Chapter 4. Evaluation of antihepatotoxic potential of *Achyranthes aspera* L.

4.1. Introduction

*Achyranthes aspera* Linn. (Family: Amaranthaceae) commonly known as Apamarga herb, occurs throughout India and Tropical Asia (Nadkarni and Nadkarni, 1976). Various parts of the herb, *A. aspera* have been studied for several pharmacological actions. Saponin isolated from *A. aspera* was tested for its effect on the phosphorylase activity of rat heart (Ram et al., 1971). Extracts of *A. aspera* were evaluated for their hypoglycemic effect in normal and alloxan diabetic rabbits (Akhtar & Iqbal, 1991). Formulation containing equal parts of *A. aspera*, *Chicorium intybus* and *Berberis aristata* were put to therapeutic trial in paracetamol-induced hepatopathy in sheeps (Bhaumik and Sharma, 1993). Therefore present study was aimed to evaluate anti-hepatotoxic effect of methanol extract of *A. aspera* against aflatoxin-induced hepatotoxicity. The efficacy of *Achyranthes aspera* L. was tested by using biochemical and histological evaluation.

4.2. Materials and Methods

4.2.1. Plant material

Root parts of *A. aspera* were collected from outfield near Muthayammal College of Engineering, Rasipuram, and Tamilnadu, India. The plant was authenticated by comparison with reference specimens preserved at the Rapinat Herbarium, St. Joseph's College, and Tiruchirapalli. Voucher Herbarium specimens were kept in the Herbarium for future references. The root parts of the plant was properly cleaned and dried first in air and then artificially in an oven at 60°C for approximately 4h.
4.2.2. Extract preparation

Soxhlet extraction of powdered root parts was done using methanol. The extract was left in 13.0% (w/w). Preliminary phytochemical evaluation of the extract was done by the method recommended by Kokate, 1996.

4.2.3. Animal model

Male albino rats of Wistar strain (*Rattus norvegicus*) weighing 140 ± 20g (12–14 weeks) were obtained from Tamilnadu Veterinary and Animal Sciences University, Chennai, India. The rats were maintained under standard conditions of humidity, temperature (25 ± 2 °C) and light (12h light/12h dark) and fed a standard rat pellet diet (M/s Pranav Agro Industries Ltd, Sangli, India); drinking water was given *ad libitum*. The animal experiments were conducted according to National Institute of Health guidelines for the care and use of laboratory animals (1985).

4.2.4 Experimental design – Biochemical evaluation

Ten groups each containing six animals were used in this experiment. The experimental period was 21 days. Group-I was fed with basal diet. Silymarin and *A.aspera* root extracts were given orally with water for 12 days to III, IV (*A.aspera* root extract 100mg, 200mg), V and VI (Silymarin 25mg, 50mg) groups of rats. A single dose of aflatoxin (1mg/Kg) was given to each rat on 13th day with drinking water. Group- II received aflatoxin alone on 13th day. On 15th day rats in group I – VI were separated and from the rats, blood was collected from retro-orbital plexus using fine needles.

The blood samples were allowed to clot for 15 minutes at room temperature. Serum was separated by centrifugation at 2500rpm for 15 minutes and analyzed for biochemical parameters such as serum glutamic oxaloacetate transaminase (SGOT)
(Reitman and Frankel, 1957), serum glutamic pyruvic transaminase (SGPT) (Reitman, and Frankel, 1957), and alkaline phosphatase (ALKP) (King, 1965a, b, and c) and total bilirubin (T.Bil.) (Malloy and Evelyn, 1937). Liver and kidneys from one animal of each group were isolated to study the histopathological changes. Group VII– X were given 1mg/kg aflatoxin as a single dose on 13th day. *A. aspera* root extract was given to group VII (100mg/kg), VIII (200mg/kg). Silymarin was given daily at a dose level of 25mg and 50mg/kg for group IX, X till 21st day. On the 21st day rats were sacrificed and the procedure was followed as mentioned earlier.

4.2.4.1. Estimation of Serum glutamate oxalo acetate transferase (SGOT) activity. Reitman and Frankel, 1957.

**Reagents**

1. SGOT Buffered Substrate: 13.3g of aspartic acid was dissolved in 90ml of 1N NaOH and 0.146g of α-ketoglutaric acid was added, dissolved by adding few drops of 1N NaOH, adjusted the pH to 7.4. The substrate was made up to 500ml with Phosphate buffer.

2. Dinitrophenylhydrazine (DNPH) Reagent: 99mg of DNPH was dissolved in 50ml conc. HCl and made up to 500 ml with water and stored.

3. 0.4 N NaOH

4. Stock pyruvate standard: 220mg of sodium pyruvate was dissolved in 100ml of phosphate buffer.

5. Working standard: 1ml of Stock was diluted in 100ml of phosphate buffer

**Procedure**

Two tubes named test and blank were taken and to each tube 0.5ml of SGOT buffered substrate was added and incubated at room temperature for few minutes and
to the test tube 0.1 ml of serum was added and incubated at 37°C. 0.5 ml of DNPH was added, mixed well and incubated at room temp for 20 minutes. Then 5 ml of 0.4N NaOH was added to the mixture and waited for 10 minutes. Then the OD value was read using a green filter in spectrophotometer (S - 20). The activity of SGOT was measured as U/ ml of serum.

4.2.4.2. Estimation of Serum glutamate pyruvate transferase (SGPT). Reitman and Frankel, 1957.

Reagents

i. Phosphate buffer (pH 7.4): 11.3 g of dry anhydrous Na₂HPO₄ and 2.7 g of dry anhydrous potassium dihydrogen phosphate were dissolved and overall volume was made up to 1 litre with water.

ii. SGPT Buffered Substrate: 9 g of alanine was dissolved in 90 ml of water and the pH was adjusted to 7.4 by adding 2.5 ml of 1 N NaOH. 0.146 g of α-keto glutaric acid was dissolved by adding few drops of NaOH and adjusted to pH 7.4. The volume was made up to 500 ml with phosphate buffer.

iii. DNPH Reagent: 99 mg of DNPH was dissolved in 50 ml of conc. HCl and made up to 500 ml with water.

iv. 0.4 N NaOH

v. Stock Pyruvic acid Standard: 200 mg of sodium pyruvate was dissolved in 100 ml of phosphate buffer.

vi. Working standard: 1 ml of the stock solution was diluted to 10 ml of phosphate buffer.
**Procedure**

SGPT substrates of 0.5ml were taken in two test tubes and were incubated at room temperature for a few minutes. The tubes were added with 0.5ml of serum, incubated for 30 minutes at 37°C. 0.5ml of DNPH was added, mixed well and incubated at room temp for 20 minutes. 5 ml of 0.4N NaOH was added. After 10 minutes the OD was read using a green filter in a spectrophotometer. The SGPT activity was measured as U/ml of serum.

4.2.4.3. Estimation of serum alkaline phosphatase (ALKP). King, 1965 a,b,c.

**Reagents**

(i) Disodium phenyl phosphate: - (0.01M). 1.09g of Disodium phenyl phosphate was dissolved in water and made up to 500ml and cooled.Little chloroform was added and kept it in the refrigerator.

(ii) Sodium carbonate:- NaHCO₃ buffer (0.1M) - 3.18g of anhydrous Na₂CO₃ and 1.68g of NaHCO₃ were dissolved in water and made up to 500ml.

(iii) Buffered substrate was prepared by mixing equal volume of solution (1) & (2) with pH of 10 for use.

(iv) Tri chloro acetic acid (TCA) 20% (w/v).

(v) Acid molybdate Reagent: - 5g of Ammonium molybdate was dissolved in 5N H₂SO₄.

(vi) 1, 2, 4, Amino napthal sulphanilic acid (ANSA);-0.25% of ANSA was prepared by adding 0.5g ANSA to 195ml of 15% NaHSO₄ and 5ml of the 20% Na₂SO₃.

(vii) Stock Solution: - 2.194g of pure KH₂PO₄ was dissolved in water and made up to 500ml. A few drops of chloroform was mixed to the stock solution and stored.
(viii) Working standard: - 2ml of stock standard was diluted to 500ml of water (5ml is equal to 0.02mg)

**Procedure**

Buffered substrate of 6ml was poured into a test tube and placed it in a water bath at 37°C for few minutes, and then added 0.3ml of serum. The tubes were mixed well and incubated for 15 minutes. 1.2ml of 20% TCA was added, shaken well and filtered. At the same time control and Blank was set up. To blank and control 1.2ml of 20% TCA was added, mixed well and filtered. 5ml of filtrate was taken from test, blank and control, 0.8ml of acid molybdate was added and followed by 0.2ml of ANSA. The preparation was mixed well and kept undisturbed for 10 minutes and taken reading at 680nm in a spectrophotometer. The activity of ALKP was measured by μ moles x 10^-2 phenol liberated /minute.

4.2.4.4. Estimation of Total Bilirubin (T.Bil). Malloy and Evelyn, 1937.

**Reagents**

**Diazot reagent:** Diazot reagent was freshly prepared by mixing 10ml solution A and 0.3ml of solution B.

**Solution A:** 1g of sulphanilic acid and 1.5ml of conc. HCl were mixed and made up to one litre with water. This solution can be kept indefinitely at room temperature.

**Solution B:** 0.5g sodium nitrite was dissolved in 100ml of water and kept in refrigerator.

(i) 1.5% HCl: 1.5ml of conc. HCl was added to 100ml of water.

(ii) Bilirubin standard (10mg per 100ml). 10mg of bilirubin was weighed (working away from bright light) dissolved in a minimum (about 5ml) of 0.1N
sodium carbonate solution as quickly as possible in a 100ml volumetric flask, since it was unstable in alkaline solution. The final volume of 100ml was obtained by adding methanol or chloroform.

**Procedure**

0.2ml of serum and 1.8ml of water were taken in two small test tubes (blank and test). 0.5ml of diazo-reagent was added to one tube (test) and 0.5ml of 1.5% of HCl was added to the next (blank). Finally 2.5ml of methanol was added to both test tubes. The test tubes were undisturbed for 30 minutes and OD was read using a green filter (540nm) in a spectrophotometer. Total bilirubin was calculated as μ moles/ml of serum.

4.2.4.5. Statistical analysis

Results of the biochemical estimations are reported as mean ± standard error of mean (S.E.M.). Total variations, present in a set of data was estimated by one-way analysis of variance (ANOVA), Student's t-test was used for determining significance.

4.3. Results and Discussion

To assess the hepatic functioning, standard liver functional tests like SGPT, SGOT, AIKP, and T.Bil. were done for control, aflatoxin induced rats and plant extract treated rats. The mean SGPT value in the control rats was 19.90 ± 5.82 (U/ml serum). After the administration of AF at a dose level of 1mg kg⁻¹ body weight, the SGPT level was elevated to 137.71 ± 21.70 (U/ml serum) (Table 4:1) (Fig. 4.1). The percentage of increase in SGPT level was due to the aflatoxin consumption. This clearly indicates that aflatoxin interferes with the normal functioning of liver in rats and hyper production of SGPT level, indicates the disfunctioning of liver. To
ameliorate the hepatotoxicity of aflatoxin two different doses of the extract of the plant *A. aspera* was given to the aflatoxin treated rats.

**Table 4.1.** Effect of methanolic extract of *A. aspera* L. on aflatoxin induced hepatotoxicity in rats (Pretreatment).

<table>
<thead>
<tr>
<th>Groups</th>
<th>SGPT</th>
<th>SGOT</th>
<th>ALKP</th>
<th>T.Bil.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>19.90±5.82</td>
<td>33.20±5.14</td>
<td>36.41±6.13</td>
<td>0.30±0.06</td>
</tr>
<tr>
<td>AF(1mg/kg)</td>
<td>137.71±21.70***</td>
<td>169.15±6.88***</td>
<td>193.21±37.61***</td>
<td>2.02±0.14***</td>
</tr>
<tr>
<td></td>
<td>(592)</td>
<td>(409.48)</td>
<td>(430.65)</td>
<td>(573.33)</td>
</tr>
<tr>
<td>Test ext.(100 mg/kg)</td>
<td>36.04±5.93***</td>
<td>49.75±0.18***</td>
<td>51.27±11.91***</td>
<td>0.52±0.07***</td>
</tr>
<tr>
<td>+AF (1mg/kg)</td>
<td>(73.82)</td>
<td>(70.58)</td>
<td>(73.46)</td>
<td>(74.25)</td>
</tr>
<tr>
<td>Test ext (200mg/kg) +</td>
<td>43.23±7.88****</td>
<td>51.33±10.22***</td>
<td>73.52±5.32***</td>
<td>0.85±0.06***</td>
</tr>
<tr>
<td>AF (1mg/kg)</td>
<td>(73.52)</td>
<td>(71.69)</td>
<td>(64.17)</td>
<td>(78.31)</td>
</tr>
<tr>
<td>Silymarin (25 mg/kg)</td>
<td>34.89±3.22 ***</td>
<td>47.88±4.57***</td>
<td>28.91±2.72***</td>
<td>0.05***</td>
</tr>
<tr>
<td>+AF (1mg/kg)</td>
<td>(74.66)</td>
<td>(71.69)</td>
<td>(85.03)</td>
<td>(86.13)</td>
</tr>
<tr>
<td>Silymarin (50 mg/kg)</td>
<td>33.57±5.20 ***</td>
<td>50.85±4.83***</td>
<td>75.61±8.01***</td>
<td>0.12***</td>
</tr>
<tr>
<td>+ AF (1mg/kg)</td>
<td>(79.44)</td>
<td>(71.95)</td>
<td>(63.11)</td>
<td>(77.80)</td>
</tr>
</tbody>
</table>

The levels of biochemical parameters were expressed as U/ml of serum.

Values are expressed as mean ± SEM. Aflatoxin group was compared with control group. Test extract groups were compared with aflatoxin group. **p<0.01; ***p<0.001; Values of test group in parenthesis indicate the % change in relation to the aflatoxin group and aflatoxin group to the control.
Table 4.2. Effect of methanolic extract of *A.aspera* L. on aflatoxin induced hepatotoxicity in rats (Drug and *A. aspera* root extract given after administration of aflatoxin).

<table>
<thead>
<tr>
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</tr>
<tr>
<td></td>
<td>(592)</td>
<td>(409.48)</td>
<td>(480.65)</td>
<td>(573.33)</td>
</tr>
<tr>
<td>AF(1mg) + Test ext(100mg/kg)</td>
<td>79.72±7.8**</td>
<td>75.81±9.46**</td>
<td>83.57±5.78**</td>
<td>1.31±0.17**</td>
</tr>
<tr>
<td></td>
<td>(42.11)</td>
<td>(55.18)</td>
<td>(56.74)</td>
<td>(35.14)</td>
</tr>
<tr>
<td>AF(1mg) + Test ext(200mg/kg)</td>
<td>69.09±6.41**</td>
<td>75.17±8.77**</td>
<td>79.34±1.42**</td>
<td>1.03±0.11***</td>
</tr>
<tr>
<td></td>
<td>(57.68)</td>
<td>(58.54)</td>
<td>(61.33)</td>
<td>(73.72)</td>
</tr>
<tr>
<td>AF(1mg) + Silymarin (25mg/kg)</td>
<td>65.89±5.41**</td>
<td>69.54±2.33**</td>
<td>58.32±13.81**</td>
<td>0.59±1.22***</td>
</tr>
<tr>
<td></td>
<td>(52.15)</td>
<td>(58.88)</td>
<td>(69.81)</td>
<td>(70.79)</td>
</tr>
<tr>
<td>AF (1mg) + Silymarin (50mg/kg)</td>
<td>48.32±3.23***</td>
<td>54.67±6.21***</td>
<td>53.12±1.3***</td>
<td>0.91±2.11***</td>
</tr>
<tr>
<td></td>
<td>(70.40)</td>
<td>(69.84)</td>
<td>(74.11)</td>
<td>(76.78)</td>
</tr>
</tbody>
</table>

The levels of biochemical parameters were expressed as U/ml of serum.

Values are expressed as mean ± SEM. Aflatoxin group was compared with control group. Test extract groups were compared with aflatoxin group. **p<0.01; ***p<0.001; Values of test group in parenthesis indicate the % change in relation to the aflatoxin group and aflatoxin group to the control.

On treatment with 100mg/ Kg *A. aspera* extract for the rats prior to the administration of 1mg AF/ kg, the SGPT level had decreased significantly. However when the extract of *A.aspera* was given at a dose level of 200mg/ kg, the decrease in SGPT level was little less when compared to the previous treatment. This study indicated that *A.aspera* extract at a quantity of 100 mg/ kg was the minimal effective
dose. The result was compared with the effect of standard hepatoprotective drug silymarin. Two different dose levels of silymarin was given (25mg/ kg, 50mg/ kg).

![Graph showing changes in serum biochemical parameters in rats pretreated with A.aspera, Silymarin and aflatoxin.]

From the result, it was inferred that silymarin was able to reduce the hike in SGPT level considerably (36.04±5.93 (73.82%), 43.23±7.88 (73.52%)). The effectiveness of A.aspera treatment was compared with silymarin treatment. From the comparison, it was found that the plant extract was more effective than the silymarin. So A.aspera is having a novel bioactive compound that can be used for hepatoprotective function in the cure of aflatoxicosis.

The effectiveness of A.aspera and silymarin treatment, before the administration of aflatoxin and after the administration of aflatoxin was carried out. When A. aspera was given after the administration of aflatoxin it was found that the decrease in SGPT level was a little higher when compared to administration of A.aspera prior to treatment (group III, IV). In the case of pretreatment with A.aspera
(100 mg/kg), the decline in SGPT level was 36.04 ± 5.93. However, after treatment of *A. aspera* was able to give a lesser impact and the SGPT level was 79.72±7.8 (U/ml of serum) (non-significant).

The second biochemical marker serum glutamate oxaloacetate transaminase (SGOT) level in control rats fed with feed alone was found to be 33.205 ± 5.14 U/ml of serum. Drastic change was observed in the level of SGOT in aflatoxin (1 mg) treated rats which was about 169.15 ± 6.88 U/ml of serum (table 4.1). The elevation of enzyme level is due to the damage of liver.

![Figure 4.2](image)

**Fig:4.2.** Changes in serum biochemical parameters (U/ml) in rats treated with *A. aspera*, silymarin (mg/kg) and aflatoxin (1 mg/kg). 1: AF control; 2: AF + *A. aspera* 100 mg/kg; 3: AF 1 mg + *A. aspera* 200 mg/kg; 4: AF 1 mg + Silymarin 25 mg/kg; 5: AF + Silymarin 50 mg/kg.

*A. aspera* root extract of two doses (100 mg/kg, 200 mg/kg) were given to different groups of rats as a prophylactic measure prior to aflatoxin (1 mg/kg) administration. The mean SGOT level of 100 mg/kg root extract treated animals was found to be 49.75 ± 1.18 (Table 4.1). And the percentage of decrease in elevated level of SGOT was nearly normal (P<0.001) (Fig.4.1). The mean value of SGOT in rats
treated with 200mg/kg *A. aspera* root extract was 51.33 ± 10.22 U/ml of serum (Table 4.1). The liver damage induced by aflatoxin (1mg/kg) was repaired by the minimal dose of *A. aspera* root extract of 100mg/kg. Whereas increased level of root extract (200 mg/kg) is not much effective in curing liver damage.

Pre-treatment with standard drug silymarin at two levels of doses (25 mg, 50 mg/kg) showed increased curative effective (mean) level of SGOT for silymarin 25 mg/kg treated rats was 47.88 ± 4.57 (P < 0.001) (Table 4.1) and for silymarin 50 mg/kg treated rats was 50.85 ± 4.83 U/ml of serum (P < 0.001). The percentage of reduction, in elevated levels of SGOT in silymarin (25mg, 50mg/kg) pretreated rats were 71.69, 71.95 respectively. (Fig. 4.1).

The rats treated with *A. aspera* root extracts (100mg, 200mg/kg) and silymarin (25mg, 50mg/kg) after administration of aflatoxin (1mg/kg) showed less curative effect or protection (N.S) (Table 4.2); (Fig.4.2) when compared to pretreatment with the silymarin and *A. aspera* root extract.

The third biochemical marker alkaline phosphatase (ALKP), isoenzyme mean level of normal control rat was identified as 36.41 ± 6.13 U/ml of serum. (Table 4.1). The aflatoxin treated rats showed elevated level of ALKP, and it was about 193.21 ± 37.61 U/ml of serum (Table 4.1). The percentage of increase in ALKP is about 472 (Fig. 4.1) at the significance level of P < 0.001%.

The pretreated rats with *A. aspera* at different dosage levels 100 mg, 200mg/kg, showed marked decrease in ALKP; in rats treated with 100 mg/kg *A. aspera* root extract, the level was 51.27 ± 11.91 U/ml of serum (Table 4.1). The percentage of protection is comparatively increased to 73.46 when compared to all other groups (Fig. 4.1) at 0.001% level; in rats, treated with 200mg/kg root extract, the ALKP level
was found to be $73.52 \pm 5.32$ U/ml of serum (Table 4.1). When compared to hepatoprotective activity of 100mg/kg *A. aspera* extract for 1mg/kg aflatoxin, the percentage of protection is less.

The rats treated with silymarin (25mg, 50mg/kg), before administration of aflatoxin (1mg/ kg) as prophylactic measure, showed marked decrease in the ALKP level. The mean value of ALKP was $28.91 \pm 2.72$ U/ml of serum for 100mg/kg silymarin treated rats and it was $75.61 \pm 8.01$ (Table 4.1). Generally, the percentage of reduction of elevated ALKP level were also more (85.03 (25mg/kg silymarin), 63.11 (50mg/kg silymarin)) (P < 0.001%).

The rats treated with *A. aspera* root extract (100mg, 200mg/ kg) and silymarin (25mg, 50mg/1kg) after the administration of aflatoxin (1mg/kg) showed slow improvement in regaining normal level of ALKP level (Fig. 4.2) (Table. 4.2) (Non significant).

Total bilirubin (T.Bil) of control rats showed the normal value (Mean value) of bilurubin was $0.30 \pm 0.06$ U/ml of serum (Table 4.1). High level of total bilirubin ($2.02 \pm 0.14$) was found in aflatoxin (1mg/kg) treated rats. The percentage of increase against control was at the level of 0.001% significance. (Fig 4.1). The elevated level of bilirubin was decreased effectively in rats pretreated with *A. aspera* root extract (100mg/kg) and the level was $0.52 \pm .07$ (U/ml of serum). The percentage of protection was found as 74.25 (Fig 4.1) at the level of 0.001% significance. The rats treated with 200mg/Kg *A. aspera* root extract showed little protection when compared to the previous group and the value is $0.85 \pm .06$ (Table 4.1)
The standard antihepatotoxic drug (silymarin) (25mg, 50mg/kg) pretreated rats showed significant decrease in increased bilirubin level. Silymarin 25mg/kg administered rats showed the mean bilirubin level of 0.28 ±0.05 (Table 4.1) and the percentage of reduction was found to be 86.13 (P<0.001) (Table 4.1). However increase in dose of silymarin (50mg/kg) showed less protection against hepatotoxicity (77.80%) (Fig 4.1).

The rats treated with A.aspera root extract (100mg, 200mg/kg) and silymarin (25mg, 50mg/kg) after administration of aflatoxin (1mg/kg) showed low activity in regaining the normal level. The mean value of total bilirubin was found to be 1.31±0.17 U/ml of serum (Table 4.2) for rats treated with 100mg/kg A.aspera root extract and 1.03±0.11U/ml of serum for rats treated with 200mg/kg A.aspera root extract. The percentages of protection for these two groups were also decreased when compared to the same dosage levels given in groups of pretreatment (Table 4.1).

Silymarin (25mg, 50mg/kg) were given to the last two groups. The mean bilirubin levels for these two groups were found to be 0.57±1.22, 0.91±2.11 (Table 4.2). The protection percentage were also decreased to 70.79 and 76.78 (Fig 4.2) when compared to silymarin pretreated rats. This may be due the inability of the liver to repair the liver due to aflatoxin induced damage.

The effective role of A.aspera to correct the impaired liver was due to its phyto chemical constituents. According to Bowman & Rand (1982), the hepatoprotectivity activity of drug against aflatoxicosis was due to the inhibitory effect
on the formation of active metabolites which in turn reduces dry metabolizing enzymes and its binding ability to RNA polymerase to inhibit transcription.

*Ichnocarpus frutensis* Linn (Deepak *et al.*, 2007), *Aclypa recemosa* (Iniaghe *et al.*, 2008) were known to exert hepatoprotection. Some herbal drug formulations have active compounds against hepatotoxicity. According to Khalid *et al.*, 2002 alkaloids are good hepatoprotective compounds in *Artemisia scoparia*. *A.aspera* is also having bioactive compounds to impose hepatoprotective activity in a similar way.