Beneficial effects of *Lagenaria siceraria* (Mol.) Standley fruit epicarp in animal models

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*Lagenaria siceraria* (Mol.) Standley fruit (bottle gourd), a commonly used vegetable in India is described as cardiotonic and as a general tonic in Ayurveda. Keeping in view the presence of free radical scavenging activity in *L. siceraria* and involvement of free radicals in the development of various disorders, present studies were designed to evaluate the ethanolic extract of *L. siceraria* fruit against the disorders where free radicals play a major role in pathogenesis. The extract was found effective as hepatoprotective, antioxidant, antihyperglycemic, immunomodulatory, antihyperlipidemic and cardiotonic agent. The results showed that the radical scavenging capacity of *L. siceraria* fruit may be responsible for various biological activities studied.

Keywords: Antioxidant, Antihyperlipidemic, Antihyperglycemic, Cardiotonic, Hepatoprotective, Immunomodulatory, *Lagenaria siceraria*

*Lagenaria siceraria* (Mol.) Standley fruit (Syn. *L. vulgaris* Ser., *Cucurbita lagenaria* Linn., *L. leucantha* Rusbey, family Cucurbitaceae) commonly known as bottle gourd is used as a vegetable in India. The fruit is traditionally used as a cardiotonic, aphrodisiac and general tonic¹, liver tonic and against liver disorders and pain², anti-inflammatory, expectorant and diuretic agent³. Further, antihapatotoxic activity of fruit pulp⁴, analgesic and anti-inflammatory activity of fruit juice⁵ and hypolipidimic activity of the fruit have also been evaluated⁶. Recently, the antioxidant activity of ethanolic extract of epicarp and fresh juice of *L. siceraria* fruit have been reported⁷.

Lagenin, a ribosome inactivating protein (RIP) isolated from the seeds of *L. siceraria* possesses immunoprotective, antitumor, antiHIV and antiproliferative properties⁸. Phytochemical screening of the fruit revealed the presence of fucosterol and compesterols⁹ flavonoids, cucurbitacins, saponins and polyphenolics⁵, triterpenoids and C-flavone glycosides⁹-¹² and ellagitannins⁷.

Cardiovascular disorder is claimed to be relieved following regular intake of bottle gourd juice for about 4-6 months¹³. ‘Hriday Mitra Mandal’—a registered society, devoted to relief for cardiac patients, forcefully advocates daily use of the juice in order to prevent cardiovascular disorder in young as well as elderly persons¹³. The existence of interrelationship between production of free radicals and induction of cardiovascular disorders is well documented¹⁴-¹⁶. Similarly, the association of exogenous and endogenous generation of reactive oxygen species (ROS) with the pathogenesis of diseases like cancer¹⁷, diabetes¹⁸, arthritis¹⁹ and aging²⁰ is also established. The present study has been undertaken to evaluate immunomodulatory hepatoprotective, antihyperglycemic, antihyperlipidemic and cardiotonic properties of ethanolic extract of *L. siceraria* (EELS).

**Materials and Methods**

Sprague-Dawley rats (150-200 g) of either sex and male Swiss albino mice (20-27 g) obtained from National Institute of Nutrition, Hyderabad were used. While rats were used for assessment of hepatoprotective, *in vivo* antioxidant, antihyperglycemic and antihyperlipidimic activities, albino mice were used for determination of immunomodulatory activity.

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Male frogs (*Rana tigrina*, 20-30g) were used for evaluation of cardiotonic activity.

The fruits of *L. siceraria* (Mol.) Standley, purchased locally were authenticated from Department of Botany, Rashtrasant Tukadoji Maharaj Nagpur University, Nagpur. The fruit was separated into epicarp, mesocarp and pulp containing seeds. These were shade dried, coarsely powdered and soxhlet extracted successively with petroleum ether, benzene, chloroform and ethanol. The extracts were concentrated in vacuum to a syrupy consistency and evaporated to dryness at 45°C to a constant weight. Since preliminary *in vitro* experiments with 1-1 diphenyl-picryl-2 hydrazyl (DPPH) assay revealed that ethanolic extract of epicarp exhibits maximum free radicals scavenging activity 

preliminary experiments.

**Acute toxicity studies** were performed as per OECD-423 guide lines on randomly selected rats of either sex (n=6). EELS (5 mg/kg) was administered orally after overnight fasting. Since mortality was not observed, the procedure was repeated with higher doses viz. 50, 300 and 2000 mg/kg body weight.

**Immunomodulatory activity—Preparation of antigen:** The blood withdrawn from the external jugular vein of the sheep (Sheep Farm, Nagpur Veterinary College, Nagpur) was mixed (1:1) with Alsever’s solution, centrifuged at 2500 rpm for 20 min and washed twice with sterile phosphate buffer saline (PBS). A 2% sheep red blood cells (SRBC) suspension in PBS was prepared and stored at 4°C in refrigerator and used as required. A known amount (0.5×10^9 cells/ml/100g body weight) was injected (ip) to the mice as the antigenic challenge.

The mice were divided into 6 groups of 8 animals each. Group I served as control and received normal saline only. The mice of group II-VI were injected with SRBCs on 14 and 21 day (0.5×10^9 cells/ml/100g; ip). Gr II animals received only pyrogallol (50 mg/kg, ip) five days before the first antigen challenge. (Mice in Gr III were administered orally with vitamin E (50 mg/kg) and vitamin C (50 mg/kg) once daily. Gr IV, V and VI were administered with 4.6, 9.2 and 23 mg/kg of EELS respectively thrice daily, orally. Immunomodulatory activity was evaluated by the assessment of (a) humoral immune response (HIR) to SRBC by hemagglutination reaction (HR) (b) cell mediated immune response (CMIR) by delayed type hypersensitivity reaction (DTH) (c) neutrophil adhesion to nylon fibres 

Hepatoprotective and antioxidant activity—Hepatoprotective activity was assessed by examining the influence of the EELS (in doses 100 and 200 mg/kg) on hepatotoxicity induced by administration of CCl₄ (30% in liquid paraffin) 

Blood was collected from retro-orbital plexus under light ether anaesthesia on 15th day for analysis of levels of serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), alkaline phosphatase (ALP), acid phosphatase (ACP) and bilirubin. SGOT, SGPT and ALP kits were obtained from Erba Diagnostics, Mannheim, Germany, Merck Ltd, Mumbai and SPAN Diagnostic Ltd, Surat, respectively and methods prescribed by manufacturer of kits were followed. Histopathology of the hepatic cells was carried by using hematoxylin-eosin dye to stain the cells of rats sacrificed under light ether anaesthesia. Protein content, lipid peroxidation (LPO), superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) were determined by homogenizing the remaining hepatic tissue in 0.1 M PBS (pH 7.4). Doses were based on preliminary experiments.

**Antihyperglycemic activity**—Hyperglycemia was induced in rats by alloxan monohydrate (150 mg/kg, ip single dose). Rats in which hyperglycemia (blood glucose level above 260 mg/dl) was induced after 48 hr of alloxan administration were divided into 5 groups of 6 rats each. Gr I served as the normal control and Gr II as a hyperglycemic control to which saline solution was administered. Animals in Gr III and IV were hyperglycemic and treated with EELS (100 and 200 mg/kg, po) for 14 days. Gr V was treated with glibenclamide (5 mg/kg po) for 14 days. Plasma levels of glucose were analysed on 0, 7 and 14th day of treatment, serum lipid levels were determined on 14th day which included total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), triglycerides (TGL), low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C).

**Antihyperlipidimic activity**—Hyperlipidemia was induced in rats by administration of Triton WR 1339 (iso-octyl polyoxyethylene phenol) in phosphate buffer (pH 7.2, 0.05 M). The rats were divided into 5 groups of 6 animal each. Gr I served as a normal control and rats of group II to V were administered...
with Triton WR 1399 (350 mg/kg/ip). Gr II served as hyperlipidemic control. Animals of Gr III and IV were administered with EELS (100 and 200 mg/kg, po respectively). The fifth group received simvastatin (0.57 mg/kg, po). The blood samples were withdrawn at 0, 24 and 48 hr after Triton injection, blood serum was separated by centrifugation and analysed for TC and TGL.

Action on frog heart—The action of EELS on frog heart was recorded by observing chronotropic and inotropic effect on heart preparation by using conventional method38. The frog heart was exposed and the venous cannula was inserted in the inferior vena cava. The universal lever was attached to heart by a thread and heart beats were measured on rotating drum. When normal rhythm of heart was attained, 1 µg CaCl$_2$ was injected in the perfusion tube very close to venous canula and effect was observed. This was followed by injecting two doses of EELS viz 0.1 and 1 µg separately and observing the effect. Same procedure was repeated using a modified ringer solution when concentration of Ca$^{2+}$ was ¼ to that of original concentration used.

Statistical analysis—The results are expressed as mean±SE and the difference between the group were analysed by ANOVA followed by Dunnets test ($P<0.05$) and Student’s $t$ test.

Results and Discussion

Daily intake of homogenized bottle gourd (about 200 to 300 g) in the form of a juice serves as a prophylactic against cardiac disorders. Also, patients claim that their heart block as well as lipid profile came to normal following taking the bottle gourd juice regularly13. Perusal of their case papers amply support their claim. There is however no scientific data available to support the usefulness of bottle gourd against cardiac disorders. The results of the present work seem to support this practice.

Association between oxidative stress, generation of free radicals and cardiovascular disorders is now well recognized14-16. It is also generally believed that scavenging the excessive free radicals could prevent occurrence of related disorders. EELS appears to exert significant free radical scavenging, antihyperlipidimic, immunomodulatory, hepatoprotective and antioxidant effect. These properties of EELS seem responsible for prevention of CVD symptoms and also alleviating the pathophysiological symptoms associated with CVD such as alteration in lipid profile and coronary block. Interestingly EELS seems to provide protection against stress hyperglycemia also.

Acute toxicity studies—No mortality occurred in the group of rats receiving upto 2000 mg/kg po of EELS. L. siceraria is being used since ancient time as a vegetable, its LD$_{50}$ was not determined. Fresh juice obtained from 200-300 g L. siceraria fruit is recommended in ‘Dudhi therapy’13. Therefore the dose was selected on the basis of EELS obtained from epicarp of 200 g of fruit and calculated for adult human as mg/kg body weight.

Immunomodulatory activity—Effects of EELS on humoral immune response (HIR), cellular immune response (CIR) and percent neutrophil adhesion (PNA) in mice in the presence of chemical stressor i.e. pyrogallol are presented in Table 1. EELS prevented to a significant extent the reduction of HIR, CIR and PNA induced by pyrogallol. In this respect 23 mg/kg of

<table>
<thead>
<tr>
<th>Group</th>
<th>Doses</th>
<th>Humoral immune response (HIR)</th>
<th>Cellular immune response (% increase in paw volume)</th>
<th>Neutrophil adhesion (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Primary</td>
<td>Secondary</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>Control</td>
<td>7.0 ± .92</td>
<td>10.25 ± 0.46</td>
<td>27.51 ± 1.92</td>
</tr>
<tr>
<td>II</td>
<td>Pyrogallol (50 mg/kg)ip</td>
<td>4.12 ± 0.64</td>
<td>7.25 ± 0.46</td>
<td>16.36 ± 1.36</td>
</tr>
<tr>
<td>III</td>
<td>Pyrogallol (50 mg/kg) + Vitamin E (50 mg/kg) + Vitamin C (50 mg/kg) once daily</td>
<td>7.37 ± 0.51**</td>
<td>10.37 ± 0.51**</td>
<td>27.92 ± 2.13**</td>
</tr>
<tr>
<td>IV</td>
<td>Pyrogallol (50 mg/kg) + Extract (4.6 mg/kg) three times daily</td>
<td>5.25 ± 0.46**</td>
<td>7.75 ± 0.46</td>
<td>17.67 ± 0.98</td>
</tr>
<tr>
<td>V</td>
<td>Pyrogallol (50 mg/kg) + Extract (9.2 mg/kg) three times daily</td>
<td>6.5 ±0.53**</td>
<td>8.37 ± 0.51**</td>
<td>21.66 ± 0.96**</td>
</tr>
<tr>
<td>VI</td>
<td>Pyrogallol (50 mg/kg) + Extract (23 mg/kg) three times daily</td>
<td>6.87 ± 0.83**</td>
<td>9.37± 0.51**</td>
<td>26.96 ± 1.49**</td>
</tr>
</tbody>
</table>

ANOVA followed by Dunnett’s test; $P$ values: *<0.05; **<0.01 as compared to group II
EELS seems to be equivalent to 50 mg/kg of vit. C + 50 mg/kg of vit. E.

The cellular components involved in immune system are rich in polyunsaturated fatty acids, which on oxidative attack lead to highly cytotoxic products of peroxidation. Enhancement in prostaglandin level also results in immunomodulation. Enhanced endogenous generation of ROS, because of aging and pathophysiological conditions causes depression of lipoxygenase and cycloxygenase which adversely influence integral membrane function and cell mediated immune reaction. Such conditions may be effectively combated by enhancing cellular defence in the form of radical scavengers or antioxidants.

The increase in humoral immune response exhibited by EELS may be due to increase in antibody responsiveness to SRBC because of enhanced responsiveness of macrophages and β-lymphocytes involved in antibody synthesis. The sensitized lymphocytes, when challenged with SRBC antigen, are converted to lymphoblasts, secrete lymphokines to attract more scavenger cells to site of reaction. The infiltrating cells become more defensive and cause inflammatory reaction. This was evident when an increase in foot paw volume was observed with increased dose of EELS. The oral administration of EELS increased neutrophil adhesion of blood to nylon fibres indicating process of migration of cells to blood vessels.

Hepatoprotective and antioxidant activities—CCl₄ induced hepatotoxicity was evidenced by significant rise in marker enzyme levels in blood viz. SGOT, SGPT, ALP, ACP and bilirubin (Table 2). EELS (100 and 200 mg/kg, po) and silymarin (100 mg/kg, po) could prevent CCl₄ induced rise in the levels of these markers.

The histopathological examination revealed that CCl₄ treatment results in swollen hepatocytes loaded with fatty droplets and pushing of nuclei towards periphery of the hepatocytes (Fig. 1b). In a group which received 100 mg/kg of EELS there was mild degree of degenerative changes, which were perilobular and a mild degree of perivascular infiltration of leucocytes indicating partial hepatoprotective activity (Fig. 1d). EELS 200 mg/kg effectively prevented hepatic damage induced by CCl₄ as there was only mild fatty degeneration and focal necrosis was totally absent (Fig. 1e). The histopathological observations in the group treated with sylimarin (Fig. 1c) were almost comparable to that of the control group (Fig. 1a).

CCl₄ induced hepatotoxic rats showed significant increase in TBARS and about 50% decrease in SOD, CAT and GPx levels. EELS effectively prevented the lipid peroxidation of the liver tissue. The values of TBARS, SOD, CAT and GPx in the group treated with EELS were also close to normal (Table 2).

<table>
<thead>
<tr>
<th>Treatment</th>
<th>SGOT or ASAT (IU / L)</th>
<th>SGPT or ALAT (IU / L)</th>
<th>ALP (IU / L)</th>
<th>ACP (IU / L)</th>
<th>Total bilirubin (mg / 100 ml of blood)</th>
<th>TBARS (n mol Malondialdehyde / mg liver protein)</th>
<th>Superoxide Dismutase (U / mg liver protein)</th>
<th>Catalase (µM of H₂O₂ Decomposed / min / mg liver protein)</th>
<th>Glutathione Peroxidase (n mol of NADPH Decomposed / mg liver protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>97.3 ± 1.18</td>
<td>35.08 ± 186.7</td>
<td>15.92 ± 0.72</td>
<td>10.5 ± 0.064</td>
<td>0.39 ± 0.04</td>
<td>1.29 ± 1.79</td>
<td>75.81 ± 1.94</td>
<td>296.83 ± 0.14</td>
<td>0.992 ± 0.05</td>
</tr>
<tr>
<td>CCl₄ (1 ml/kg ip)</td>
<td>186.7 ± 1.82</td>
<td>136.9 ± 1.82</td>
<td>98.3 ± 1.48</td>
<td>38.6 ± 2.9</td>
<td>0.89 ± 0.76</td>
<td>1.79 ± 0.14</td>
<td>47.84 ± 0.50</td>
<td>179.73 ± 0.61</td>
<td>0.61 ± 0.03</td>
</tr>
<tr>
<td>EELS (100 mg/kg)</td>
<td>132.64 ± 1.82</td>
<td>78.1 ± 1.82</td>
<td>61.3 ± 2.32</td>
<td>23.2 ± 0.76</td>
<td>0.7 ± 0.05</td>
<td>1.43 ± 0.14</td>
<td>65.74 ± 0.50</td>
<td>261.25 ± 0.82</td>
<td>0.82 ± 0.03</td>
</tr>
<tr>
<td>Silymarin (100 mg/kg)</td>
<td>105.3 ± 7.85</td>
<td>49.4 ± 3.7**</td>
<td>34.8 ± 3.7**</td>
<td>16.2 ± 1.2*</td>
<td>0.24 ± 0.04</td>
<td>1.26 ± 0.16</td>
<td>88.34 ± 0.84</td>
<td>268.27 ± 0.95</td>
<td>0.95 ± 0.07</td>
</tr>
</tbody>
</table>

P values: * < 0.01, ** < 0.001

Table 2—Effect of *L. siceraria* extract on CCl₄ induced hepatotoxicity and liver antioxidant defence system in rats. [Values are mean ± SE from 6 animals in each group]
Fig. 1—Photomicrograph of (a) normal liver section of rat; (b) liver section of rat treated with CCl₄; liver section of rat treated with CCl₄ + (c) 100 mg/kg silymarin; (d) 100 mg/kg EELS; (e) 200 mg/kg EELS [a-e: 200X]
CCl₄ causes cell lysis with release of cytoplasmic hepatic enzyme in circulation and is metabolized into trichloromethyl radical (CCl₃ *) which reacts with molecular oxygen to form trichloromethyl peroxy radical (CCl₃O₂ *). CCl₃O₂ * induce lipid peroxidation ultimately gives rise to various cytotoxins like molondialdehyde (MDA) and 4-hydroxynonenenal. In the present studies it appears that EELS detoxifies free radicals generated by CCl₄ administration.

Administration of EELS reduced the level of these enzymes significantly in dose dependent manner. Petroleum ether extract of whole fruit of *L. siceraria* has also been reported to exert antihepatotoxic effect and this was attributed to fucosterol and compesterol isolated from the fraction.

**Antihyperglycemic activity**—Alloxan (150 mg/kg, ip) induced hyperglycemic rats showed significant increase in blood sugar levels which were reduced to a significant extent by EELS (100 and 200 mg/kg, po) on 7th and 14th day of treatment, (Table 3). The percentage reduction by glibenclamide was 57.8 and 64.3% on 7th and 14th days respectively. Alloxan also elevated levels of TC, TG, VLDL-C and LDL-C and reduced that of HDL-C leading to 3-fold rise in TC. This effect was counteracted by simultaneous administration of EELS and may be mediated through its antioxidant activity.

**Antihyperlipidemic activity**—The blood levels of cholesterol and triglycerides were increased by 3.5 and 9.5-folds respectively 24 hr after the administration of Triton. EELS (100 and 200 mg/kg, po) administration effectively prevented this rise in cholesterol and triglycerides. The effect of EELS is almost comparable to that of simvastatin (Table 4).

It is presumed that triton enhances intra- and extramitochondrial synthesis of cholesterol by induction of related enzymes such as HMG-CoA reductase. Antioxidants adversely influence the activity of reductase due to their free radical scavenging capability thus reducing the rate of cholesterol synthesis.

**Table 3—Effect of *L. siceraria* on blood sugar and lipid levels in alloxan induced hyperglycemic rats**

<table>
<thead>
<tr>
<th>Group</th>
<th>Groups and dose (mg/kg)</th>
<th>Blood sugar level</th>
<th>TC</th>
<th>TGL</th>
<th>HDL-C</th>
<th>VLDL-C</th>
<th>LDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control (Normal saline) (2)</td>
<td>90.3 7th day 90.8 14th day</td>
<td>82.53</td>
<td>75.15</td>
<td>25.6</td>
<td>15.03</td>
<td>41.9</td>
</tr>
<tr>
<td>II</td>
<td>Diabetic control (Alloxan) (150)</td>
<td>4.6 7th day 3.6 14th day</td>
<td>7.21</td>
<td>5.03</td>
<td>1.83</td>
<td>1.006</td>
<td>4.37</td>
</tr>
<tr>
<td>III</td>
<td>EELS (100)</td>
<td>18.4 7th day 19.2 14th day</td>
<td>10.49</td>
<td>8.5</td>
<td>6.52</td>
<td>1.7</td>
<td>2.27</td>
</tr>
<tr>
<td>IV</td>
<td>EELS (200)</td>
<td>16.0 7th day 10.3* 14th day</td>
<td>8.62</td>
<td>7.79</td>
<td>2.63*</td>
<td>1.56</td>
<td>4.44**</td>
</tr>
<tr>
<td>V</td>
<td>Glibenclamide (5)</td>
<td>18.9 7th day 9.6** 14th day</td>
<td>8.47</td>
<td>5.83*</td>
<td>3.52*</td>
<td>1.17</td>
<td>3.79**</td>
</tr>
</tbody>
</table>

*P values < 0.01; ** < 0.001*

TC = Total cholesterol  TGL = Triglycerides
HDL-C = High density lipoprotein cholesterol  VLDL-C = Very low density lipoprotein cholesterol
LDL-C = Low density lipoprotein cholesterol
Effect on frog heart—EELS (0.1 mg) caused increase in force of contraction with decrease in rate of contraction (from 66 to 44) in isolated frog heart perfused with normal Ringer solution. The contraction of heart could not be recovered to normalcy when depleted Ringer solution was used (Fig. 2).

Table 4—Effect of *L. siceraria* on triton induced hyperlipidemia in rats

[Values expressed in mg/dl are mean ±SE from 6 animals in each group]

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment and dose (mg/kg)</th>
<th>Cholesterol</th>
<th>Triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 hr</td>
<td>24 hr</td>
<td>48 hr</td>
</tr>
<tr>
<td>I</td>
<td>Normal</td>
<td>83.16</td>
<td>85</td>
</tr>
<tr>
<td>II</td>
<td>Triton (350)</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td></td>
<td>2.414</td>
<td>1.915</td>
<td>1.839</td>
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<tr>
<td>III</td>
<td>EELS (100)</td>
<td>83.5</td>
<td>283.16</td>
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<tr>
<td></td>
<td>±</td>
<td>±</td>
<td>±</td>
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<td></td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td>IV</td>
<td>EELS (200)</td>
<td>85.5</td>
<td>149.16</td>
</tr>
<tr>
<td></td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td></td>
<td>2.029</td>
<td>4.408*</td>
<td>3.91*</td>
</tr>
<tr>
<td>V</td>
<td>Simvastatin (0.57)</td>
<td>85.16</td>
<td>75.16</td>
</tr>
<tr>
<td></td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
<tr>
<td></td>
<td>1.95</td>
<td>1.352*</td>
<td>2.088*</td>
</tr>
</tbody>
</table>

*P* values * < 0.05 ; ** < 0.001

Fig. 2—Effect of *L. siceraria* extract on frog heart preparation by using normal ringer (upper panel) and modified ringer (lower panel) solution [HB = heart beat]
The cardiotonic action of EELS may be considered as a contributory factor for its overall beneficiary effect on prevention of disorders related to cardiovascular system in general. The possibility of cardiac stimulation action of EELS due to presence of calcium salts was carefully examined by using perfusion fluid with reduced concentration of calcium. The position of cessation of cardiac contraction by higher doses of EELS was also different from that of calcium indicating that it does not resemble to that of Digitalis glycosides. The lack of any action on isolated blood vessels as indicated by hind limb perfusion rules out any adrenergic involvement.

It is interesting to note that the radical scavenging antioxidant activity of the fruit is located largely in its epicarp which may be used for extraction of the active principal.

Of further interest is the influence of EELS on cardiac function in frogs. The mechanism and significance of this aspect needs further investigation. It should also be interesting to isolate and characterize the active principle in the fruit.

References


