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STUDIES ON THE EFFICACY OF HERBAL AND MEDICINAL USED FOR THE MANAGEMENT OF HEPATOTOXICITY

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Abstract

The liver is a key organ that helps the body break down and flush out foreign substances. Liver damage or dysfunction is a serious public health issue that poses difficulties for everyone involved, from doctors and nurses to drug manufacturers and regulators. There is a lot of research on the negative effects of toxic chemicals (including some antibiotics, chemo-therapeutic medicines, carbon tetrachloride (CCl4), thioacetamide (TAA), etc.) on the liver. Unfortunately, the synthetic medications now used to treat liver diseases in this context are themselves harmful to the liver. As a result, the usage of herbal medications has grown increasingly common. Treatment of liver problems using herbal remedies has a long history. There are a variety of herbal supplements you can buy with the help of cutting-edge scientific methodology; Aiming to aggregate data on promising phytochemicals from medicinal plants that have been studied in hepatotoxicity models; this review compiles findings from those studies.

Keywords: hepatoprotective, Alternative medicine, liver disease, herbal drugs, Phytoconstituents

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Introduction

The liver is the primary organ of the biliary system and plays a significant role in regulating many other bodily functions as well. Its role in the body's physiology is distinct. The liver's key roles are in the metabolism of nutrients, including glucose, lipids, and proteins; blood clotting; and immunomodulation. Liver illnesses impact one third of the population. There are about over 2000,000,000 people currently living with hepatotoxicity. The world's leading health issue is hepatotoxicity. The capacity of chemicals, medications, or other exposure to cause liver damage is known as hepatotoxicity (Rajesh and Latha, 2004). Tissue glutathione (GSH) depletion, a decrease in lipid peroxidation, and cellular necrosis are all hallmarks of liver injury. Alanine amino transaminase (ALT), Aspartate amino transaminase (AST), serum glutamic pyruvic transaminase (SGPT), serum glutamic oxaloacetic transaminase (SGOT), total bilirubin (TB), and total cholesterol are just some of the biochemical markers measured in the blood. Mild to severe hepatic problems might arise from a number of different causes. Some liver issues manifest as a direct result of people's unorthodox behavior. However, there are other liver toxicities that occur as a result of unavoidable factors. The individual must experience several systemic effects regardless of the cause of the poisoning. Difficulties, which push the protagonist to the verge of death. Therefore, it is essential for general well-being to take care of one's liver. A variety of liver diseases, such as alcoholic liver disease, cirrhosis, and hepatitis, can develop, however, due to the liver's constant exposure to environmental contaminants as well as the abuse of unhealthy drug habits, alcohol, prescription drugs, and over-the-counter drugs (Sharma *et al.*, 1991; Subramonium and Pushpangadan 1999). Liver illnesses are among the leading causes of death worldwide. It demonstrates a significant threat to global public health. Rather than using synthetic pharmaceuticals, people with liver problems often turn to plant-based remedies (Karan *et al.*, 1999; Chaterrjee, 2000). The use of herbalbased therapies for liver problems has a long history in India and has been widely pushed by major pharmaceutical companies. There is still no approved way to treat liver problems using herbal medications, despite the widespread acceptance of many of these remedies. Insufficient standardization of herbal medications, identification of active ingredients/principles, randomized controlled clinical trials (RCTs), and toxicological assessment are all problems that work against this goal (Dhiman and Chawla, 2005) Traditional medical systems from all over the world, including Ayurveda, Chinese medicine, European medicine, and others, have long relied on natural therapies for the treatment of liver problems (Thyagarajan *et al*., 2002). There are a lot of herbs and pills out there that claim to be hepatoprotective. Approximately 160 phytoconstituents extracted from 101 plants have been demonstrated to have liver-protective effects. It is estimated that there are 87 unique plant species used in 33 unique multi-plant ingredient compositions that are either patented or privately held in India (Handa *et al.*, 1986). Even with all the progress that has been made, there are still no effective and safe hepatoprotective medicines in the current therapeutic arsenal. Therefore, there has been a worldwide emphasis on discovering and creating plant-based hepatoprotective medications that are effective against a wide

range of liver illnesses. The purpose of this review is to compile information about hepatoprotective activity-tested medicinal plants' prospective phytochemicals and pharmacological properties (Bhattacharyya *et al*., 2005; Sagar *et al.* 2014)

***Acacia mellifera* (Vahl.), Fabaceae:** The hepatoprotective activity of *A. mellifera* leaves ethanolic extract and fractions were evaluated against DCFH- (dichlorofluorescein) and CCl4-induced hepatotoxicity on cultured liver cells and rats (Arbab *et al.,* 2015). When added to DCFH-poisoned cells, a crude extract of A. mellifera (AM) at a concentration of 100 g/ml stimulated hepatocyte proliferation by about 20%. The hexane and dichloromethane fractions were inactive, while the water and n-butanol fractions showed promise hepatoprotective effectiveness. After 3 weeks of oral administration of AM ethanolic extract at 250 and 500 mg/kg. b. wt in combination with CCl4 in liquid paraffin (1: 1, 1.25 ml/kg), intraperitoneal (IP) levels of alkaline phosphatase (ALP), bilirubin (Bil), cholesterol, triglyceride, and lipoprotein were significantly normalized. Liver congestion in the central veins, considerable hepatocyte necrosis, and fatty changes were observed following a dose of 250 mg/kg of AM, as determined by histopathology. Both AM and silymarin, at a dose of 500 mg/kg, maintained hepatocyte and central vein health in rats.

Through phytochemical research, we found that the fractions contained alkaloids, flavonoids, polyphenolic tannins, sterols, and saponins. Antioxidant activity of AM fractions was also evaluated using the 1,1-diphenyl-2-picrylhydrazyl (DPPH) radical. Antioxidant activity was highest in dichloromethane and hexane extracts, lowest in water-based extracts, and highest in ethyl acetate and n-butanol. The hepatoprotective benefits of the plant have been linked to phenolic compounds, flavonoids, and saponins (Ai *et al.,* 2013; Saboo *et al.,* 2013; Tran *et al.,* 2002; Wang *et al.,* 2010). Flavonoids' hepatoprotective effect is due to their ability to scavenge free radicals (Saboo *et al.,* 2013).

The effects of a hepatoprotective extract of *Adansonia digitata* L. fruit pulp on rats exposed to CCl4-induced hepatotoxicity were evaluated (100 and 200 mg/kg). Silimarin (at 25 mg/kg) was used as a benchmark. A. digitata showed dose-dependent hepatoprotective effects in CCl4-exposed rats. Serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and bilirubin were all lower compared to the CCl4 group.

Protecting against CCl4-induced liver damage and restoring biochemical values may be the antioxidant, anti-inflammatory, analgesic, immunostimulant, and antimicrobial activities of the fruit pulp of *A. digitata* L. and its content of triterpenoids (Ramadan *et al.,* 1994), - sitosterol, - amyrin palmitate or/and -amyrin, and ursolic acid.

Argemone mexicana L. was evaluated for its potential to prevent liver damage caused by CCl4 in rats using extracts made from both its water and methanolic parts. Hepatic injury was induced by injecting 3 ml/kg s.c. of CCl4 in olive oil (1:1 v/v). Oral doses of 100, 200, and 400 mg/kg/day of methanolic extracts and 400 mg/kg/day of aqueous extracts were given for 5 days. Administering CCl4 occurred on day 3. Serum ALT, AST, and ALP were all considerably reduced by the methanol extract at 100 mg/kg, showing hepatoprotective effect (P 0.05). The 100 mg/kg dose of methanol extract shown to repair and regenerate liver parenchyma in histopathology. Similar results were seen when silymarin (70 mg/kg orally) was compared to this method. The mechanism through which *A. mexicana* provides hepatoprotection is unknown at this time (Adam *et al.,* 2011).

Anogeissus leiocarpus (DC) Wall is a member of the Combretaceae family. The hepatoprotective properties of an ethanolic extract of the bark of *Anogeissus leiocarpus* were investigated in rats exposed to carbon tetrachloride (Ahmed *et al.,* 2015). The rats were administered the plant extract at a 0 hour, 12 hour, and 24 hour interval. After a single injection of 1.25 ml/kg CCl4, the first dose of test extracts was given 30 minutes later. The rats were sacrificed after 36 hours, and samples of their blood and livers were obtained for histopathology, as well as haematological and biochemical analyses. The bark of *A. leiocarpus* contains several different types of phytochemicals, including tannins, saponins, flavonoids, sterols, triterpenoids, and cumarins, just to name a few. Ethanolic extract of the plant at 200 mg/kg significantly (P 0.05) reduced liver biomarker levels (AST, ALT, and ALP) and kidney biomarker levels (urea, creatinine), and normalized haematological parameters in comparison to the gold standard drug silymarin histopathological.

Research on liver and kidney tissues confirmed the protective effects of the plant extract. The high concentration of flavonoids, tannins, sterols, and triterpenes in the extract may be responsible for its antioxidant activity and protective effects (Latha *et al.,* 2003; Victor and Grace, 2013 and Arbab 2014).

***B. aegyptiaca (L.)* (Balanitaceae):** After exposing rats to 0.2 ml/kg carbon tetrachloride (CCl4) for 10 days, researchers examined the effects of *Balanites aegyptiaca* bark extracts on the animals' livers. Extracts were administered orally at 250-500 mg/kg in conjunction with CCl4 for a total of 10 days. The AST, ALT, and ALP activities, as well as bilirubin levels, and mild hepatocyte lesions, were all significantly reduced in the plant extract group compared to the CCl4 group (Elhag 2001). According to phytochemical analysis (Ojo et al., 2015), aqueous extracts of *B. aegyptiaca* stem bark contain alkaloids, flavonoids, glycosides, phenols, saponins, and tannins. Flavonoids, phenolic chemicals, and saponins have been credited with antioxidant and hepatoprotective effects. Flavonoids and saponins from *B. aegyptiaca* may also stabilize reactive oxygen species by converting them to less reactive radicals through an oxidation process (Ojo *et al.,* 2015; Suky *et al.,* 2011).

***Cannabis sativa L.,* Cannabaceae**, was investigated for its potential to protect the liver from the damage caused by CCl4 in rats (Musa *et al.,* 2012). Daily oral doses of 1 and 0.5 ml/kg body weight of C. sativa L. oil were given to rats. Hepatotoxicity caused by 0.2 ml/kg subcutaneous injections of CCl4 in paraffin oil (1: 9 v/v) was reduced by concomitant oral treatment with 1 and 0.5 ml/kg of C. sativa oil. The decrease in serum levels of AST, ALT, ALP, and bilirubin demonstrates this. Liver changes were suppressed in the silymarin group, in contrast to the control rats. Carbon tetrachloride can cause damage to the liver, although the oil extracted from C. sativa plants has a hepatoprotective component (Musa *et al.,* 2012).

Capparaceae: Capparis decidua (Forsk.) *Capparis decidua* stem extracts (both aqueous and methanolic) were tested for their ability to protect against CCl4-induced hepatotoxicity in rats. Hepatotoxicity caused by CC14 in paraffin oil (1: 9 v/v) at a dose of 0.2 ml•kg1 was inhibited by concurrent oral administration of aqueous and methanolic extracts of *C. decidua* stems (200, 400 mg•kg1 b.wt.) for 10 days. Serum AST, ALT, ALP, and bilirubin levels were reduced. In addition, when both extracts were given to rats with CC14 for 10 days, the hepatotoxic effects of CC14 were mitigated. Results were comparable to those seen with the hepatoprotective drug considered the gold standard, silymarin. The higher activity of the aqueous extract of *C. decidua* over the methanolic extract may be due to the higher concentration of polar phytoconstituents in the former (Ali *et al.,* 2011; Ali *et al.,* 2009) Powdered plant material was subjected to preliminary phytochemical screening, wherein a variety of active compounds were identified. The study discovered antioxidants such flavonoids, cyanogenic glycosides, and triterpenes, which may be responsible for the hepatoprotective impact (Evans *et al.,* 2002; Al-yahya 1986; Pattanayak and Pryashree 2008; Satyanarayana *et al.,* 2008).

By adjusting the levels of superoxide dismutase and catalase, *C. decidua* was also found to reduce oxidative stress. Iron and vitamin C, two of the plant's abundant minerals and vitamins, may also play a role in the extract's hepatoprotective effects (Duhan *et al.,* 1992).

Combretaceae, Combretum as a genus: Researchers looked at whether or not a methanolic extract of Combretum hartmannianum leaves could mitigate the harmful effects of CC14 and paracetamol on the liver. Methanolic extract of C. hartmannianum (12.5, 25, 50 mg/kg) was given intraperitoneally (IP) one hour before CC14 (800 mg/kg IP) and paracetamol (1 g/kg P.O.). This research compared the effects of the hepatoprotective drug silymarin to the standard of care. C. hartmannianum leaf extract was found to be hepatoprotective against CC14-induced hepatotoxicity, as evidenced by an increase in serum total protein and albumin and a decrease in AST, ALT, and bilirubin. C. hartmannianum leveas, a methanolic extract of C. hartmannianum leaves, significantly decreased AST and ALT in rats when administered intraperitoneally (IP) one hour before oral administration of paracetamol to induce hepatotoxicity (Mohammed 2008). There were no cumarins, alkaloids, or triterpenoids discovered in the methanolic extract of C. hartmannianum leaves (Mohammed 2008), but there were flavonoids, tannins, saponins, and unsaturated sterols. Possible explanations for the plant's hepatoprotective effect include its high antioxidant activity in the DPPH free radical scavenging assay and its high flavonoid content (Hassan *et al.,* 2014).

Dobera glabra (Forsk.), Salvadoraceae: Dobera glabra leaf extracts, both aqueous and methanolic, were tested for their ability to protect the livers of rats exposed to CCl4 through a process known as hepatotoxicity. Injecting 0.2 ml•kg1 of CC14 in paraffin oil (1: 9 v/v) for 10 days. Leaf extracts were given orally at 200 and 400 mg•kg1 b.w. for 10 consecutive days. The plant extract groups had considerably greater levels of ALT, AST, and bilirubin than the CC14 group or the traditional medication silymarin. Necrotic lesions with a diffuse centrilobular pattern formed in hepatocytes following 10 days of treatment with both extracts and CC14(Ali, 2011). Alkaloids, flavonoids, tannins, sterols, saponins, cyanogenic glycosides, and cumarins are just some of the important components found in the powdered plant, as demonstrated by preliminary phytochernical screening (Ali, 2011).

Khaya senegalensis (Desr.), Meliaceae, had its hepatoprotective activity against CCl4-induced liver damage in rats evaluated using an aqueous extract of its bark. The bark extract was orally administered at 250 and 500 mg/kg on days 4 and 5, following a subcutaneous injection of CCl4 (3 ml/kg body weight/rat) on day 3. Regular dosing of silymarin (at 50 mg/kg) was also performed. Liver tissue histopathology results were consistent with a hepatoprotective effect (Ali, 2011). In comparison to the silymarin group, the AST, ALT, ALP, and bilirubin levels were significantly higher in the extract group. Because of its historical usage in treating jaundice, the hepatoprotective benefits of a methanolic extract of the bark of the Sudanese shrub *Khaya senegalensis* have been investigated. To test the hepatoprotective properties, rats were exposed to CCl4 and paracetamol poisoning. The extracts were injected intraperitoneally an hour before the administration of CCl4 (800 mg/kg IP) and paracetamol (1 and 2 g/kg P.O.). Well-known hepatoprotective medication silymarin was used as a comparison in this study.

K. as indicated by a significant decrease in alanine aminotransferase (ALT), aspartate aminotransferase (ALP), and alanine aminotransferase (AAT), s. senegalensis bark methanolic extract protected the liver from CCl4- and paracetamol-induced damage. Furthermore, there was no decrease in ALT or AST activity, showing that neither the dichloromethane nor the petroleum ether extracts had any hepatoprotective effect against paracetamol-induced hepatotoxicity. Although paracetamol is damaging to the liver, studies have found that extracts preserved in chloroform and ethyl acetate considerably reduce this toxicity. K. had some mild histopathological lesions. Groups treated with P. senegalensis bark extract compared to paracetamol groups revealed a preventative effect (Elagib *et al.,* 2014; Elagib *et al.,* 2014).

An aqueous extract of Khaya senegalensis bark was tested for its hepatoprotective effects against CCl4-induced liver injury in rats (Ali *et al.,* 2011). K doses of 250 and 500 mg/kg were given orally. The senegalensis bark treatment lasted for five days. Total protein, albumin, AST, ALT, and bilirubin levels were all lower in the extract-treated groups compared to the CCl4 group. Liver tissue histology results compared to those of a control group given silymarin offered strong backing for these conclusions. According to these findings, the hepatoprotective properties of an aqueous extract of K. tree with the senegalensis bark (Ali *et al.,* 2011). K. The hepatoprotective effects of P. senegalensis bark ethanolic extract against CCl4 in rats (Ahmed, 2015) were investigated. The extract was given orally at a dose of 200 mg/kg on three separate occasions (0, 12, and 24 hours). After the initial vehicle injection, 1.25 ml/kg CCl4 diluted in liquid paraffin (1: 1) was injected subcutaneously. Blood samples were obtained 36 hours after infection and analyzed biochemically and haematologically. Serum levels of ALT, AST, and ALP were all lower in the extract group compared to the CCl4 group, as were total bilirubin and direct bilirubin concentrations. The suppression of CCl4-induced liver enlargement further supported the hepatoprotective action of

the plant extract. Initial phytochemical screening of the ground plant material revealed the presence of tannins, sterols, saponins, cumarins, and triterpenes. The plant material lacked any anthraquinone, flavonoid, or alkaloid components. Minimal antioxidant activity is demonstrated by the DPPH experiment (Ahmed, 2015).

Kigelia africana (Lam.), a member of the Bignoniaceae family, was studied for its capacity to protect the livers of male Wistar rats from CCl4-induced damage using both aqueous and methanolic extracts. Subcutaneous injections of CCl4 (1:1 dilution with olive oil) at a dose of 3 ml/kg were performed on day 3 of the trial. Oral silymarin (50 mg/kg) was taken for five days. The methanolic extract doses were 100, 200, and 400 mg/kg/day, while the aqueous extract dose was 400 mg/kg/day for 5 days. Both aqueous and methanol extracts of the plant seeds were revealed to have harmful effects, as evidenced by changes in haematological indices (Hb, WBCs MCH, MCHC, and granulocytes) and AST, ALT, and ALP activities. The kidneys and liver also showed histopathological abnormalities (Shama *et al.,* 2013).

Hepatoprotective properties of the methanolic extract of Lawsonia inermis leaves against CCl4-induced hepatotoxicity in rats were investigated. The L. vegetation's leaves Participants were administered a methanolic extract of inermis that had been prepared through maceration.

Two doses of oral 100 mg/kg and 200 mg/kg are available. In this analysis, the hepatoprotective drug silymarin (25 mg/kg) served as the gold standard. Histological liver sections were enhanced, and serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (ALP), and bilirubin were decreased in CCl4-only treated mice. Antioxidant properties of this plant material may have a hepatoprotective effect, as indicated by the &e findings (Mohamed et al., 2016). By neutralizing potentially dangerous free radicals such superoxide anions, peroxynitrite, peroxyl, and hydroxyl, the flavonoid content of the plant extract has been shown to have hepatoprotective effect (Sanni *et al.,* 2010).

Lepidium sativum **(L.)** (Brassicaceae) has protective properties. A methanolic extract of the seeds was tested for its ability to prevent CCl4-induced liver damage in rats. The extracts were administered together with 0.2 ml/kg of CCl4 that had been dissolved in paraffin oil (1: 9 v/v) for 10 days. Serum levels of a variety of enzymes were significantly reduced in L-treated rats. Extract from the sativum plant. Rats given CCl4 in the liver revealed significant fatty changes, although they were considerably reduced in animals given plants. The hepatoprotective properties of the plant may stem from an unproven mechanism involving an inhibition of lipid peroxidation in the liver. L has a wide variety of bioactive substances. sativum is able to shield the liver since it impedes the production of free radicals. Flavonoids, triterpenes, and tannins are all examples of antioxidant chemicals that act as free radical generation inhibitors (Abuelgasim *et al.,* 2008).

Rats were subjected to CCl4-induced liver injury, and the hepatoprotective effects of an aqueous extract of *Moringa oleifera* leaves were investigated. Daily administration of 0.2 mg•kg1 CCl4 in paraffin oil (1: 9 v/v) caused hepatocellular damage in rats for 10 days. M. 400 mg•kg1 oleifera extract orally CCl4 was given at the same time. After 10 days of treatment with the aqueous extract with CCl4, serum levels of alanine aminotransferase (ALT), alanine aminopeptidase (ALP), and bilirubin were all reduced. Liver fatty change was also reduced in the intoxicated control rats, especially at the 200 mg•kg1 dose. Degenerative modifications were observed in the precentral areas of the livers of M-treated rats, including cytoplasmic rarefication and acidophilic cytoplasm with pycknotic nuclei. 400 milligrams per kilogram of Olea oleifera leaf water extract. According to the results of this investigation, an aqueous M. even lesser dosages, oleifera leaves may offer substantial protection against CCl4-induced liver injury in mice (Ali *et al.,* 2010) It was also discovered in the preliminary phytochemical research (Ali *et al.,* 2010) that the plant included alkaloids, saponins, flavonoids, tannins, sterols, glycosides, and cumarins. The liver benefits from phytoconstituents such flavonoids, triterpenoids, and sterols that have antioxidant properties. Because of the presence of these substances in M. possible protective effect of Olea oleifera (Gupta *et al.,* 2004; Manjunatha *et al.,* 2008) against CCl4-induced liver injury in rats. Several studies have corroborated the fact that M. oleifera is a good source of antioxidants due to its high levels of total phenolics, vitamin A, and vitamin E (Diallo *et al.,* 2009; Anwar *et al.,* 2007).

Extract (NSSE) of black seed (*Nigella sativa* L., Ranunculaceae) and black cumin (*Nigella sativa* var. nigella) was tested for its ability to prevent acetaminophen (APAP)-induced hepatotoxicity in TIB-73 cells and rats. TIB-73 cells were exposed to 10 mmol/L of APAP and then tested for NSSE's protective effects at concentrations of 25, 50, 75, and 100 mg/mL. For the in vivo experiment, 30 rats were split into 5 groups: vehicle, APAP (800 mg/kg body weight single IP injection) as a hepatotoxic control, APAP and NS pretreated (2 weeks) (100 mg/kg APAP + NSSE, 300 mg/kg APAP + NSSE, and 900 mg/kg APAP + NSSE), and APAP alone. TIB-73

Cell viability was greatly decreased (49.0 1.9%) after treatment with 10 mmol/LAPAP, and reactive oxygen species production was significantly increased. Cotreatment with NSSE at doses of 25, 50, 75, and 100 mg/mL dramatically enhanced cell viability and decreased generation of reactive oxygen species. When NSSE was administered prior to APAP administration in vivo, blood lactate, pH, anionic gap, and ion (HCO3, Mg2+, and K+) levels returned to near-normal values. The NSSE has been demonstrated to reduce hepatic lipid peroxidation (malondialdehyde) and superoxide dismutase activity in addition to reducing blood ALT, AST, and ALP levels.

All groups, excluding APAP, showed improved hepatic histology (Adam *et al.,* 2016). N. benefits of phosphite's healing properties. sativa seed extract's capacity to prevent oxidative stress, lipid peroxidation, and reactive oxygen species (ROS) in rats may explain why it was particularly effective in preventing APAP-induced hepatotoxicity and metabolic abnormalities. N. Extract of nivalis in water. Seeds of *Cannabis sativa* for their potential to shield the livers of CCl4-exposed rats. Both 250 and 500 mg/kg of plant extract were given orally over the course of 5 days. On day 3, CCl4 (3 ml/kg) was injected subcutaneously. Comparative reference was the hepatoprotective drug silymarin (50 mg/kg). Scarification of the rats occurred after 5 days. Treatment with extract (250 and 500 mg/kg) significantly decreased serum alanine aminotransferase (ALT), alanine aminopeptidase (ALP), and total protein (TP) levels in comparison to CCl4 treatment. The biochemical results were

verified by histopathological investigation of rat liver slices (Ali, 2011). On the other hand, N. Cannabis sativa seed methanol extract was tested for its ability to reverse CCl4-induced liver damage in rats. CCl4 (0.2 ml•kg1 diluted in liquid paraffin 1:9) and iodine intraperitoneal (IP) injections

N. nivalis methanolic extract. The dosages of sativa used were 250 and 500 mg•kg1. mixed sativa and indica

Dimethylsulfoxide- The rats were scarified after 10 days. The levels of ALT, AST, and ALP were significantly elevated in CCl4- and N. Those in the N-treated groups. sativa had a lower increase than the controls. Methanolic extract of the sativa plant. On histology, the livers of the CCl4 group showed severe centrilobular vacuolation and congestion, but the livers of the 250 mg•kg1 and 500 mg•kg1 groups did not. b. weight-wise, the changes were very somewhat noticeable (Abuelgasim *et al.,* 2008).

Ocimum basilicum L., Lamiaceae has been shown to have hepatoprotective effects. Whole-plant ethanol extract's efficacy was investigated. Oral administration of an extract from We administered a hepatoprotective dosage of 200 mg/kg 0 hours before CCl4 injection, and then again 12 and 24 hours later. Mice and rats received an A+. basilicum extract at a time zero, 12 hours after the first dose, and 24 hours after the first dose, with a single injection of 1.25 ml/kg CCl4 given 30 minutes prior to the first administration of test extracts. A blood sample was collected after 36 hours for Histopathology, hematology, and biochemistry were all performed on liver samples taken from the rats prior to their deaths. *O. Basilicum* ethanol extract at 200 mg/kg significantly (P 0.05) attenuated the elevations in serum AST, ALT, and ALP caused by CCl4. This result was on par with that achieved by the gold standard drug silymarin. Haematological indicators and the silymarin group were found to share similarities. These biochemical findings were supported by histopathological examination of the liver, demonstrating the plant extract's protective impact on the organ. There was evidence for *O. Basilicum* ethanolic extract has hepatoprotective potential against CCl4-induced hepatotoxicity in rats (Ahmed *et al.,* 2015).

Phytoconstituent extraction from onion (O. The total basilicum plant ethanolic extract contained flavonoids, alkaloids, tannins, saponins, triterpenes, sterols, and cumarins. Superoxide radical and nitric oxide radical scavenging abilities (Meera *et al.,* 2009; Mushtaq & Ahmad, 2013; Marzouk *et al.,* 2011) and a high concentration of flavonoids, saponin, tannins, sterols, and triterpenes suggest that the extract has an antioxidant quality that adds to its protective effect.

Albino rats were used to examine pollen grain powder extracted from the Sudanese dates palm (*Phoenix dactylifera* Linn., Palmae) for its hepatoprotective properties. Hepatotoxicity was induced using a subcutaneous injection of CCl4 (3 ml/kg diluted in olive oil 1: 1) on day 3. Date palm pollen grains at 250 and 500 mg/kg were given orally over the course of five days. As a comparison, silymarin (50 mg/kg orally, once day, for 5 days) was utilized as a control medicine. Hepatoprotective impact was established by significant (P 0.05) reductions in alanine aminotransferase (ALT), aspartate aminotransferase (ALP), and alanine aminoketone (ALP) levels in the treatment groups compared to the CCl4 group. Centrilobular necrosis with moderate congestion was confirmed histopathologically, corroborating the findings. The mechanism by which the extract of date palm fruit exerts its hepatoprotective action is unknown. *P. dactylifera* L. contains a compound called -sitosterol, which may be to blame. This may help explain why hepatotoxicity hasn't been observed (El-mougy *et al.,* 1991). *P. dactylifera* L. contains flavonoids. Possible cancer-preventing properties could be partially explained by the fact that it inhibits cytochrome P-450 aromatase (Kowalska *et al.,* 1990). The high vitamin C content of date palm pollen grains is hypothesized to be responsible for their hepatoprotective properties (Abuowf & Abuowf, 2009).

Raphanus sativus *L.* is a member of the Cruciferae family of plants. Water and methanolic extracts were tested for their potential to preserve the livers of rats exposed to carbon tetrachloride. Both 200 and 400 mg•kg1 of CCl4 methanolic and aqueous extracts (0.2 ml/kg in paraffin oil 1: 9 v/v) were administered IP. The animals were allowed to mature for 10 days before being slaughtered. Biochemical indicators of CC14-induced hepatotoxicity, such as serum alanine aminotransferase (ALT), alanine aminopeptidase (ALP), and bilirubin concentrations, and histological changes, were all reduced in those who consumed the plant. Phytochemistry (Mohammed et al., 2008) showed that the plant samples included triterpenes, alkaloids, flavanoids, tannins, saponin, and cournarin, but lacked cyanogenic glycosides and anthraquinone glycosides.

Solanum nigrum L., Solanaceae, was analyzed for its hepatoprotective effects in rats given 0.2 ml/kg CCl4 intraperitoneally over the course of 10 days. For multiple days, every day. *S. Nigrum* extracts between 250 and 500 mg/kg were orally administered to individuals for 10 days. Hepatoprotective effects of the extracts against CCl4-induced liver injury in rats were demonstrated by a reduction in blood AST, ALT, and ALP activity and bilirubin levels, as well as minimal histological abnormalities. Water extract may be better at preserving the liver than methanolic one since it contains more polar phytoconstituents (El-hag & El-Badwai, 2011).

In albino rats with carbon tetrachloride (CCl4)-induced liver injury, ethanolic and ethyl acetate extracts of stem bark were investigated for their hepatoprotective activity. Ten days of therapy with 0.2 ml/kg CCl4 led to hepatotoxicity. The same as the letter S. Doses of 200 and 400 mg/kg of Setigera stem bark were given orally. &e extracts significantly lowered serum levels of ALT, AST, ALP, total protein, albumin, and bilirubin, showing a substantial protective effect. The hepatoprotective effect of the traditional medication Silymarin was compared to the CCl4-induced liver abnormalities in rats, and it was found that the plant extracts effectively masked the CCl4-induced alterations. However, plant extracts prepared from ethanol and ethyl acetate were able to reduce the severity of necrotic lesions in the liver caused by CCl4 (Abdel Rahman, 2016).

Tamarindus indica (L., Caesalpinaceae) fruit pulp was extracted using ethanol and examined for its potential to protect against CCl4-induced liver damage. The T. alcohol-soaked rex root. For 5 days, subjects were administered an indica dosage of 150 mg/kg. We used silymarin (50 mg/kg) as the drug standard. After 5 days of oral saline administration, on days 2 and 3, subcutaneous administration of 0.2 ml/kg of CCl4 diluted (1: 9) in liquid paraffin was

performed. The results led researchers to conclude that a T. The livers of the indica group displayed a better histological image compared to the livers of the CCl4 group, showing that the plant had successfully alleviated the damage induced by CCl4. From what we can tell, T. The ethanolic extract of indica has been shown to have hepatoprotective effects (El-Badwi *et al.*, 2013). The T. reesei ethanol extract is a. Fruit pulp has low levels of flavonoids and saponin and moderate levels of tannins and alkaloids. The membrane-stabilizing characteristics of indica (Daniyan & Muhammad, 2008) suggest its probable inclusion.

Conclusion

This study reveals the importance of specific medicinal plants in avoiding chemical-induced liver damage by analyzing their effects on in vivo and in vitro models. Herbal medications play a significant role in basic health care because conventional pharmaceuticals used to treat liver disorders are both costly and ineffective. Ninety percent of the population relies on medicinal plants to alleviate symptoms of illness (UNIDO, 1996).The medicinal effects of these plants may be due, in part, to their phytoconstituents, antioxidants, and anti-inflammatory characteristics. More research on these plants is needed to establish efficacy, safety, and the precise mechanism of action before they can be considered a moral alternative in the treatment of liver disorders. Cutting-edge methods and technology are needed for identification, isolation, and purification of the active componen ts of hepatoprotective plants so that their efficacy and safety can be determined and controlled clinical studies may be conducted.

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