PHYTOCHEMICAL INVESTIGATION AND HEPTOPROTECTIVE EVALUATION ACACIA RUBICA EXTRACT ISOMIZED AND PARACETAMOL INDUSED ANIMAL TOXICITY

Akshit Kumar¹, Surbhi Gupta², Dr Sachin Tyagi³, Surykant Verma⁴, Shivam⁵, Dhananjay Taumar⁶, Roshan Kumar⁷, Anubhav Dubey⁸

¹,²,³,⁴,⁵,⁶Dept. Of pharmacology, bit meerut, up, india.
⁷Dept. Of pharmacology, dev bhoomi institute of pharmacy and research, dehradun, india.
⁸Assistant professor, department of pharmacology, maharana pratap college of pharmacy kanpur (u.p.) – india.

ABSTRACT

OBJECTIVE

The danger of liver toxicity has recently grown with the growth in exposure to environmentally friendly poisons, pesticides, and chemical therapies which has led to the liver disease being a critical public health concern. Developments in several major medical fields such as emergency medicine, surgery, radiography, antibiotics have occurred, etc.; however, the production of hepatoprotective drugs and the efficient treatment of liver disease has not succeeded. And most traditional therapies took this matter seriously. Many medicinal herbs in India are utilized in the traditional restaurant technique to treat liver illness.

MATERIAL AND METHODS

The studies were performed on Swiss four-month albino mice of both sexes between 20 and 25 g. They have been supplied from Animal House, Pharmacy School, B.I.T. Meerut. The animals were acclimatised to the normal laboratory settings at the cross ventilated animal housing at 25±2°C of relative humidity of 44 –56% and light and dark cycles of 12:12 hours.

RESULT

Histopathological analysis of the liver sections obtaining from several Swiss albino mice has supported the observation of the enzyme research. Centrilobular necrosis was prevalent in paracetamol treatment. Hepatic cells have been deficient with cytoplasm, and hepatic cells have seen with vacuolation. The cell limits were indistinguishable. Total amount A. arabica group necrosis alcoholic extract and methanol extract were portals. The lobular vein containing blood cells was conspicuous. Prominent were the cell nucleus and nucleoli.

CONCLUSION

Alcohol (after defeating with petroleum ether) and other solvents using soxhlet extractor used for extraction from both plants A. Arabica. A. arabica was measured by the total alcoholic extracts 28,71 percent and various successive extracts. phytochemical testing comprised chemical assays and chromatography of various extracts produced by complete alcoholic and subsequent the presence of carbohydrates, glycosides, steroids, saponin, flavonoids, alkaloids, tannins, phenolics, proteins, and amino acids demonstrated in extracts from A. arabica.

I. INTRODUCTION

The liver is one of the most significant organs in the body and contributes roughly 2% to around 1.5 kg of the typical bodyweight of an adult. Hepatic cells or hepatocytes conduct many liver activities. Almost every organ in the body has liver support and is necessary for survival. The liver likewise has a crucial position and multifaceted
function that is prone to various illnesses. Chronic liver diseases are among the world's top issues, with liver cirrhosis and liver drug-induced damage as the ninth most significant cause of mortality in Western and developing nations. In addition, several hepatic diseases are created in the intestinal poisons ingested. The liver is an effective detox organ for the use and disposal of endogenous chemicals.

Liver disease
Various liver diseases include:

Cirrhosis
The term cirrhosis originates from the Greek word kirrhós; tawny signifies an orange-yellow hue of the sick liver. Cirrhosis is the consequence of chronic liver illness with decreased hepatic function caused by fibrosis, scar tissue, and regenerating nodules. Cirrhosis commonly caused by alcoholism, hepatitis B, and hepatitis C. However, there are many more possible explanations for Cirrhosis besides Cirrhosis. Idiopathic cases indeed (i.e., of unknown cause).

fatty liver
Large liver fatty triglyceride vacuoles in hepatic cells build up during the process of stenosis, commonly known as Fatty Hepatic Disease (FLD) (i.e., abnormal retention of lipids within a cell). Although various reasons exist, fatty liver can treat as a single illness for those who use excessive alcohol and are obese globally (with or without effects of insulin resistance). The condition linked with other disorders that impair fat metrology.

Fibrosis of the liver
Fibrosis of the liver is the hepatitis scarring process. Like other skin and tissue, the liver substitutes for injury by depositing collagen and other matrix elements in fresh collagen. This process can lead to liver cirrhosis, which damaged by the architecture of the functional units of the liver that blood flows into the liver and liver function. Severe liver disease consequences, such as portal hypertension, liver failure, and hepatic malignancy, can arise when Cirrhosis established. Furthermore, with the progression of Cirrhosis as a premalignant condition, the risk of liver cancer increases substantially. Cirrhosis and liver cancer are world-leading 10 causes of mortality in many advanced nations, and liver disease is one of the five most important causes of medium-aged death.

Hepatitis virus
Inflammation of the liver is viral hepatitis induced by viral infection. It can occur acutely or chronically (recent rash, relatively fast onset). The most frequent causes of viral hepatitis include Hepatitis A, Hepatitis B, Hepatitis C, Hepatitis D, and Hepatitis E. In addition to nominal hepatitis viruses, herpes simplex and cytomegaloviruses also induce inflammation of hepatitis. Liver illness can influence the disposition of drugs in several ways (particularly liver cirrhosis). Reduces liver cells and improves portocaval shunting Serum albumin is lowered, acidic drug-protein binding (diclofenac, warfarin, etc.) is diminished, and extra medicine in its open form is accessible. There is a reduced metabolism dose and a decrease in some medicines (morphine, lidocaine, and Propranolol)

Hepatoprotective agents are drugs that reduce liver injury.

Drug and chemical hepatotoxicity are the most prevalent cause of iatrogenic illness. Some inorganic chemicals that induce hepatotoxicity include arsenic, arsenic, copper, and iron. In addition, the synthetic community of organic molecules is a substantial number of medicinal medicines. Hepatotoxic exposure may also be unintentional, murderous, or suicidal, occupational, environmental, or domestic.

Acacia is a genus containing over 1000 species, most of which reside in Asia. In every location, from seashore to arid inland highlands, acacia exists. Acacias grouped as "wattles," an early English word for twigs and rods. The individual blooms are small but have a wide variety of rod-like or round heads. Wattle may found in the flora at any time of year. Seeds are growing in flat, short, elongated, or cylindrical pods (legumes)—the complex leaves which appear in most Acacia plants quickly replaced by flattened stalks known as phyllodes. Phyllodes are leaf-like and can vary enormously in size and form. The seeds themselves are generally quite durable. Historically, the bark is a firm tannin, astringent, and tonic utilized as food, tannins, and medicinal resources for many Acacia species. Several organizations recently started research to examine the nutritional capability and harmful consequences of different species.
Morphology of Plant

A little tree fissured with a longitudinal dark brown or black. When young, the bark is a thin, terete, pubescent branchlet. The blades are pinnate and 5-10 cm long, downy, and occasionally decorated with Chachi glans (carrying flower stalks in short intervals). The petioles are 2.5-5 cm long, and the spines are pretty varied, lengthy, and fluid. 4-9 pinnae pairs, 2.5 cm long, briefly stalked. Flyers, 10-15 pieces, 3-6 linear-oblong by 1,2-2 mm. Bloom is yellow, and in 2-6 axillary, heretic, and pubescent fascicles seen in globose heads (paddle of fiber). Bracteoles are 2 above the middle of the peduncle, widely oval, sharp, hairy. Bell-shaped, The length of Calyx is 1.25 mm. Bell-shape. 3 mm long. It is 3 mm long.

II. MATERIAL & METHOD

Collection and authentication of plants

Arabian Acacia (Lam.) The wild animals' stalk bark. It was gathered in Meerut It included the seeds and the flower after drying.

EXTRACTION

The production of 200 g of petroleum ether (40 to 600°C), chloroform, ethyl acetate, acetone, methanol and water is followed by Acacia Arabica extracted from plant powder with a broad variety of growing resolvents. Each extract was condensed in tiny amounts and desiccated through CaCl2. After drying, the weight and the % extractive values of the different portions were calculated.

<table>
<thead>
<tr>
<th>S. no.</th>
<th>Extract name</th>
<th>Color/consistency of Extract</th>
<th>Practical yield (in g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Total alcoholic</td>
<td>Dark brown/powder</td>
<td>47.42</td>
</tr>
<tr>
<td>2</td>
<td>Pet. ether</td>
<td>Yellow/sticky</td>
<td>01.80</td>
</tr>
<tr>
<td>3</td>
<td>Chloroform</td>
<td>Reddish brown/powder</td>
<td>04.47</td>
</tr>
<tr>
<td>4</td>
<td>Ethyl acetate</td>
<td>Dark reddish brown/powder</td>
<td>22.30</td>
</tr>
<tr>
<td>5</td>
<td>Acetone</td>
<td>Reddish brown/powder</td>
<td>16.13</td>
</tr>
<tr>
<td>6</td>
<td>Methanolic</td>
<td>Reddish brown/powder</td>
<td>10.26</td>
</tr>
<tr>
<td>7</td>
<td>Water</td>
<td>Brown/powder</td>
<td>03.19</td>
</tr>
</tbody>
</table>

PHYTOCHEMICAL TEST

There are two categories of phytochemicals: primary and secondary. Chlorophyll, sugar protein, and amino acids are the main components. A secondary feature includes terpenoids, saponins, flavonoids, phenolic chemicals, and alkaloids. A significant amount of study has carried out in connection to the phytochemical testing of species, geographical settings, extraction methods, and the employed solvents, which differ in biochemical variations. Full alcohol and all subsequent plant extracts were extensively analyzed phytochemically.
<table>
<thead>
<tr>
<th>S. no.</th>
<th>List</th>
<th>Test</th>
<th>Observation indicating presence of compound</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>[1] Test for carbohydrate</td>
<td></td>
</tr>
<tr>
<td>A)</td>
<td>Molish test: only a few drops of alcohol naphthol solution added to 2.3 ml of watery extract. Shake the concentrate and add it. Shake. H2SO4 from the edges of the test tube.</td>
<td>Violet color</td>
<td></td>
</tr>
<tr>
<td>B)</td>
<td>Pentose sugar test: in combination with HCl and the same solution volume. Phloroglucinol crystal heated and added</td>
<td>Red color appears</td>
<td></td>
</tr>
<tr>
<td>C)</td>
<td>Hexose sugar test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I)</td>
<td>Tollen galactose test: 2-3 ml concentration. Tollen galactose test. A 1.2 ml test solution used with HCl and 4 ml 0.5 percent phloroglucinol.</td>
<td>Yellow and red color appear</td>
<td></td>
</tr>
<tr>
<td>II)</td>
<td>Cobalt chloride test: 3 ml of mixed test solution, heated and cooled with 2 ml of cobalt chloride solution. A few NaOH drops have been added.</td>
<td>Solution appears Greenish blue color</td>
<td></td>
</tr>
<tr>
<td>D)</td>
<td>Non-reduction screen for polysaccharides (starch)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I)</td>
<td>Test iodine: 3 ml mixed solution and a few drops of iodine solution.</td>
<td>Blue color appears</td>
<td></td>
</tr>
<tr>
<td>II)</td>
<td>The starch tannic acid test: apply 20% tannic acid to the test solution</td>
<td>PPT</td>
<td></td>
</tr>
<tr>
<td></td>
<td>[2] Test for protein</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A)</td>
<td>Biuret test: applied to the 3 ml test solution 4% NaOH and just a small decline of 1% CUSO4 Settlement</td>
<td>Appears Violet color</td>
<td></td>
</tr>
<tr>
<td>B)</td>
<td>Millions of tests: 2ml solution combined with one million reagents</td>
<td>White ppt., wash the ppt, it turn brick red.</td>
<td></td>
</tr>
<tr>
<td>C)</td>
<td>Xanthoprotein test: 3 ml blended 1 ml concentration test solution. H2SO4 H2SO4</td>
<td>White ppt is formed</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Test for sulfur-containing proteins: 5 ml mixed test solution with 2 ml 40% NaOH and 2 drops 10% lead acetate solution and then boiled.</td>
<td>Solution turn black or brownish due to ppt formation</td>
<td></td>
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<tr>
<td>---</td>
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<td></td>
</tr>
<tr>
<td>3</td>
<td>Test for amino acid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Test for cysteine: A few decreases of 40 percent NaOH and 10 percent lead acetate and solution have been applied to 5 ml of the test solution.</td>
<td>Black ppt of lead sulphate is formed</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Test for steroid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Test for glycosides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A)</td>
<td>Salkowski reactions: add 2 ml of chloroform and 2 ml of concentration extract. Shake well, H2SO4.</td>
<td>A layer of chloroform appears red. The acid layer displays greenish fluorescence yellow.</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Test for anthroquinone glycosides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A)</td>
<td>Borntrager anthraquinone glycoside test: applied to dilute to 2ml extract, boiled and filtered, H2SO4. Add the same amount of benzene or chloroform to cooled filtrate and shake well.</td>
<td>Separate and bind ammonia to the organic solvent. The layer of ammonia turns pink and red.</td>
<td></td>
</tr>
<tr>
<td>B)</td>
<td>Changed Borntrager test for C-glycoside: add 5 percent FeCl3 and 5 ml of dilute HCl to 2 ml extract. Heated in boiling water for around 5 minutes, cooled, and add benzene or some organic solvent and shake well.</td>
<td>A separate organic solvent layer applies the same dilute ammonia level. Ammonia layer displays rosy red color.</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Test for flavonoids</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Evaluation of hepatoprotective activity of *Acacia arabica* bark extract

- Animal care and handling

The studies were performed on Swiss four-month albino mice of both sexes between 20 and 25 g. They have been supplied from Animal House, Pharmacy School, B.I.T. Meerut. The animals were acclimatised to the normal laboratory settings at the cross ventilated animal housing at 25±2°C of relative humidity of 44 –56% and light and dark cycles of 12:12 hours. The experiment was authorised by the Committee on Institutional Ethics in accordance with the rules of the CPCSEA (approval number. 1147/Ab/07/CPCSEA).

- Chemicals

The Local Medical Store, Meerut, purchased paracetamol pills (Paracip-500, Cipla), Isoniazids tablets (Solonex, 300mg, Macleods) as well as silymarin capsules (Limarin-140, International Serum). All other substances utilised in this investigation were analytical.

### Scheme for hepatoprotective activity

**Table 3:** Potent extracts of *A. arabica* at dose 100 mg/kg body weight, selected to evaluate hepatoprotective activity.
For the determination of hepatoprotective activity of selected extracts of *A. arabica* bark two animal models were used

**a) Paracetamol (PCM) induced hepatotoxicity animal model**

Paracetamol poisonous to the liver is heavily caused by N-acetyl-p-benzoquinineimine (NAPQI), one of the paracetamol's metabolites after metabolization is metabolized cytochrome P-450 (CYP450) mono-oxygenase. Several CYP-450 enzymes participating in the NAPQI bioactivation have identified. Usually, NAPQI is combined with glutathione (GSH) and eliminated in urine. GSH emphasized as responsible for the antioxidant protection of our bodies by scavenging the free radicals created in the liver during the metabolic processes to avoid subsequent harm to our cells.

**Isoniazid (INZ) induced hepatotoxicity animal model**

Tuberculosis is one of the deadliest illnesses of communication and spreads rapidly across the population. The specific mechanism of liver damage produced by this medicine is not understood. Isoniazid is acetylated and subsequently hydrolyzed, resulting in isonicotinic acid and monomethylhydrazine, activated by cytochrome P-450 as an unstable molecule.

**Induction of hepatotoxicity by paracetamol**

Paracetamol has produced for oral administration in a solution of 0.5% sodium carboxymethylcellulose (CMC). Previous dose-finding tests have shown that paracetamol 2g/kg selected as the toxicant dosage in this investigation.

**Preparation of Silymarin**

Silymarin was dissolved in normal saline and 100mg/kg per oral (p.o.) dose was selected as a standard.

A total of 25 Swiss albino mice utilized in the experiment. The Swiss albino mice split into five groups, with 5 individuals each.

**Group I:** Standard control Swiss albino mice got 1 ml/100 g of sodium methylcellulose sodium carboxy (CMC) using a 7-day intragastric tube.

**Group II:** Negative monitoring Paracetamol was administered by Swiss albino mice 2g/kg p.o. on the 6th day to induce hepatotoxicity

**Group III:** Swiss albino mice received 100 mg/kg silymarin, p.o. 7 days, paracetamol 2 g/kg, p.o. Six days.

**Group IV** Total alcoholic extract of *A. arabica* 100 mg/kg once daily for 7 days was taken from Swiss albino Mice and paracetamol 2 g/kg, p.o. on the sixth day.

6. **Group V** Swiss albino mice get *A. arabica* 100mg/kg methanol extract once daily for 7 days, and paracetamol 2g/kg, p.o. on 6th day.

**Sample collection**

At the end of the 7th day trial, blood was obtained by orbital punching and coagulated at room temperature for 30 minutes. At 3,000 revolutions per minute (rpm) the serum was separated at 30°C for 15 minutes. Swiss albino mice have been killed for liver histology via cervical dislocation.

**Induction of hepatotoxicity by isoniazid**

Isoniazid (INH) solution in sterile distilled water was produced. At a dosage of (100 mg/kg b. wt., i.p.) to the experimental animal, Swiss albino mice were treated with INH for 10 days.

**Experimental Design**

A total of 25 Swiss albino mice were utilized in the experiment. The Swiss albino mice separated into five groups, each of which comprised 5 individuals.

**Group I:** Standard control Swiss albino mice got 0.5 percent sodium CMC through an intragastric tube for ten days with 1ml/100 gm.

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Group II: Control negative Swiss albino mice got 100 mg/kg isoniazid, in particular for hepatotoxicity induction

Class III: Swiss albino mouse got 10 days of silymarin (100 mg/kg, p.o.) and ten days of isoniazid100 mg/kg, i.p.

Group IV Swiss albino mice got whole A. arabica 100 mg/kg alcohol extract once a day for 10 days and 100 mg/kg isoniazid, i.p. for 10 days.

Group V Swiss albino mouse has received A. arabica 100 mg/kg methanol extract once daily for 10 days and 100 mg/kg isoniazid, i.p. 10 days.

Collection of samples

At the end of the 10th-day trial, blood was obtained using orbital puncture and left to clot at room temperature for 30 minutes. The serum was separated for 15 minutes by centrifugation at 3000 rpm at 30°C. Swiss albino mice killed for liver histology via cervical dislocation.

<table>
<thead>
<tr>
<th>GROUP</th>
<th>SGOT/AST (U/L)</th>
<th>SGPT/ALT (U/L)</th>
<th>TOTAL BILURUBIN (mg/dl)</th>
<th>ALP (U/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (Normal Control)</td>
<td>71.28 ± 0.48</td>
<td>65.32 ± 2.23</td>
<td>1.48 ± 0.19</td>
<td>66.25 ± 1.42</td>
</tr>
<tr>
<td>Group II (-ve Control PCM, 2 g/kg, p.o.)</td>
<td>124.07 ± 3.76 a***</td>
<td>84.28 ± 2.82 a***</td>
<td>4.25 ± 0.28 a***</td>
<td>121.18 ± 2.22 a***</td>
</tr>
<tr>
<td>Group III (Std. Silymarin 100 mg/kg)</td>
<td>91.72 ± 4.58 a**,b***</td>
<td>52.42 ± 1.26 a**,b***</td>
<td>2.22 ± 0.26 a**,b***</td>
<td>80.35 ± 0.40 a**,b***</td>
</tr>
<tr>
<td>Group IV (Total alcoholic extract of A. arabica, 100 mg/kg)</td>
<td>103.21 ± 2.2 a***,b**,c*</td>
<td>86.48 ± 1.58 a***,c***</td>
<td>2.60 ± 0.72 a**,b**,c***</td>
<td>85.45 ± 2.76 a***,b***</td>
</tr>
</tbody>
</table>

Figure 1: Blood collection by orbital puncture

FIG: 4 Effect of different extracts of Acacia arabica on various biochemical parameters in paracetamol induced hepatic injury in Swiss albino mice
III. RESULTS

EXTRACTION

During plant powder extraction of Acacia Arabica, different solvents with increasing polarity sequentially extracted chloroform, ethyl acetate, acetone, methanol and water. Each extract was condensed and dried in tiny quantities and dried with desiccators over CaCl₂. After drying, the relevant sections and extractive values were calculated by %.

PHYTOCHEMICAL STUDIES

Bark A. Arabica has total alcoholic content of carbohydrates, glycosides, steroids, saponins, flavonoids, alkaloids, tannins and amino-acids. The extract of ethyl acetate has been positive for any of the aforementioned except steroids and alkaloids. Carbohydrates, saponins, proteins and amino acids have been discovered, and extracts of methanol, carbohydrates, glycooids, saponins, flavonoids and tannins have detected.

HEPATOPROTECTIVE OF PARACETAMOL

The control group of rats exhibited normal levels of all the SGOT (71.28 ± 0.48 u/l), SGPT (65.32 ± 2.23 u/l), total Bilirubin (1.48 ± 0.19 mg/dl), ALP enzymes (66.25 ± 1.42 u/l), based on examination of biochemical markers of paracetamol-induced hepatotoxicity. The levels of SGOT (124.07 ± 3.76 u/l), SGPT (84.28 ± 2.82 u/L) and total bilirubin (4.25 ± 0.28 mg/dl) and ALP (121.18 ± 2.22 u/l) have observed to be high about the normal group of treating animals after paracetamol in the negative control group. Methanol extract from A. Arabica at 100 mg/kg b dosage. Wt. Produce higher hepatoprotective action than the whole A alcoholic extract. Arabica at 100 mg/kg b dosage. Wt. Dose.

INDUCED HEPATOTOXICITY BY ISONIZID

The animal control groups exhibited normal level of all SGOT enzymes (73.24 ± 0.48 u/l), SGPT enzymes (64.73 ± 2,94 u/l), full bilirubins (1.34 ± 0.17 mg/dl), ALP enzymes (66.23 ± 1,48 u/l). Following isoniazid treatment of negative groups of controls, SGOT (95.66 ± 1.32 u /l), SGPT (92.97 ± 1.32 u/l), total bilirubin (3.15 ± 0.13 mg/dl), ALP (109.25 ± 2.40 u/l) were found to be higher than normal group of animals treated. The dose of A. arabica methanol extract is 100 mg/kg b.wt. More significantly hepatoprotective than the dose of 100 mg/kg b.wt. With the whole alcoholic extract, A. arabica.

FIG : 2 Paracetamol-induced hepatotoxicity histological slice of group I (control) has shown normal hepatocytes with a well-preserved cytoplasm, nucleus, and central vein. In these animals, there was no indication of inflammation, lipid changes, or necrosis.
FIG 3: Paracetamol-induced hepatotoxicity histological slice of group I (control) has shown normal hepatocytes with a well-preserved cytoplasm, nucleus, and central vein. In these animals, there was no indication of inflammation, lipid changes, or necrosis.

FIG 4: Histological section of paracetamol-induced hepatotoxicity of group III (silymarin) mice showed nearly normal hepatic lobule without necrosis in the centrical region. In the centrical area, just a few inflammatory cells detected.
IV. CONCLUSION

Descriptions Animals with paracetamol hepatotoxicity produced by the histological section of group IV (Total Arabica 100 mg/kg Alcoholic extract) exhibited a more significant decrease of the necrosed region and scarce inflammatory cell infiltration around the central veins.

Paracetamol induced hepatotoxicity animal model

Total alcoholic and methanol extracts of A. arabica (100 mg/kg b.w.) have investigated to control paracetamol-induced hepatotoxicity. The modification of SGOT, SGPT, ALP, total bilirubin, and histological alterations to the liver have evaluated for both treated and untreated groups. The control group has shown an average level with all enzymes of SGOT (72.23±0.48 u/l), SgPT (62.73±2.94 u/l), total bilirubin (1.36 ±0.17 mg/dl), ALP (65.23±1.46 u/l), indicating the standard cellular structure of your liver to animals receiving 1ml / 100 g sodium carboxymethylcellulose (CMC). After treatments with paracetamol, the level of enzymes was higher than regular treatment groups of animals. The compromised structural integrity of the liver has to a high amount of enzymes. The treatment group of animals with Silymarin (standard group) demonstrated a near-average level of enzymes following induction of paracetamol-induced liver damage compared to the treated animals of the regular group. These extracts were able to prevent liver tissue releases of these enzymes. The findings show that A. arabica methanol extract is hepatoprotective.

Isoniazide induced hepatotoxicity animal model

To evaluate hepatoprotection efficacy against isoniazid-induced hepatotoxicity, total alcoholic and methanolic extracts of A. arabica (100 mg/kg b. wt). Changes in the SGPT, SGOT, total bilirubin, and histopathological alterations of the biochemical markers have examined for both treated and untreated groups. Control group animals received an average level of SGOT (72.23±0.48 u/l), SGPT (62.73±2.94 u/l), total bilirubin (1.36±0.17 mg/dl), ALP (65.23±1.46 u/l) enzymes, showed that they have treated with 1ml/100gm of 0.5 percent of sodium carboxymethylcellulose (CMC) using an intrusion tube for 10 days. After treatments with isoniazid, the level of enzymes has been high compared to the usual treatment group of rats—the distortion of the structural integrity of the liver results in high levels of enzymes. Total alcoholic extracts from A. arabica significantly decreased the biochemical indicators, but methane extracts from A. arabica were SGOT (80.68±0.44 u/l; 73.46±1.45 u/l), SGPT (70.77±0.98 U/l) and SGPT (73.17±1.2 u/l), total bilirubin (2.74±0.21mg/dl; 2.16±0.12mg/dl) and ALP (72.63±3.48 u/l and 83.76±2.06 u/l).

Paracetamol and hepatotoxicity caused by isoniazid effect against Based on biochemical and histological investigation of plant extracts, A. arabica methanol extract, has substantial hepatoprotective.
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