glucose levels reduced at a dose of 150 mg/kg body weight suggesting the ameliorative role of BLE on the 15th day of exposure in extending protection to diabetic animals, compared to other doses. TBARS from liver and brain samples in BLE treated groups were significantly decreased, especially at 150 mg/kg body weight (Table 2). In both liver and brain samples, the levels of SOD and GST exhibited significant decrease and CAT activity and GSH levels significantly increased on banaba exposure, when compared to that of diabetic control mice. The dose response studies made on serum glucose as well as tissue antioxidant status showed that aqueous extract of banaba has significant effect after 15 days of exposure and the extent of increase was higher in group treated with 150 mg/kg body weight than other groups.

![Image](image-url)

**Fig. 1—Antioxidant potential of aqueous leaf extract of *L. speciosa* (banaba)**

| Table 1—Effect of banaba leaf extract (50-500 mg/kg bw/day) on blood glucose levels at 5th, 10th, 15th, 30th, 45th and 60th day in diabetic mice |

<table>
<thead>
<tr>
<th>Groups</th>
<th>5th day</th>
<th>10th day</th>
<th>15th day</th>
<th>30th day</th>
<th>45th day</th>
<th>60th day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>55.69 ± 6.74†</td>
<td>54.68 ± 3.68†</td>
<td>58.02 ± 1.31†</td>
<td>56.82 ± 2.33†</td>
<td>56.41 ± 4.44†</td>
<td>58.11 ± 3.32†</td>
</tr>
<tr>
<td>STZ</td>
<td>118.58 ± 0.99*</td>
<td>122.75 ± 16.73*</td>
<td>119.75 ± 2.2*</td>
<td>121.58 ± 11.15*</td>
<td>119.75 ± 5.56*</td>
<td>119.75 ± 5.56*</td>
</tr>
<tr>
<td>STZ + B 50</td>
<td>119.25 ± 7.75*</td>
<td>109.34 ± 6.37*</td>
<td>109.34 ± 0.80*</td>
<td>111.03 ± 3.63*</td>
<td>112.75 ± 6.12*</td>
<td>112.75 ± 3.63*</td>
</tr>
<tr>
<td>STZ + B 100</td>
<td>120.92 ± 3.57*</td>
<td>111.03 ± 4.86*</td>
<td>111.03 ± 2.07*</td>
<td>111.03 ± 4.86*</td>
<td>111.03 ± 4.86*</td>
<td>111.03 ± 4.86*</td>
</tr>
<tr>
<td>STZ + B 150</td>
<td>120.25 ± 4.08*</td>
<td>112.75 ± 4.02*</td>
<td>112.75 ± 2.81*</td>
<td>112.75 ± 2.81*</td>
<td>112.75 ± 2.81*</td>
<td>112.75 ± 2.81*</td>
</tr>
<tr>
<td>STZ + B 250</td>
<td>114.08 ± 4.36*</td>
<td>112.38 ± 5.83*</td>
<td>112.38 ± 0.59*</td>
<td>112.38 ± 0.59*</td>
<td>112.38 ± 0.59*</td>
<td>112.38 ± 0.59*</td>
</tr>
<tr>
<td>STZ + B 500</td>
<td>118.92 ± 5.46*</td>
<td>115.15 ± 4.83*</td>
<td>115.15 ± 0.47*</td>
<td>115.15 ± 0.47*</td>
<td>115.15 ± 0.47*</td>
<td>115.15 ± 0.47*</td>
</tr>
</tbody>
</table>

STZ = streptozotocin; B = banaba leaf extract

*P<0.05 as compared to * control mice; † diabetic controls
concentrations (1-10µg) showed less effective in H
hydroxyl radical. In this study, BLE in increasing
acts as toxicant to cell by converting itself to
membranes and being non-reactive, where it
important because of its ability to penetrate biological
antioxidant potential of the banaba leaf extract.

H
consumption of superoxide anion in the reaction
curve (R
red. The calculated results with trolox standar d
activity

activity of an extract or compound cannot be

Antioxidant activity; however, the total antioxidant
activity, ABTS and potassium persulfate and in the presence of
the plant extract or trolox, preformed cation radical gets
reduced. The calculated results with trolox standard
curve (R² = 0.865) and the TEAC value explains the
antioxidant potential of the banaba leaf extract.

The superoxide anion radicals are derived in
the pathological conditions, reacts with superoxide
anion and form potentially cytotoxic molecule
'peroxynitrite'. Comparatively, the BLE is found to be
less scavenging on nitric oxide, when compared to
ascorbic acid, the reference compound. The reducing
capacity of any compound may serve as a significant
indicator of its potential antioxidant activity.
For measurements of the total reduction ability,
the Fe²⁺ to Fe³⁺ transformation in the presence of
BLE was investigated and an increasing absorbance
was found in a dose dependent manner, with
increasing concentration, and confirmed significant
reducing ability.

Streptozotocin (STZ), a diabetogenic agent, is well
known for its selective pancreatic islet beta cell
cytotoxicity by damaging the beta cell membrane and
breaks the DNA strand leading to the activation of
poly (ADP-ribose) synthetase and NAD depletion
The decomposition of STZ leads to formation of
highly reactive carbonium ions which cause
alkylation of DNA bases causing cell death. It’s
observed that in post STZ – injury, the surviving beta
cells are able to maintain most of the metabolic
functions but fail to maintain an adequate blood
sugar levels. Monitoring the antihyperglycemic effect
in the present study, the efficacy of plant extract on
diabetic model was measured for two months. While
exposure to BLE, initially up to 10 days showed
was found in a dose dependent manner, with
increasing concentration, and confirmed significant
reducing ability.

The superoxide anion radicals are derived in
PMS-NADH-NBT system, where the decrease in
absorbance at 560 nm with BLE indicates the
consumption of superoxide anion in the reaction
mixture, thereby exhibiting a dose dependent increase
in superoxide scavenging activity. H₂O₂ is highly
important because of its ability to penetrate biological
membranes and being non-reactive, where it
acts as toxicant to cell by converting itself to
hydroxyl radical. In this study, BLE in increasing
concentrations (1-10µg) showed less effective in H₂O₂
scavenging activity. Similarly, nitric oxide (NO),
being a potent pleiotropic mediator of many
physiological processes and a diffusible free radical in
the pathological conditions, reacts with superoxide
anion and form potentially cytotoxic molecular

Discussion
There are numerous methods for evaluation of
antioxidant activity; however, the total antioxidant
activity of an extract or compound cannot be
evaluated by using a single method, due to oxidative
processes. Therefore, at least two methods should be
employed in order to evaluate the total antioxidant
activity. In the total antioxidant activity, ABTS⁺ is a
blue chromophore produced by the reaction between
ABTS and potassium persulfate and in the presence of
the plant extract or trolox, preformed cation radical gets
reduced. The calculated results with trolox standard
curve (R² = 0.865) and the TEAC value explains the
antioxidant potential of the banaba leaf extract.

<table>
<thead>
<tr>
<th>Groups</th>
<th>LPO (µM of MDA/g tissue)</th>
<th>SOD (IU/mg protein)</th>
<th>CAT (IU/mg protein)</th>
<th>GST (IU/mg protein)</th>
<th>GSH (mg of GSH/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>A 0.34 ± 0.009</td>
<td>0.65 ± 0.01</td>
<td>0.80 ± 0.02</td>
<td>0.18 ± 0.004</td>
<td>4.29 ± 0.15</td>
</tr>
<tr>
<td></td>
<td>B 0.89 ± 0.03</td>
<td>0.03 ± 0.002</td>
<td>0.19 ± 0.01</td>
<td>0.41 ± 0.01</td>
<td>1.08 ± 0.01</td>
</tr>
<tr>
<td>STZ</td>
<td>A 1.40 ± 0.02</td>
<td>0.84 ± 0.01</td>
<td>0.08 ± 0.01</td>
<td>0.71 ± 0.01</td>
<td>2.78 ± 0.08</td>
</tr>
<tr>
<td></td>
<td>B 1.69 ± 0.03</td>
<td>0.07 ± 0.003</td>
<td>0.07 ± 0.01</td>
<td>1.32 ± 0.03</td>
<td>0.85 ± 0.15</td>
</tr>
<tr>
<td>STZ + BLE 50</td>
<td>A 0.55 ± 0.01</td>
<td>0.32 ± 0.01</td>
<td>0.15 ± 0.01</td>
<td>0.57 ± 0.002</td>
<td>6.84 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>B 0.61 ± 0.02</td>
<td>0.05 ± 0.004</td>
<td>0.23 ± 0.01</td>
<td>0.56 ± 0.02</td>
<td>1.29 ± 0.04</td>
</tr>
<tr>
<td>STZ + BLE 100</td>
<td>A 0.85 ± 0.08</td>
<td>0.52 ± 0.02</td>
<td>0.38 ± 0.02</td>
<td>0.13 ± 0.008</td>
<td>3.75 ± 0.06</td>
</tr>
<tr>
<td></td>
<td>B 1.11 ± 0.06</td>
<td>0.03 ± 0.003</td>
<td>0.14 ± 0.01</td>
<td>0.37 ± 0.04</td>
<td>1.1 ± 0.03</td>
</tr>
<tr>
<td>STZ + BLE 150</td>
<td>A 0.39 ± 0.01</td>
<td>0.47 ± 0.02</td>
<td>0.52 ± 0.01</td>
<td>0.21 ± 0.004</td>
<td>3.72 ± 0.07</td>
</tr>
<tr>
<td></td>
<td>B 0.73 ± 0.12</td>
<td>0.02 ± 0.002</td>
<td>0.47 ± 0.02</td>
<td>0.34 ± 0.02</td>
<td>1.04 ± 0.02</td>
</tr>
<tr>
<td>STZ + BLE 250</td>
<td>A 0.88 ± 0.08</td>
<td>0.47 ± 0.01</td>
<td>0.41 ± 0.22</td>
<td>0.40 ± 0.004</td>
<td>5.38 ± 0.32</td>
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<tr>
<td></td>
<td>B 1.16 ± 0.09</td>
<td>0.05 ± 0.003</td>
<td>0.07 ± 0.004</td>
<td>0.34 ± 0.01</td>
<td>1.15 ± 0.01</td>
</tr>
<tr>
<td>STZ + BLE 500</td>
<td>A 1.06 ± 0.04</td>
<td>0.73 ± 0.01</td>
<td>0.70 ± 0.01</td>
<td>0.58 ± 0.015</td>
<td>4.45 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>B 1.32 ± 0.02</td>
<td>0.06 ± 0.003</td>
<td>0.98 ± 0.05</td>
<td>0.21 ± 0.01</td>
<td>1.15 ± 0.04</td>
</tr>
<tr>
<td>F - value</td>
<td>A 61.53</td>
<td>160.26</td>
<td>9.8</td>
<td>436.91</td>
<td>66.16</td>
</tr>
<tr>
<td></td>
<td>B 37.72</td>
<td>53.34</td>
<td>235.44</td>
<td>219.78</td>
<td>4.83</td>
</tr>
</tbody>
</table>

P values: <0.001, <0.01, <0.05 compared to control group and <0.01, <0.05 compared to STZ treated group
STZ. Studies suggest that the tissue content having secondarily, the effects of the diabetogenic agent, which upon auto-oxidation generate free radicals and could be due to augmented blood glucose levels, increase in oxygen free radicals in diabetes primarily in the present study, elevated LPO in the liver and development of diabetes and its complications of hyperglycemia, oxidative stress is increased in stimulating glucose transport in cell model. The presence of three active ellagitannins in banaba extract, lagerstroemin, flosin B and reginin A, in optimum concentrations are found to be activators of hexose uptake in rat adipocytes, similar to that employed by insulin and could act intracellularly or bind and activate the insulin receptor (IR) extracellularly. Studies on corosolic acid based banaba extract product glucosol; support the present result, which revealed dose dependent effect on glucose transport activation, which is likely to be the effect of corosolic acid. In contrast, studies by Liu et al. suggest that the glucose transport activity was caused by the tannin component of the extract, and not corosolic acid and while pure corosolic acid tested was found ineffective in stimulating glucose transport in cell model.

Growing evidence indicates that as a result of hyperglycemia, oxidative stress is increased in diabetes due to overproduction of reactive oxygen species (ROS) and decreased efficiency of antioxidant defences through enzymatic and non-enzymatic components. Glucose auto-oxidize, in the presence of transition metal ions, to generate oxygen free radicals make the membrane vulnerable to oxidative damage. Further, when exposed to free radicals, oxidation of lipids, proteins and other macromolecules such as DNA occurs during the development of diabetes and its complications. In the present study, elevated LPO in the liver and brain samples of STZ diabetic mice indicated an increase in oxygen free radicals in diabetes primarily could be due to augmented blood glucose levels, which upon auto-oxidation generate free radicals and secondarily, the effects of the diabetogenic agent, STZ. Studies suggest that the tissue content having relatively high concentration of easily peroxidizable fatty acids and increased activities of enzymes like fatty acyl coenzyme, coenzyme A oxidase due to hypoinsulinaemia initiate the beta-oxidation of fatty acids resulting in lipid peroxidation. Increased LPO and its products (lipid radicals and lipid peroxide) are predominantly associated with atherosclerosis and brain damage. Administration of BLE, at a dose 150 mg/kg body weight reduced the LPO markers to near normal in both liver and brain tissues showing a dose independent antiperoxidative effect of the BLE, supporting its in vitro antioxidant activity.

The altered balance of the antioxidant enzymes with an increase in the SOD and GST activities, in STZ diabetic condition, may be due to increased production of O$_2^-$ and H$_2$O$_2$ by the auto-oxidation of the glucose and non-enzymatic glycation. Similarly, decrease in CAT level depicts the inactivation of the enzyme by O$_2^-$ anions, as observed earlier. These enzymes are suggested to play an important role in maintaining physiological levels of oxygen and H$_2$O$_2$ by hastening the dismutation of oxygen radicals and by eliminating the organic peroxides and hydroperoxides generated from inadvertent exposure to STZ. GST is a family of enzymes involved in the binding, transport and detoxification and cellular defence. Increased free radicals in STZ diabetic liver and brain may have enforced GST detoxification thereby increasing its activity to a significant level. The beneficial role of BLE, especially a dose of 150 mg/kg body weight, helps in ameliorating the oxygen free radicals, as seen in the in vitro studies, may have brought the enzyme levels nearly to normal and may help to control free radical generation during diabetes.

GSH plays an important role in the detoxification and metabolism as a cofactor or as a substrate for some enzymes and as an antioxidant agent protecting tissue from oxidative stress. In the present study, the decreased GSH levels in diabetic liver and brain tissues, may be due to the enhanced GST activity, supporting the earlier studies. Elevated GSH content in liver and brain samples on BLE administration to diabetic animals may help in offering protection to cellular proteins against oxidation through glutathione redox cycle and further also helps in detoxifying reactive oxygen species generated during diabetic state.

In conclusion, *L. speciosa* has the potential to inhibit LPO and effectively intercept/neutralize ROS such as super oxide radical, H$_2$O$_2$ and NO based...
free radicals. Thus the aqueous leaf extract of *L. speciosa* (banaba) especially at a dose of 150 mg/kg body weight duly reduced STZ generated reactive intermediates and radical species helping to keep normal levels of enzymatic and non-enzymatic antioxidants. It possesses optimal antioxidative, antiperoxidative and hypoglycemic property, which could exert an ameliorating action in the pathological alterations.

Acknowledgements

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