Antioxidant, Antihyperlipidaemic and Antidiabetic Activity of Eugenia floccosa Bedd Leaves in Alloxan Induced Diabetic Rats

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ABSTRACT
The ethanol extract of Eugenia floccosa Bedd (Family: Myrtaceae) leaf was investigated for its antioxidant, antihyperlipidaemic and antidiabetic effect in Wistar Albino rats. Diabetes was induced in Albino rats by administration of alloxan monohydrate (150mg/kg, i.p.). The ethanol extracts of E. floccosa at a dose of 150 and 300mg/kg of body weight were administered at single dose per day to diabetes induced rats for a period of 14 days. The effect of ethanol extract of E. floccosa leaf extract on blood glucose, plasma insulin, creatinine, glycosylated haemoglobin, urea serum lipid profile [total cholesterol (TC), triglycerides (TG), low density lipoprotein – cholesterol (LDL-C), very low density lipoprotein – cholesterol (VLDL-C), high density lipoprotein – cholesterol (HDL-C) and phospholipid (PL)], serum protein, albumin, globulin, serum enzymes [serum glutamate pyruvate transaminases (SGPT) and serum glutamate oxaloacetate transaminases (SGOT) and serum glutamate oxaloacetate transaminases (SGOT)], and alkaline phosphatase (ALP)], lipoprotein peroxidation (LPO), antioxidant enzymes (catalase (CAT), superoxide dismutase (SOD), reduced glutathione (GSH) and glutathione peroxidase (GPx)) were measured in the diabetic rats. The ethanol extract of Eugenia floccosa leaf elicited significant reductions of blood glucose (P<0.05), lipid parameters except HDL-C, serum enzymes and significantly increased HDL-C and antioxidant enzymes. The extracts also caused significant increase in plasma insulin (P<0.05) in the diabetic rats. From the above results, it is concluded that ethanol extract of Eugenia floccosa possesses significant antidiabetic, antihyperlipidaemic and antioxidant effects in alloxan induced diabetic rats.

INTRODUCTION
Diabetes mellitus is a major and growing public health problem throughout the world, with an estimated worldwide prevalence of 150 million people in 2000, which is expected to increase to 320 million by 2025 [1]. Besides hyperglycemia, several other factors including dyslipidemia or hyperlipidemia are involved in the development of micro and macro vascular complications of diabetes which are the major causes of morbidity and death [2]. Although numerous oral hypoglycemic drugs exist alongside insulin, still there is no promising therapy to cure diabetes [3]. India has a rich emporium of various potent herbs and herbal components for treating various diseases including diabetes. In recent years, numerous traditional medicinal plants were tested for their antidiabetic potential in the experimental animals [4,5,6].

Eugenia floccosa Bedd is one of the medicinally important plants belongs to Myrtaceae family. The leaf paste of E. floccosa is given to treat rheumatic pain by Kanikkar tribe of Agasthiarmalai Biosphere Reserve, Tamil Nadu. The ethanol extract of E. floccosa has been reported for its anti-tumour activity [7]. The current investigation is an attempt to study the antidiabetic, antihyperlipidaemic and antioxidant activities of ethanol extract of E. floccosa leaf in alloxan induced diabetic rats.

MATERIALS AND METHODS
Plant Material
The leaves of Eugenia floccosa Bedd were freshly collected from the well grown healthy plants inhabiting the natural forests of Koottiyar, Agasthiarmalai Biosphere Reserve, Western Ghats, Tamilnadu. The plant were identified and authenticated in Botanical Survey of India, Southern Circle, Coimbatore, Tamilnadu, India. A voucher specimen was deposited in Ethnopharmacology Unit, Research Department of Botany, V.O.Chidambaram College, Tuticorin, Tamilnadu.

Preparation of plant extract for phytochemical screening and antidiabetic studies
The E. floccosa leaves were shade dried at room temperature and the dried leaves were powdered in a Wiley mill. Hundred grams of powdered E. floccosa leaves was packed in a Soxhlet apparatus and extracted with ethanol. The extract were subjected to qualitative test for the identification of various phytochemical constituents as per the standard procedures [8,9,10]. The ethanol extracts were concentrated in a rotary evaporator. The concentrated ethanol extract were used for antidiabetic studies.

Animals
Normal healthy male Wistar albino rats (180–240g) were housed under standard environmental conditions at temperature (25±2º C) and light and dark (12: 12 h). Rats were fed with standard pellet diet (Goldmohur brand, MS Hindustan lever Ltd., Mumbai, India) and water ad libitum.

Acute Toxicity Study
Acute oral toxicity study was performed as per OECD – 423 guidelines (acute toxic class method), albino rats (n=6) of either sex selected by random sampling were used for acute toxicity study [11]. The animals were kept fasting for overnight and provided only with water, after which the extracts were administered orally at 5mg/kg body weight by gastric intubations and observed for 14 days. If mortality was observed in two out of three animals, then the dose administered was assigned as toxic dose. If mortality was observed in one animal, then the same dose was repeated again to confirm the toxic dose. If mortality was not observed, the procedure was repeated for higher doses such as 50,100, and 2000 mg/kg body weight.

Induction of Experimental Diabetes
Rats were induced diabetes by the administration of simple intraperitoneal dose of alloxan monohydrate (150 mg/kg) [12]. Two days after alloxan injection, rats...
screened for diabetes having glycosuria and hypoglycemia with blood glucose level of 200-260 mg/100 ml were taken for the study. All animals were allowed free access to water and pellet diet and maintained at room temperature in plastic cages.

Experimental Design
In the present investigation, a total of 30 rats (24 diabetic surviving rats and 6 normal rats) were taken and divided into five groups of 6 rats each.
  - Group I: Normal untreated rats
  - Group II: Diabetic control rats
  - Group III: Diabetic rats given ethanol extract of *E. floccosa* leaf (150mg/kg body weight)
  - Group IV: Diabetic rats given ethanol extract of *E. floccosa* leaf (300mg/kg body weight)
  - Group V: Diabetic rats given standard drug glibenclamide (600mg/kg body weight).

Biochemical analysis
The animals were sacrificed at the end of experimental period of 14 days by decapitation. Blood was collected, sera separated by centrifugation at 3000g for 10 minutes. Serum glucose was measured by the O-toluidine method [13]. Insulin level was assayed by Enzyme Linked Immunosorubant Assay (ELISA) kit [14]. Urea estimation was carried out by the method of Varley [15]; serum creatinine was estimated by the method of Owen et al [16]. Glycosylated haemoglobin (HBA1C) estimation was carried out by a modified colorimetric method of Karunanayake and Chandrasekharan [17]. Serum total cholesterol (TC) [18], total triglycerides (TG) [19], low density lipoprotein cholesterol (LDL-C), very low density lipoprotein cholesterol (VLDL-C) [20], high density lipoprotein cholesterol (HDL-C) [21] and phospholipids [22] were analyzed. Serum protein [23] and serum albumins were determined by quantitative colorimetrically method by using bromocresol green. The total protein minus the albumin gives the globulin, serum glutamate pyruvate transaminase (SGPT) and serum glutamate oxaloacetate transaminase (SGOT) were measured spectrophotometrically by utilizing the method of Reitman and Frankel [24]. Serum alkaline phosphatase (ALP) was measured by the method of King and Armstrong [25]. Catalase (CAT) [26], superoxide dismutase (SOD) [27], lipid peroxidation (LPO) [28], reduced glutathione (GSH) [29] and glutathione peroxidase (GPx) [30] were analyzed in the normal, diabetic induced and drug treated rats.

STATISTICAL ANALYSIS
The data were analyzed using student’s t-test statistical methods. For the statistical tests a p values of less than 0.01 and 0.05 was taken as significant.

Table 1: Effect of ethanol extracts of *Eugenia floccosa* leaves on the serum insulin, glucose, urea, creatinine and HBA1C level of normal, diabetic induced and drug treated adult albino rats.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Insulin (MIu/ml)</th>
<th>Glucose (mg/dl)</th>
<th>Urea (mg/dl)</th>
<th>Creatinine (mg/dl)</th>
<th>Glycosylated Hb (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>24.50±1.4</td>
<td>69.50±1.2</td>
<td>11.54±1.9</td>
<td>0.61±0.8</td>
<td>3.6±0.05</td>
</tr>
<tr>
<td>Group II</td>
<td>0.50±0.6**</td>
<td>201.00±11.2*</td>
<td>39.5±5.6*a</td>
<td>1.72±0.7</td>
<td>12.5±1.1*</td>
</tr>
<tr>
<td>Group III</td>
<td>12.60±1.1*a</td>
<td>105.50±6.3*a</td>
<td>26.21±4.2</td>
<td>1.23±0.1</td>
<td>9.36±1.7*</td>
</tr>
<tr>
<td>Group IV</td>
<td>17.80±1.3** a</td>
<td>76.50±57aa</td>
<td>9.34±5.1aa</td>
<td>0.89±0.4</td>
<td>7.07±1.03a</td>
</tr>
<tr>
<td>Group V</td>
<td>22.50±0.8**</td>
<td>91.50±6.9aa</td>
<td>12.74±1.9a</td>
<td>0.81±0.5</td>
<td>4.91±0.7</td>
</tr>
</tbody>
</table>

Each value is SEM of 6 animals. Comparison made between normal control to diabetic control and drug treated groups * p < 0.05; **p<0.01 and comparison made between diabetic control to drug treated groups a p<0.05; aa p<0.01
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When compared to control (Group I) and glibenclamide treated rats (Group V). On administration of ethanol extract of *E. floccosa* to the diabetic rats, the levels of protein, albumin and globulin were found to be restored in normal. These results were in accordance with the effects of *Wattakaka volubilis* [6], *Senna auriculata* [38] and *Pterocarpus marsupium* [36] in diabetic rats.

Table 2: Effect of ethanol extract of *Eugenia floccosa* leaves on the serum protein, albumin, globulin, SGOT, SGPT and ALP level of normal, diabetic induced and drug treated adult albino rats.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Protein (g/dl)</th>
<th>Albumin (g/dl)</th>
<th>Globulin(g/dl)</th>
<th>SGPT (u/l)</th>
<th>SGOT (u/l)</th>
<th>ALP (u/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>8.94±0.11</td>
<td>4.14±0.64</td>
<td>4.87±0.11</td>
<td>19.2±3.2</td>
<td>21.4±3.3</td>
<td>164.55±5.4</td>
</tr>
<tr>
<td>Group II</td>
<td>6.51±0.71*</td>
<td>3.91±0.35</td>
<td>2.65±0.08*</td>
<td>35.4±6.2*</td>
<td>35.3±5.9*</td>
<td>196.65±3.4</td>
</tr>
<tr>
<td>Group III</td>
<td>7.14±0.92</td>
<td>4.04±0.87</td>
<td>3.17±0.10</td>
<td>29.1±3.2</td>
<td>27.4±4.1</td>
<td>113.42±4.4</td>
</tr>
<tr>
<td>Group IV</td>
<td>8.14±0.89</td>
<td>4.99±0.43</td>
<td>4.15±0.06</td>
<td>20.6±3.4</td>
<td>24.5±2.8</td>
<td>151.43±5.6</td>
</tr>
<tr>
<td>Group V</td>
<td>7.94±0.30*</td>
<td>4.11±0.32</td>
<td>3.83±0.04</td>
<td>16.5±4.8*</td>
<td>28.4±2.2</td>
<td>147.33±5.7</td>
</tr>
</tbody>
</table>

Each value is SEM of 6 animals. Comparison made between normal control to diabetic control and drug treated groups * p < 0.05

Table 3: Effect of ethanol extract of *Eugenia floccosa* leaves on the serum lipid profile of normal, diabetic induced and drug treated adult albino rats.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>TC (mg/dl)</th>
<th>TG(mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>VLDL (mg/dl)</th>
<th>HDL (mg/dl)</th>
<th>PL (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>104.51±3.56</td>
<td>88.51±2.84</td>
<td>52.59±2.05</td>
<td>17.70±1.22</td>
<td>34.21±0.52</td>
<td>161.01±13.91</td>
</tr>
<tr>
<td>Group II</td>
<td>184.53±8.42*</td>
<td>206.55±8.41***</td>
<td>116.69±6.25*</td>
<td>41.31±3.45*</td>
<td>26.53±3.84*</td>
<td>232.23±12.17*</td>
</tr>
<tr>
<td>Group III</td>
<td>121.51±6.31</td>
<td>151.56±3.99**</td>
<td>60.38±2.33a</td>
<td>30.31±1.23*</td>
<td>30.81±1.91</td>
<td>176.14±16.39</td>
</tr>
<tr>
<td>Group IV</td>
<td>116.64±5.21</td>
<td>109.32±4.14a</td>
<td>55.44±5.55a</td>
<td>21.86±2.33**a</td>
<td>32.33±2.11</td>
<td>171.80±10.96</td>
</tr>
<tr>
<td>Group V</td>
<td>113.56±4.52</td>
<td>109.53±7.33a</td>
<td>62.34±4.12</td>
<td>21.90±1.98*</td>
<td>29.31±4.23</td>
<td>169.06±13.84aa</td>
</tr>
</tbody>
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diabetic rats (Group II) when compared to control (Group I) and glibenclamide treated rats (Group V). On administration of ethanol extract of *E. floccosa* to the diabetic rats, the levels of protein, albumin and globulin were found to be restored in normal. These results were in accordance with the effects of *Wattakaka volubilis* [6], *Senna auriculata* [38] and *Pterocarpus marsupium* [36] in diabetic rats.

Table 2 summarized the effect of alloxan on the activity of the hepatic marker enzymes in serum. In the present study, the levels of SGPT and SGOT in alloxan induced diabetic rats were elevated. It may be due to leaking out of enzymes from the tissues and migrating into the circulation by the adverse effect of alloxan [34]. Aspartate amino transaminases and Alanine transaminase were used as markers to assess the extent of liver damage in streptozotocin induced diabetic rats [39]. In this study, the ethanol extract of *E. floccosa* regulated the activity of SGPT and SGOT in liver of rats intoxicated with alloxan. The effect of glibenclamide on the recovery of hepatic enzyme activity in serum was very similar to that of earlier study [40].

The restorations of SGPT and SGOT to their respective normal levels after treatment with both glibenclamide and ethanol extract of *E. floccosa* further strengthen the antidiabetic effect of this extract. Moreover, SGPT and SGOT levels also act as indicators of liver function and restoration of normal levels of these parameters indicate normal functioning of liver. Since the alloxan can also affect the liver by free radical mechanism.

In addition to the assessment of SGPT and SGOT levels during diabetes the measurement of enzymatic activities of phosphatases such as acid phosphatase (ACP) and alkaline phosphatase (ALP) is of clinical and toxicological importance as changes in their activities are indicative of tissue damage by toxicants. In the present study, serum ALP increased in alloxan induced diabetic rats (Table 2). Elevated level of this enzyme in diabetes may be due to extensive damage to liver in the experimental animal by alloxan. Treatment with ethanol extract of *E. floccosa* in alloxan induced diabetic rats produces a decline in ALP level.

The levels of serum lipid profile, total cholesterol (TC), triglycerides (TG), LDL-C, VLDL-C and HDL-C in control, diabetic induced and drug treated rats were presented in Table 3. Alloxan induced rats showed significant increase in serum lipid profiles except HDL-C when compared with normal rats. The glibenclamide (Group V) and ethanol extract of *E. floccosa* (Group III and IV) treated rats...
since it inhibits the activity of hormone sensitive lipase in adipose tissue and sup-

an important role in the lipid metabolism. Insulin is a potent inhibitor of lipolysis,

Scoparia dulcis

Cassia auriculata

[49,50]

flower [35] floxocosa

nol extract of E. floccosa

oxidative damage due to the antiperoxidative eff ect of ingredients present in etha-

μ

μ

is defined as the<br>

<0.05. x-One unit of SOD is defined as the enzyme concentration which gives 50% inhibition of NBT reduction in one minute. y-One unit of CAT is defined as the µ mole of hydrogen peroxide consumed per minute.

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showed a significant decrease in the content of lipid profile when compared with diabetic induced rats. Similarly HDL-C level decreased in alloxan induced diabetic rats when compared to normal rats. On administration of ethanol extract of E. floccosa and glibenclamide to the diabetic rats, HDL-C level was found to be restored to normal. The level of serum lipid profiles are usually raised in diabetic rats in the present study and such elevation represents risk factor for coronary heart diseases [41]. Lowering of the serum lipid level through dietary or drug therapy seems to be associated with a decrease in the risk of vascular disease [42]. During diabetes, enhanced activity of the enzyme, increased lipolysis and releases more fatty ac-

ids into the circulation [43]. The increased fatty acid concentration also increases the β-oxidation of fatty acids, producing more acetyl Co-A and cholesterol during diabetes. In normal condition, insulin increases receptor-mediator removal of LDL-cholesterol and decreased activity of insulin, during diabetes causes hyper-

cholesterolemia. Hypercholesterolemia and hypertriglyceridemia have been reported to occur in diabetic rats [41]. The increased concentration of free fatty acid may be due to lipid break-down and this may cause increased generation of NADPH-

dependent microsomal lipid peroxidation. Phospholipids were increased in al-

loxan induced diabetic rats. Phospholipids are present in cell membrane and make up vast majority of the surface lipoprotein forming a lipid bilayer that acts as an interface with both polar plasma environment and non-polar lipoprotein of li-

poprotein core [44]. Increased phospholipids levels in tissues were reported by Venkateswaran et al. [45]; Pari and Satheesh, [46] in streptozotocin diabetic rats. Administration of ethanolic extract of E. floccosa leaf and glibenclamide decreased the levels of phospholipids.

The results (Table 4) showed increased lipid peroxidation (LPO) of alloxan induced diabetic rats. Earlier studies have reported that there was an increased li-

pid peroxidation in liver, kidney and brain of diabetic rats [47,48]. This may be be-

cause the tissues contain relatively high concentration of early peroxidizable fatty acids. In the present study, an increase in the levels of LPO was found and these changes were reversed in diabetic rats [41]. The increased concentration of free fatty acid may be due to lipid break-down and this may cause increased generation of NADPH-

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Table 4: Effect of ethanol extracts of Eugenia floccosa leaves on the LPO, SOD, CAT, GPX and GSH enzymes in the plasma of normal, diabetic induced, and drug treated adult albino rats.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>LPO (mmole/dl)</th>
<th>SOD (unit x/mg protein)</th>
<th>CAT (unit y/mg protein)</th>
<th>GPX(unit z/mg protein)</th>
<th>GSH (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>0.83± 0.34</td>
<td>6.93± 0.74</td>
<td>0.49± 0.31</td>
<td>7.36± 0.81</td>
<td>91.54± 3.05</td>
</tr>
<tr>
<td>Group II</td>
<td>3.11± 0.98</td>
<td>14.56± 0.54**</td>
<td>2.96± 0.88*</td>
<td>13.23± 0.92**</td>
<td>27.31± 2.86**</td>
</tr>
<tr>
<td>Group III</td>
<td>1.15 ±0.88</td>
<td>9.39 ±0.65</td>
<td>0.57± 0.77a</td>
<td>8.59 ±1.65</td>
<td>42.16±1.54a</td>
</tr>
<tr>
<td>Group IV</td>
<td>0.74 ± 0.41a</td>
<td>4.91 ± 0.33a</td>
<td>0.45 ± 3.9a</td>
<td>5.81 ± 1.64a</td>
<td>89.62±3.85</td>
</tr>
<tr>
<td>Group V</td>
<td>0.89± 0.14a</td>
<td>7.99± 0.33</td>
<td>0.84± 0.14</td>
<td>8.03± 0.65a</td>
<td>87.4±5.18a</td>
</tr>
</tbody>
</table>

Each value is SMM of 6 animals. Comparison made between normal control to diabetic control and drug treated groups. * p < 0.05; **p<0.01 and comparison made between diabetic control to drug treated groups a p<0.05. x-One unit of SOD is defined as the enzyme concentration which gives 50% inhibition of NBT reduction in one minute. y-One unit of CAT is defined as the µ mole of hydrogen peroxide consumed per minute.

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