CHAPTER 7: 
HEPATOPROTECTIVE 
ACTIVITY OF CRUDE LEAF 
EXTRACTS OF Lasianthus 
lucidus Blume

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7.2 Materials and methods
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7.1 INTRODUCTION

The liver is the most important organ in the body. It plays a significant role in regulating various physiological processes. It is also involved in several vital functions in our body, such as secretion, metabolism and storage. It has to offer a lot to detoxicate toxic substances and synthesize useful principles (Guillouzo, 1992; Halliwell and Gutteridge, 1999). If this organ gets any injury or its function impairs, it may lead to serious implications on one’s health. The liver transforms and excretes many drugs and toxins. These substances are frequently converted to inactive forms by reactions that occur in the hepatocytes (Kim, 2005; Adewusi and Afolayan, 2010; Parmar, 2010). Management and cure of liver diseases is still a challenge to modern healthcare practices. For the alleviation of hepatic ailments the most important representatives are phytochemicals. Historically, plants have been used in the folk medicine to treat various diseases. Experimental works on several plants have been carried out to evaluate their efficacy against chemically induced liver toxicity (Mitra et al., 1998; Pingale, 2008). Many studies also suggest that, natural antioxidants from plant sources have been found to prevent and cure liver disorders by adopting various mechanisms of actions, so antioxidizing property of a plant may further be related with its hepatoprotective activity (Ashoush et al., 2013).

Carbon tetrachloride (CCl₄) was the first toxin to be discovered which have shown to produce injury mediated by free radical mechanism. It generally shows its toxicity symptoms in liver. Its toxic dose administered to experimental animals produce accumulation of fats in the liver. This is because it blocks the synthesis of the lipoproteins that carry triglycerides away from this organ. It is believed that CCl₄ is metabolised by the P₄₅₀ system to give a carbon-centred radical i.e., the trichloromethyl radical. Several P₄₅₀ are involved including CYP2E1, the ethanol inducible cytochrome P₄₅₀. Hence, CCl₄-induced hepatotoxicity serves as an excellent model to study the cellular, molecular and morphological changes in the liver (Clawson, 1989; Halliwell and Gutteridge, 1999).
Hepatoprotective activity was not reported from any species of the genus *Lasianthus*. No ethno-medicinal data was also available regarding the plant's (*L. lucidus*) use in liver disorders. As the leaf extracts possess alkaloids, fatty acids, phenols and flavonoids and these phytochemicals generally exhibit hepatoprotection (Germano *et al.*, 1999; Latha *et al.*, 2003; Sikdar and Dutta, 2008) and as the leaf extracts also exhibited antioxidant potential, hence a study on hepatoprotective activity on various leaf extracts of *L. lucidus* have been conducted.

### 7.2 MATERIALS AND METHODS

#### 7.2.1 Preparation of Leaf Extracts

The protocol for preparation of the four selected leaf extracts namely, PE, EA, AC and ME have been discussed earlier in section 2.2 of this thesis.

#### 7.2.2 Preparation of Water Decoction of *L. lucidus* Leaves

Firstly, 100 g of *L. lucidus* leaves were cleaned with water to clear the dust and sand and boiled in one litre and a half of water. The water boiled for 10-15 minutes and then the mixture was taken off and drained. Then, the fluid was collected in a glass container for the usage.

#### 7.2.3 Experimental Animals

Age-matched (7- to 10-week-old) pathogen free female Swiss albino mice were used to study the hepatoprotective activity of the *L. lucidus* leaf extract. The Institution Animal Ethics Committee of Assam University has approved the animal study for this research work. The animals were kept at 27°C, relative humidity 45–58% and light and dark cycles of 10 and 14 hours respectively, for a week before and during the experiments. Animals were provided with standard diet (Lipton India Ltd., Mumbai, India) and boiled water, ad libitum. The food was withdrawn 18–24 hours before starting the experiment. All experiments were performed in the morning. Ethical
guidelines and guidelines for the care of the laboratory animals were strictly followed (Zimmerman, 1983).

7.2.4 Acute Toxicity Studies

The WHO has set guidelines for toxicity studies of medicinal plants. It supports appropriate usage of medicinal plants and encourages the remedies, which are proved to be used with safety and efficacy (Research guidelines for evaluating the safety and efficacy of herbal medicine, 1993). The animals were kept fasting for over night providing only water, after which the extracts were administered orally at the dose of 100 – 1200 mg/kg and observed for 16 days. If mortality was observed in 2 out of 3 animals, then the dose administered was assigned as a toxic dose. If the mortality was observed in mice, then the same dose was repeated to confirm its toxicity. If mortality was not observed, the procedure was repeated for further higher dose. The changes in body weight, food and water intake as well as movements were also observed.

7.2.5 Drugs and Chemicals

Carbon tetrachloride: SD Fine Chemicals, Mumbai
Silymarin: Microlabs, Bangalore
Liquid paraffin: HiMedia Laboratories Pvt. Ltd., Mumbai
Reduced glutathione: HiMedia Laboratories Pvt. Ltd., Mumbai
All drugs and chemicals were purchased commercially and were of analytical grade.

7.2.6 Silymarin Dose Regimen

Silymarin tablets were obtained from a nearby clinic. Each tablet contains 500 mg of silymarin. 25 mg/ kg dose of silymarin was administered to mice orally and it was used as positive control. The Silymarin was turned into a fine powder using a mortar and pestle to increase the dissolution. The powdered silymarin was suspended in pure water and was administered orally according to the body weight of mice.
Grouping of Mice and Treatments

Fourty eight mice (25–30 g) were randomly divided into eight groups, each group consisting of six mice. The mice dose was calculated on the basis of surface area ratio (Ghosh, 2005).

Group I: Control (Pure water 10 ml/kg, oral dose)
Group II: CCl₄: Liquid paraffin (1:1; 2ml/kg, intraperitoneally)
Group III: Silymarin (25mg/kg, oral dose) + CCl₄: Liquid paraffin (1:1; 2ml/kg, intraperitoneally)
Group IV: Petroleum ether extract (PE) (200mg/kg, oral dose) + CCl₄: Liquid paraffin (1:1; 2ml/kg, intraperitoneally)
Group V: Ethyl acetate extract (EA) (200mg/kg, oral dose) + CCl₄: Liquid paraffin (1:1; 2ml/kg, intraperitoneally)
Group VI: Acetone extract (AC) (200mg/kg, oral dose) + CCl₄: Liquid paraffin (1:1; 2ml/kg, intraperitoneally)
Group VII: Methanol extract (ME) (200mg/kg, oral dose) + CCl₄: Liquid paraffin (1:1, 2ml/kg, intraperitoneally)
Group VIII: Decoction of leaf (DC) (200ml/kg, oral dose) + CCl₄: Liquid paraffin (1:1, 2ml/kg, intraperitoneally)

Test group animals (Group IV-VIII) were administered orally a dose of 200 mg/ kg of PE, EA, AC, ME extracts and aqueous decoction respectively in the form of aqueous suspension once daily. The group III- VIII animals were administered simultaneously CCl₄: liquid paraffin (1:1, 2ml/kg body weight, intraperitoneally) after 30 minutes of administration of the silymarin and extracts. All the groups were treated for 5 consecutive days (Jain et al., 2008). At the end of this period, animals were kept overnight fasting and were sacrificed on sixth day. Blood samples were withdrawn by puncturing cardiac membrane. The blood samples were allowed to clot for 45 minutes at room temperature. Serum was separated by centrifugation at 2500 rpm at 30°C for 15 minutes and analysed for various biochemical parameters. The liver was removed carefully after killing the animals by cervical dislocation. The livers were fixed in 10% buffered formalin and stored for determination of antioxidant enzyme levels and histopathological examinations.
7.2.8 Measurement of Biochemical Parameters

Blood samples were collected from heart under chloroform anaesthesia and the serum was used for the assay of marker enzymes namely serum glutamate-pyruvate-transaminase (SGPT), serum glutamate-oxalate-transaminase (SGOT), alkaline phosphatase (ALKP) and bilirubin. The enzyme levels were assayed by using Ultra Violet kinetic assay based on reference method of International Federation of Clinical Chemistry (IFCC) (Jendrassik and Grof, 1938; Henry, 1974; Wenger et al., 1984; Jain et al., 2008). The SGPT, SGOT and Bilirubin levels were assayed using standard kits obtained from Medsource Ozone Biomedicals Pvt. Ltd., India and that of ALKP was obtained from Synergy Bio, Quantum Biologials Pvt. Ltd., India.

After preparation of the liver homogenate, the clear supernatant obtained was used for the estimation of lipid peroxidation (MDA) (Ohkawa et al., 1976), total protein (Lowry et al., 1991), reduced glutathione (GSH) (Pari and Latha, 2004) and antioxidant enzymes viz. Catalase (CAT) (Sinha, 1972) and superoxide dismutase (SOD) (Budhwar and Kumar, 2000) levels.

7.2.9 Statistical analysis

Results of the biochemical estimations are reported as mean± Standard error (SE). Total variation, present in a set of data was estimated by one way analysis of variance (ANOVA) followed by Dunnett’s test using statistical package for social sciences (SPSS) version 10.0. P<0.05 was considered significant (Dunnet, 1964; Woolson, 1987).

7.3 RESULTS AND DISCUSSION

It is well-established that CCl₄ induces hepatotoxicity by metabolic activation, therefore, it selectively causes toxicity in liver cells maintaining seminormal metabolic function. The CCl₄ is biotransformed by the cytochrome P₄₅₀ system (CYP2E1) in the endoplasmic reticulum to produce trichloro methyl free radical (•CCl₃) when combined with cellular lipids and proteins in the presence of oxygen form trichloromethyl peroxy radical...
which may attack lipids on the membrane of endoplasmic reticulum faster than trichloromethyl free radical. Thus, trichloromethyl peroxyl free radical leads to cell death (Reckengel et al., 1989). Assessment of liver damage can be made by estimating the activities of serum SGPT, SGOT, ALKP and Bilirubin originally present in higher concentration in cytoplasm. When there is any hepatic disorder, these enzymes leak into the blood stream depending on the extent of liver damage (Reckengel et al., 1989).

In Rubiaceae, the plants with reported hepatoprotective activity are *Cephalis ipecacuanha*, *Cinchona succirubra*, *Coffea arabica*, *Gardenia jasminoides*, *Ixora coccinea*, *Mitracarpus scaber*, *Hedyotis corymbosa*, *Paederia foetida*, *Pavetta indica*, etc (Beers and Robert, 2004; Okonkwo and Msonthi, 1995; Arun and Asha, 2007; Govind, 2011; Latha et al., 2003; Germano et al., 1999; Rahman et al., 2006; Sikdar and Dutta, 2008). Alkaloids isolated from large number of plants have shown strong hepatoprotection. In case of most of the plants, their root extracts have been found to exhibit hepatocurative properties. Leaves of *Mitracarpus scaber*, *Hedyotis corymbosa*, *Anthocephalus cadamba*, *Paederia foetida* and *Spermacoce hispida* showed hepatoprotective agents (Germano et al., 1999; Rahman et al., 2006; Orwa et al., 2009; Sikdar and Dutta, 2008; Rathi et al., 2010). For testing the presence of hepatocurative properties, *in vivo* studies have been carried out with albino mice or rats and tested for biochemical parameters like SGPT, SGOT, ALKP, Bilirubin, etc. Hepatotoxicity was induced by hepatotoxins like carbon tetrachloride, paracetamol, etc and tested against plant extracts and standard medicine to check the degree of prevention of the disease by the extracts and compared with standards.

### 7.3.1 Acute Toxicity Studies

Before performing hepatotoxic studies by inducing CCl₄-treated liver injury in mice, acute toxicity studies of the plant extracts on mice should be carried out to find out the toxic doses of the extracts on which further biochemical and histopathological tests will be carried out. Acute toxic doses of PE, AC, EA, ME and water decoction of leaves of *L. lucidus* were found to
be 700, 500, 500, 1500 and 500 mg/kg respectively. The dose randomly selected here for studying hepatoprotective activities is 200 mg/kg in case of all the extracts and 200 ml/kg in case of water decoction of leaves.

### 7.3.2 Biochemical Parameters

The effects of *L. lucidus* extract on liver marker enzymes and serum bilirubin content are given in Table 7.1. The data showed that the control group demonstrated a normal range of SGPT, SGOT, ALKP and bilirubin levels, while the CCl₄-treated group showed elevated levels of SGPT, SGOT, ALKP and bilirubin, confirming that CCl₄ caused liver injury at higher doses. The elevation of cytoplasmic SGPT, SGOT and ALKP is considered an indicator for the release of enzymes from disrupted cells. Concentration of bilirubin has been used to evaluate hepatopathy induced by chemicals. Besides performing various normal functions, the liver excretes the breakdown product of haemoglobin, bilirubin, into bile. Various necrotizing chemical agents like CCl₄ produce sufficient injury to the hepatic parenchyma which results in large increases in bilirubin content (Sasidharan *et al.*, 2010).

On the other hand, the extract-treated groups showed interesting results. The results of Table 7.1 data showed that the tested biochemical parameters of the leaf extracts treated group were higher than those of the control group (*p* < 0.05), but it showed much lower levels of SGPT, SGOT, ALKP and bilirubin than the CCl₄-treated group, that is, the extract treatment significantly reduced the previously raised levels of SGPT, SGOT, ALKP and bilirubin in hepatotoxic mice. The decrease in the serum levels of these enzymes might be due to the presence of various secondary metabolites like phenolics and flavonoids in the leaf extracts that enhanced the liver’s regeneration ability.
Table 7.1: Effect of *Lasianthus lucidus* leaf extracts on serum biochemical parameters against CCl₄-induced hepatotoxicity in Swiss Albino mice.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>SGPT (U/L)</th>
<th>SGOT (U/L)</th>
<th>ALKP (U/L)</th>
<th>Bilirubin (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>50.63± 0.30</td>
<td>65.67± 0.50</td>
<td>105.03± 0.17</td>
<td>8.92± 0.23</td>
</tr>
<tr>
<td>Group II</td>
<td>144.33± 0.15</td>
<td>215.33± 2.14</td>
<td>156.63± 0.22</td>
<td>11.6± 0.18</td>
</tr>
<tr>
<td>Group III</td>
<td>76.24±0.78</td>
<td>41.32±0.23</td>
<td>76.88±0.19</td>
<td>10.52±0.18</td>
</tr>
<tr>
<td>Group IV</td>
<td>66.34±1.16</td>
<td>55.87±0.50</td>
<td>47±0.28</td>
<td>6.13±0.13</td>
</tr>
<tr>
<td>Group V</td>
<td>121.05± 0.79</td>
<td>65.76± 0.57</td>
<td>54.31±0.14</td>
<td>6.55±0.09</td>
</tr>
<tr>
<td>Group VI</td>
<td>58.19±0.64</td>
<td>45.91±0.88</td>
<td>86.51±0.27</td>
<td>0.684±0.01</td>
</tr>
<tr>
<td>Group VII</td>
<td>41.90± 4.24</td>
<td>171.10± 0.58</td>
<td>112.15± 0.28</td>
<td>7.85± 0.36</td>
</tr>
<tr>
<td>Group VIII</td>
<td>60.45± 0.57</td>
<td>67.84±0.37</td>
<td>87.22±0.12</td>
<td>7.90± 0.05</td>
</tr>
</tbody>
</table>

Values are expressed as mean± standard error (SE); n=6 in each group. ^P<0.01 vs control group; "P<0.05 vs control group; 'P<0.01 vs CCl₄ treated group; "P<0.05 vs CCl₄ treated group.

Figure 7.1 shows a comparison of SGPT levels of various leaf extracts with their standard control group. It is clear from the figure that the activity of SGPT in methanol extract (ME) group was close to its standard control group (P = 0.002). In figure 7.2 acetone extract (AC) exhibited SGOT level close to that of silymarin (P = 0.007) and lower than the control group. PE extract lowered the ALKP level in mice as compared with the control group (P = 0.003) followed by ethyl acetate (EA), acetone (AC), decoction (DC) and methanol (ME) extract (figure 7.3). In case of bilirubin, the lowest value has shown by AC extract much lower than that of control group (P = 0.002) (figure 7.4). The results of the study show that a significant protection from toxin CCl₄ damage may be achieved mostly through all the leaf extracts studied and the leaf decoction which suggests the presence plant’s protective constituents.
Figure 7.1: Activity of serum glutamate-pyruvate-transaminase (SGPT) (U/L) in serum of various groups of Albino mice. The activity of SGPT is shown as mean ± standard error, n=6.

Figure 7.2: Activity of serum glutamate-oxalate-transaminase (SGOT) (U/L) in serum of various groups of Albino mice. The activity of SGOT is shown as mean ± standard error, n=6.
Figure 7.3: Activity of Alkaline phosphatase (ALKP) (U/L) in serum of various groups of Albino mice. The activity of ALKP is shown as mean ± standard error, n=6.

Figure 7.4: Activity of Bilirubin (mg/ dl) in serum of various groups of Albino mice. The activity of Bilirubin is shown as mean ± standard error, n=6.
7.3.3 Lipid peroxidation

Malondialdehyde (MDA) level determination is one of the most commonly used methods for observing lipid peroxidation (Drotman and Lawhorn, 1974). The result suggests that there was a drastic increase in lipid peroxidation after CCl₄-treatment and it was inhibited by the treatment of plant extracts revealing that it exhibits potent hepatoprotective activity. The MDA level for silymarin was also found to be significantly decreased (p < 0.01) (Table 7.2).

7.3.4 Total Protein

Measurement of protein contents were mainly used to determine the purity of a specific protein. Total protein level was significantly (p < 0.05) reduced in the CCl₄ treated group when compared to the control which may be due to higher doses of CCl₄ administration which causes depletion of total proteins indicating tissue damage and were significantly elevated (p < 0.05) in plant extract treated groups. These were comparable with that of silymarin-treated group (Table 7.2).

7.3.5 Antioxidant enzymes and Glutathione Levels

Treatment with CCl₄ significantly (p < 0.01) depleted CAT, GSH and SOD levels which indicate their use in detoxification of toxic metabolites of the drug (Lowry et al., 1991). The leaf extracts and decoction restored the antioxidant enzyme levels significantly (p < 0.01) and reduced the CCl₄-induced oxidative injury, thus proving its antioxidant potential (Table 7.2).
Table 7.2: Effect of *Lasianthus lucidus* leaf extracts on liver malondialdehyde (MDA), total protein, glutathione (GSH) and antioxidant enzymes against CCl₄-induced hepatotoxicity in Swiss Albino mice.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>MDA (μmol/g tissue)</th>
<th>Protein (μg/ml)</th>
<th>GSH (μmols of GSH/g wet tissue)</th>
<th>SOD (units/mg liver protein)</th>
<th>CAT (units/mg liver protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>47.20±1.12</td>
<td>24.56±0.6 3</td>
<td>34.33±2.3 3</td>
<td>45.23±0.4 4</td>
<td>43.30±0.9 5</td>
</tr>
<tr>
<td>Group II</td>
<td>134.10±0.45 a</td>
<td>9.04±0.91 b</td>
<td>12.56±1.0 7 a</td>
<td>23.02±0.9 8 a</td>
<td>21.09±2.7 8 a</td>
</tr>
<tr>
<td>Group III</td>
<td>50.21±0.89 c</td>
<td>14.56±1.1 3 c</td>
<td>24.33±0.5 5 d</td>
<td>47.67±0.6 3 c</td>
<td>50.67±1.4 3 c</td>
</tr>
<tr>
<td>Group IV</td>
<td>56.42±0.56 c</td>
<td>19.32±0.6 4 d</td>
<td>74.34±0.6 4 c</td>
<td>56.43±2.6 7 d</td>
<td>67.11±1.0 2 c</td>
</tr>
<tr>
<td>Group V</td>
<td>75.34±2.34 c</td>
<td>17.37±0.6 4 c</td>
<td>65.01±1.1 0 c</td>
<td>64.39±0.3 4 c</td>
<td>50.23±0.1 8 c</td>
</tr>
<tr>
<td>Group VI</td>
<td>67.32±2.01 c</td>
<td>10.72±1.7 8 d</td>
<td>25.04±0.6 0 d</td>
<td>46.91±1.6 2 d</td>
<td>46.48±0.7 4 c</td>
</tr>
<tr>
<td>Group VII</td>
<td>71.27±0.53 c</td>
<td>16.21±0.2 5 d</td>
<td>30.13±0.7 3 c</td>
<td>88.03±0.9 0 c</td>
<td>70.34±3.0 1 c</td>
</tr>
<tr>
<td>Group VIII</td>
<td>85.59±0.88 c</td>
<td>11.31±3.7 8 c</td>
<td>29.75±0.9 0 c</td>
<td>39.11±0.1 8 c</td>
<td>41.43±0.7 3 c</td>
</tr>
</tbody>
</table>

Values are expressed as mean± standard error (SE); n=6 in each group. *P<0.01 vs control group; †P<0.05 vs control group; ‡P<0.01 vs CCl₄ treated group; §P<0.05 vs CCl₄ treated group.
7.3.6 Body weight of mice

Experimental data reveals a gradual increase in body weight of mice in group I i.e., the control group from 1st day to 5th day where as a gradual decrease was observed in case of CCl₄ intoxicated group (group II). In case of all other test groups (group III to group VIII) increase in body weight were recorded signifying their protection against hepatic injury. Maximum increase in body weight (31.2± 3.4 g) was reported in case of group VII i.e, the ME extract group. All the data were found to be statistically significant having P < 0.05 (Table 7.3). In the present study, it was observed that treatment of silymarin and various extracts and leaf decoction of *Lasianthus lucidus* showed an increase of body weight as compared to CCl₄-treated group indicating the sign of recovery. The plant material shows significant inhibition of the acute elevation of serum markers induced by CCl₄ than silymarin.

Table 7.3:- Effect of leaf extracts and decoction of *Lasianthus lucidus* on body weight of Swiss Albino mice.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Body Weight of Mice in Grams</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1st Day</td>
</tr>
<tr>
<td>Group I</td>
<td>25.33± 2.2</td>
</tr>
<tr>
<td>Group II</td>
<td>26.4± 2.4</td>
</tr>
<tr>
<td>Group III</td>
<td>25.2± 3.2</td>
</tr>
<tr>
<td>Group IV</td>
<td>26.3± 2.2</td>
</tr>
<tr>
<td>Group V</td>
<td>26± 1.1</td>
</tr>
<tr>
<td>Group VI</td>
<td>25.7± 3.3</td>
</tr>
<tr>
<td>Group VII</td>
<td>26.5± 4.4</td>
</tr>
<tr>
<td>Group VIII</td>
<td>26.7± 1.6</td>
</tr>
</tbody>
</table>

Values are expressed as mean± standard error (SE); n=6 in each group. *P<0.05 vs control group; ^P<0.05 vs control group
7.3.7 Histopathology

Histopathological liver sections of control group showed normal and distinct hepatic cells with sinusoidal spaces and central vein. Disarrangement of normal hepatic cells with vacuolisation and necrosis were observed in CCl₄ intoxicated group (Figure 7.5). In case of liver sections of leaf extract and leaf decoction treated groups, it was found that vacuole formation and necrosis had been minimised. Figure 7.3 represents photomicrograph of methanolic extract (200mg/kg) treated group and figure 7.2 that of silymarin treated group. The activities of leaf extracts groups were comparable with standard silymarin, supplementing the protective effect of the test drug and the standard hepatoprotective drug.
Figure 7.5: Transmission Electromicrograph of mice liver after CCl₄ treatment
Figure 7.6: Transmission Electromicrograph of mice liver after CCl₄ + silymarin (25 mg/ kg) treatment
Figure 7.7: Transmission Electromicrograph of mice liver after CCl₄ + methanolic leaf extract (200 mg/ kg) treatment
[Arrows represent vacuolisation and disintegration of membranes]
A comparison of the work conducted is done with some published works on other plants of the family Rubiaceae. In case of *Spermacoce hispida*, 200 mg/kg dose of ethanolic crude extract showed SGPT, SGOT and ALKP levels 85.56, 43.31 and 162.86 U/L respectively. Here toxicity was induced by nitrobenzene (Rathi *et al.*, 2010). In case of *Ixora coccinea*, 200 mg/kg dose of ethanolic extract represented the values 78.8, 30.1 and 60.3 U/L respectively of SGPT, SGOT and ALKP assays after toxication with aflatoxin B1 (Shymal *et al.*, 2010). CCl₄ induced hepatotoxicity in *Mitracarpus scaber* represents 191.6 and 184.4 U/L values of SGPT and SGOT levels for CCl₄ plus 250 mg/kg decoction treatment (Germano *et al.*, 1999). In all these three cases experiments were performed on Wistar albino rats and the methodology adopted for performing the experiments were different. SGPT, SGOT and ALKP levels were elevated after treatment with hepatotoxin and these elevated values were reduced after treatment with plant extracts. In case of present study also, after CCl₄ treatment an elevated level of the marker enzymes were observed and then in next phases, plant extract treatments reduced those elevated levels. In case of 200 mg/kg methanolic extract treated group of *Lasianthus lucidus* the SGPT, SGOT and ALKP levels obtained were 41.9, 171.1 and 11.2 U/L respectively.

Ethanolic extract (200 mg/kg) of *Spermacoce hispida* elevated the levels of antioxidant enzymes, SOD, CAT and GSH by 18.78, 54.7 and 25.2 U/L mg protein which were reduced in nitrobenzene treated groups (Rathi *et al.*, 2010). In case of methanolic extract of *Lasianthus lucidus* the values were 30.13, 88.03 and 70.34 U/L mg protein of GSH, SOD and CAT respectively which shows nearly similar findings in both the cases. Histopathological studies also represented similar observations in all the studied plants of the family Rubiaceae. The results reveal that the present study corroborates with the previous works performed on different plants of the same family.

### 7.4 CONCLUSION

From the present study, it can be concluded that *L. lucidus* leaf extracts and water decoction of leaf show hepatoprotective activity against CCl₄ -
induced hepatotoxicity. Hence these leaf extracts may be act as prophylactic as well as curative drug in treating hepatotoxic conditions.

REFERENCES


