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Hepatoprotective Activity: A Review

Narendra Kumawat[1], Dr. Manmeet Singh[1], Dr. Azharruddin [1], Dr. Vidhi Jain[2]

[1]Department of Pharmacy, Sunrise University, Alwar, Rajasthan

[2]SVP College of Pharmacy, Hatta, Maharastra

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Corresponding author: Narendra Kumawat

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Abstract:

The liver is a significant and essential organ in the human body, responsible for extensive metabolism and excretion. It plays a crucial role in maintaining, optimising, and regulating the body's homeostasis. It plays a crucial role in nearly all metabolic processes related to development, immune response, nutrition absorption, energy production, and reproduction. The primary roles of the liver include the metabolism of carbohydrates, proteins, and fats, detoxification, bile secretion, and vitamin storage. Therefore, ensuring the proper functioning of the liver is an essential determinant of one's overall health and well-being. However, prolonged and diverse exposure to environmental toxins, such as CCl4, drug abuse, alcohol consumption, infections, and autoimmune disorders, along with the use of prescribed antibiotics, chemotherapeutic agents, and over-the-counter drugs, can ultimately result in different liver conditions, including hepatitis, cirrhosis, and alcoholic liver disease. Hepatoprotective medications can effectively treat these diseases. Over the past years, researchers have built both in vitro and in vivo liver models to explore the effects of hepatoprotective drugs. This system assesses the efficacy of test medications in preventing or treating liver injury caused by different hepatotoxins in laboratory animals..

INTRODUCTION

The Greek phrase for liver is hepar, hence medical terminology associated with the liver frequently begin with hepato or hepatic. The liver is a crucial organ responsible for metabolism, secretion, and storage. It is often referred to as the "great chemical factory" of the body because it plays a vital role in regulating, synthesising, storing, and secreting various proteins, nutrients, and chemicals. Additionally, the liver is responsible for purifying and eliminating toxins and unnecessary substances from the body. Bile, produced by the liver, has a significant part in the process of digestion, among other functions. The incidence of liver poisoning has lately risen due to increased exposure to environmental hepatitis, which can be caused by viral, toxic, or deficiency-related factors.

a) Hepatic failure - Either acute or chronic.

b) Liver illnesses caused by reduced metabolic function.

c) Frequently, problems related to the metabolism of fat (liposis) and bilirubin (jaundice) are commonly observed.

d) Disorders related to the metabolism of fat: Fatty Liver

e) Disorders related to the metabolism of bilirubin include jaundice, which can have varied kinds depending on the underlying mechanisms and causes.

f) Hemolytic or pre-hepatic jaundice Substances such as poisons, insecticides, medicines, and chemotherapeutics are used often. Obstructive jaundice, specifically post-hepatic or cholestatic jaundice, is consistently linked to liver injury. This type of jaundice, known as hepatogenous or hepatic jaundice, is characterised by cholestasis.

Cellular necrosis, elevation in tissue lipid peroxidation, and reduction in tissue glutathione (GSH) levels. Furthermore, the levels of many biochemical markers such as serum glutamate oxaloacetate transaminase (SGOT/AST), serum glutamate pyruvate transaminase (SGPT/ALT), triglycerides, cholesterol, bilirubin, and alkaline phosphatase are increased. There are three causes that result in unconjugated hyperbilirubinemia:

1. Hereditary jaundice
2. Pure cholestasis Gilbert's syndrome, Dubin-Johnson syndrome, Crigler-Najjar syndrome, and Rotor's syndrome are examples of inherited jaundice conditions.

The following are some of the most often observed liver conditions.

Necrosis and cirrhosis are medical conditions.

Chemical or drug-induced hepatotoxicity typically manifests as hepatitis, jaundice, or carcinogenesis.

Hepatotoxicity

a) Hepatic failure - Either acute or chronic.
b) Liver illnesses caused by reduced metabolic function.
c) Frequently, problems related to the metabolism of fat (liposis) and bilirubin (jaundice) are commonly observed.
d) Disorders related to the metabolism of fat: Fatty Liver
e) Disorders related to the metabolism of bilirubin include jaundice, which can have varied kinds depending on the underlying mechanisms and causes.
f) Hemolytic or pre-hepatic jaundice Substances such as poisons, insecticides, medicines, and chemotherapeutics are used often. Obstructive jaundice, specifically post-hepatic or cholestatic jaundice, is consistently linked to liver injury. This type of jaundice, known as hepatogenous or hepatic jaundice, is characterised by cholestasis
g) Cellular necrosis, elevation in tissue lipid peroxidation, and reduction in tissue glutathione (GSH) levels. Furthermore, the levels of many biochemical markers such as serum glutamate oxaloacetate transaminase (SGOT/AST), serum glutamate pyruvate transaminase (SGPT/ALT), triglycerides, cholesterol, bilirubin, and alkaline phosphatase are increased. There are three causes that result in unconjugated hyperbilirubinemia:

1. Hereditary jaundice
2. Pure cholestasis Gilbert's syndrome, Dubin-Johnson syndrome, Crigler-Najjar syndrome, and Rotor's syndrome are examples of inherited jaundice conditions.
3. The following are some of the most often observed liver conditions.
4. Necrosis and cirrhosis are medical conditions.
5. Chemical or drug-induced hepatotoxicity typically manifests as hepatitis, jaundice, or carcinogenesis

   A hepatotoxin is a chemical compound that causes harm to the liver. Drug and chemical-induced hepatotoxicity may result in liver damage.
6. Can accurately replicate any type of liver illness found in nature.

The study investigated the hepatoprotective impact against chemically and drug-induced liver damage in rats, including alcohol, carbon tetrachloride, galactosamine, paracetamol, isoniazid, rifampicin, antibiotics, peroxidized oil, and aflatoxin. The severity of liver damage is significantly heightened if the medication is continued after symptoms have appeared. Arsenic, phosphorus, copper, and iron are some of the inorganic substances that can cause

hepatotoxicity. The organic agents consist of specific plant poisons, such as pyrrolizidine alkaloids, myotoxins, and bacterial toxins, which occur naturally. Hepatotoxic substances, such as carbon tetrachloride (CCl4), ethanol, and acetaminophen, can induce liver damage. This damage is characterised by different levels of degeneration and death of liver cells, which can occur through either apoptosis or necrosis. The production of reactive intermediate metabolites from the breakdown of hepatotoxins and the presence of reactive oxygen species (ROS) during the inflammatory response contribute to various pathological processes that result in cell death. These processes include the formation of covalent bonds, disruption of calcium balance in the cytosol, depletion of glutathione (GSH), initiation of mitochondrial permeability transition (MPT), and associated lipid peroxidation. The hepatic metabolism of hepatotoxins through certain cytochrome P-450 enzyme subtypes is a crucial process in the development of toxicity. Consequently, the use of enzyme inhibitors has been demonstrated to reduce the extent of liver damage associated with hepatotoxin exposure. Furthermore, there is significant evidence indicating that MPT plays a role in hepatocellular injury linked with ROS. Recent discoveries present a potential treatment strategy to reduce cell damage by preventing the initiation of MPT. Therefore, oxidative stress and the breakdown of lipids are essential factors that contribute to liver damage caused by hepatotoxins. Aside from providing targeted treatment for a specific hepatotoxin, the overall approach to preventing and treating damage involves decreasing the production of reactive metabolites of the hepatotoxins, utilising anti-oxidative agents, and selectively administering therapeutics to Kupffer cells or hepatocytes to address ongoing processes that contribute to a secondary phase of the injury.

classification of hepatotoxins

The two types of idiosyncrasy are intrinsic and host idiosyncrasy.

Intrinsic

These substances are hepatotoxins that can be predicted. These persons and experimental animals are identified by their high frequency of liver damage. There is a consistent period of time between being exposed to a certain substance and the occurrence of liver damage, and the severity of the damage seems to be related to the amount of exposure.

There exist two categories of intrinsic hepatotoxins:

Direct hapatotoxins

The term "metabolic products" may be used to describe substances that cause direct harm to hepatocytes and their organelles, particularly the endoplasmic reticulum. CCl4, the prototype, induces peroxidation of the membrane lipids and other molecules, resulting in membrane deterioration.

Indirect hepatotoxins

These substances are anti-metabolites and similar compounds that cause liver damage by disrupting specific metabolic pathways or activities. The structural damage caused by indirect hepatotoxins seems to be a result of metabolic dysfunction. In cases where direct hepatotoxins are involved, the metabolic disturbance is a result of the structural damage. Indirect hepatotoxins can cause hepatic damage primarily by cytotoxic injury, which disrupts metabolic pathways and processes necessary for the integrity of liver tissue. This injury can manifest as either steatiosis (accumulation of fat in the liver) or necrosis (cell death). Alternatively, the damage may primarily involve cholestasis, which specifically affects the production of bile.

Host idiosyncrasy

It comprises agents that do not consistently cause liver damage, but only affect a limited number of individuals who are exposed to them. Autoantibodies targeting normal cellular components are found in multiple cases. The

harm does not seem to be dependent on the dosage and cannot be replicated in experimental animals. It occurs after a random period of time with no consistent pattern.

Evaluation of hepatoprotective activity

Various chemical agents and medications with specialised effects on the liver are employed as hepatotoxins in laboratory animals to replicate optimal pathological states. The assessment of hepatoprotective function can be conveniently conducted by employing various model systems of liver damage in experimental animals. In every experimental model, liver damage conditions are induced and an effort is made to mitigate this toxicity by administering the substance/preparation being tested. The extent of the protective effect can be quantified by assessing the enzyme activity and survival rate, which can be confirmed through histological examination. The approaches that can be used are in vivo, ex vivo, and in vitro methods. These procedures are employed to investigate the protective or therapeutic properties of any substance being examined. For the purpose of evaluating hepatoprotective activity, the test substance and the hepatotoxin are supplied concurrently. However, in the case of antihepatotoxic or curative activity, the test substance is typically administered after the hepatotoxicity has been induced.

In vitro methods

Hepatocytes are typically obtained with the use of an in-situ, two-step recirculating collagenase perfusion process. Subsequently, these are placed in small receptacles and subjected to test samples and harmful substances. Following a designated duration, the level of toxicity or protection is evaluated by viability tests and the measurement of enzyme levels such as GOT and GPT. Various hepatoprotective screening models have been developed by utilising primary culture hepatocytes and applying hepatotoxins such as CCl4, galactosamine, thioacetamide, ethanol, and paracetamol (PCML). These approaches offer several advantages compared to in vivo methods, including the capacity to process multiple samples simultaneously, low cost and compact size, minimal variation, and consistent reproducibility of results. One significant drawback is that it may occasionally fail to accurately represent the occurrences in animals.

Ex vivo models

In this model, hepatocytes are separated after the completion of a preselected in vivo test regimen, and the proportion of viable cells and biochemical parameters are measured as liver function tests. These methods exhibit a higher degree of correlation with clinical models compared to in vitro or in vivo methods

Table 1: List of hepatoprotective activity having medicinal plants

Botanical name	Family	Plant parts used	Screening methods
Acacia catechu	Leguminosae	Powdered pale catechu	Carbontetra chloride induced
Acacia confuse	Leguminosae	Bark	Carbon tetra chloride induced
Aegle marmelos Correa	Rutaceae	Leaves	Paracetamol Induced
Aerva lanata	Amaranthaceae	Coarce powder	Plant Paracetamol Induced
Alchornea cordifolia	Euphorbiaceae	Leaves	Paracetamol Induced
Alocasia indica Linn	Araceae	Leaves	Paracetamol Induced
Aloe barbadensis	Liliaceae	Dried aerial parts	Carbontetra chloride induced
Amaranthus spinosus	Amaranthaceae	Whole plant	Carbontetra chloride induced

Amaranthus caudatus Linn	Amaranthaceae	Whole plant	Carbontetrachloride Induced
Anisochilus carnosus Linn	Lamiaceae	Stems	Carbontetrachloride Induced
Apium graveolens	Apiaceae	Seeds	Paracetamol and thioacetamide induced
Arachiodes exilis	Dryppteridaceae		Rhizomes Carbontetra chloride induced
Argemone mexicana	Solanaceae	Plant material	Carbontetra chloride Induced
Asparagus racemosus Linn	Asparagaceae		Roots Paracetamol induced
Azadirachta indica	Meliaceae	Leaf	Paracetamol Induced
Azitetracantha	Salvadoraceae		Leaves Paracetamol induced
Baliospermum montanum	Euphorbiaceae		Roots Paracetamol induced
Boerhaavia diffusa	Nyctaginaceae		Roots Thioacetamide induced
Bupleurum kaoi	Umbelliferar	Dried roots	Carbontetra chloride induced
Byrsocarpus coccineus	Connaraceae	Leaf	Carbontetra chloride induced
Bixa orellana	Bixxaceae	Plant material	Carbontetra chloride induced
Cajanus cajan Linn	Leguminosae	Pigeon pea leaf	D-galactosamine induced
Cajanus scarabaeoide	Fabaeceae	Whole plant	Paracetamol induced
Carissa carindas Linn	Apocyanaceae		Root Carbontetrachloride Induced
Carum copticum	Apiaceae	Seed	Carbontetra chloride,paracetamol induced
Calotropis procera	Asclepediaceae		Root bark Carbontetrachloride Induced
Cassia fistula	Leguminosae	Leaf	Carbontetrachloride Induced
Cassia tora	Caesalpiniaceae		Leaves Carbontetra chloride induced
Cassia Occidentalis	Caesalpiniaceae		Leaves Paracetamol and Ethyl alcohol induced
Chamomile capitula	Asteraceae	Fresh natural mature capitula	Paracetamol induced
Clerodendrum inerme	Verbenaceae	Leaves	Carbontetra chloride induced
Clitoria ternatea Linn	Fabaceae	Leaves	Paracetamol induced
Cleome viscose Linn	Capparidaceae	Leaf powder	Carbon tetra chloride induced
Cochlospermum planchoni	Coclospermaae	Rhizomes	Carbontetra chloride induced
Cichorium intybus	Asteraceae	Leaves	Thioacetamide induced
Cordia Macleodii	Boraginaceae	Leaves	Carbontetra chloride induced
Cuscuta chinensis	Convolvulaceae	Seeds	Acetaminophen induced
Decalepis hamiltonii	Asclepiadaceae	Roots	Carbontetra chloride induced
Elephrantopus scaber Linn	Asteraceae	Whole plant	D-galactosamine and acetaminophen induced
Equisetum arvense	Equisetaceae	Aerial parts	Carbontetra chloride Induced
Embelia ribes	Myrsinaceae	Fruits	Paracetamol induced
Enicostemma axillare	Gentianaceae	Whole plant	D-galactosamine
Euphorbia fusiformis	Euphorbiaceae		Tubers Rifampicin
Ficus religiosa Linn	Moraceae	Stem bark	Paracetamol induced
Fructus schisandrae	Magnoliaceae		Dried fructus Carbontetra chloride Induced
Fumaria indica	Papaveraceae	Whole plant	D-galactosamine induced
Ganoderma lucidum	Polyporaceae	Winter mushrooms	D-galactosamine induced
Ginkgo biloba	Ginkgoaceae	Dried extract	Carbontetra chloride Induced
Glyrrhiza glabra	Fabaceae	Root powder	Carbontetra chloride Induced
Gracinia indica Linn	Clusiaceae	Fruit rind	Carbontetrachloride Induced
Gmelina asiatica Linn	Verbenaceae	Aerial parts	Carbontetrachloride Induced
Gundelia			

tourenfortii	Asteraceae	Fresh edible stalk	Carbontetra chloride Induced
Halenia elliptica	Gentianaceae	Whole plant	Carbontetra chloride Induced
Hibiscus Sabdariffa	Malvaceae	Leaves	Paracetamol induced
Hibiscus esculentus	Malvaceae	Roots	Carbontetra chloride Induced
Hypericum japonicum	Clusiaceae	Whole plant	Carbontetra chloride Induced
Hygrophila auriculata	Acanthaceae	Root	Carbontetra chloride Induced
Hyptis suaveolens Linn	laminaceae	Leaves	Acetaminophen induced
Hoslundia opposite	Lamiaceae	Stem	Carbontetra chloride And paracetamol Induced
Juncus subulatus	Juncaceae	Powdered tubers	Paracetamol induced
Kalanchoe pinnata	Crassulaceae	Leaves	Carbontetra chloride Induced
Lawsonia alba	Lythraceae	Whole plant	Carbon tetrachloride induced
Lactuca indica	Compositae	Aerial parts	Carbontetra chloride Induced
Luffa echinata	Curcubitaceae	Fruits	Carbontetra chloride Induced
Laggera pterodonta	Asteraceae	Whole herb	Carbontetra chloride and D-galactosamine Induced
Mallotus japonicas	Euphorbiaceae	Cortex	Carbontetra chloride Induced
Mamoridca subangulata	Cucurbitaceae	Leaf	Paracetamol induced
Melia azhadirecta Linn	Piperaceae	Leaves	Carbontetrachloride, silymarin induced
Morinda citrifolia Linn	Rubiaceae	Fruit	Streptozotocin induced
Myoporum lactum Linn	myoporaceae	Leaves	Carbontetrachloride Induced
Myrtus communis Linn	Myrtaceae	Leaves	Paracetamol induced
Nelumbo nucifera	Nelumbonaceae	Leaves	Carbontetrachloride Induced
Nigella sativa	Ranunculaceae	Seeds	Tert –butyl hydroperoxide induced
Ocimum sanctum	Lamiaceae	Leaf	Paracetamol induced
Orthosiphan stamineus	Lamiaceae	Leaves	Acetaminophen induced
Phyllanthus amarus schum	Euphorbiaceae	Aerial part	Ehanol induced
Phyllanthus amarus	Euphorbiaceae	Whole plant except root	Aflatoxin b1 induced liver damage
Physalis minima	Solanaceae	Plant material	Carbontetra chloride induced
Phyllanthus niruri	Euphorbiaceae	Leaves and fruits	Carbontetrachloride Induced
Phyllanthus polyphullus	Euphorbiaceae	Leaves	Acetaminophen induced
Picrorhiza kurrooa	Scrophulariaceae	Root and rhizomes	Alcohol –carbon tetra chloride induced
Picrorrhiza rhizome	Scrophulariaceae	Dried underground stem	Poloxamer(PX)-407 induced
Piper chaba	Piperaceae	Fruit	D-galactosamine induced
Piper longum	Piperaceae	Fruits and roots	Carbontetra chloride induced
Pittosporum neelgherrense	Pittospoaceae	Stem bark	Carbontetra chloride, D-galactosamine and acetaminophen Induced
Plantago major	Plantaginaceae	Seeds	Carbontetra chloride induced
Pterocarpus	Papilionaceae	Stem bark	Carbontetra chloride induced

marsupium			
Ptrospermum acerifolium	Sterculiaceae	Leaves	Carbontetra chloride induced
Ricinus communis	Euphorbiaceae	Leaves	Carbon tetrachloride induced
Rubia cordifolia	Rubiaceae	Roots	Carbontetra chloride induced
Sarcostemma brevistigma	Asclepiadaceae	Stem	Carbontetra chloride induced
Saururus chinensis	Sauruaceae	Whole plant	Carbontetra chloride induced
Scoparia dulcis	Scrophulariaceae	Whole plant	Carbontetra chloride induced
Schouwia theebica	Arecaceae	Aerial part	Carbontetra chloride induced
Solanum nigram Linn	Solsnaceae	Fruits	Carbontetrachloride Induced
Tecomella undulate	Bignoniaceae	Stem bark	Thioacetamide induced
Tephrosia purpurea Linn	Fabaceae	Aerial parts	Thioacetamide induced
Thunbergia laurifolia	Acanthaceae	Leaves	Ethanol induced
Tridax procumbens	Asteraceae	Leaves	Carbontetrachloride Induced
Tylophora indica	Asclepiadaceae	Leaf powder	Ethanol induced
Vitex trifolia	Verbenaceae	Leaves	Carbontetrachloride Induced
Vitis vinifera	Vitaceae	Leaves	Carbontetrachloride Induced

In vivo methods

This method is employed not only to investigate the characteristics of the specific compound but also to examine the mechanism of the dangerous substance. Hepatotoxicity can be induced in laboratory animals by administering specific doses of hepatotoxins such as CCl4, galactosamine, thioacetamide, ethanol, and paracetamol. These substances cause significant and measurable effects on the liver, which can be assessed through various liver function tests. These tests include evaluations of liver morphology, metabolism, function, biochemistry, and histopathology. Despite its high level of convenience, the consistency of outcomes in this laboratory approach is somewhat low. The substances with hepatoprotective properties are also assessed to determine their choleretic or anticholestatic activity, which helps determine if the liver disorder is caused by an imbalance in bilirubin metabolism or not. Choleretics are substances that enhance the production of bile by stimulating the liver, while anticholestatics are substances that correct the buildup and retention of bile caused by internal and external causes in the liver. The assessment of these activities involves the examination of bile flow composition in both awake and anaesthetized animals over a period of 5 hours.

Experimental models for hepatoprotective screening

Hepatotoxins are classed as chemical reagents and medications that induce liposis, necrosis, cirrhosis, carcinogenesis, and hepatobiliary dysfunctions in experimental animals. Here are some experimental models that demonstrate the effects of significant hepatotoxins.

CCl_4 model

Various models of CCl4 are developed based on its dose via different methods of administration.

Acute hepatic damage: Oral or subcutaneous dose of CCl4 (1.25ml/kg) causes acute liver injury, which is characterised by ischemia, hydropic degeneration, and central necrosis. The peak elevation of biochemical parameters is

often observed 24 hours following the administration of CCl4, which is commonly given as a 50% v/v solution in liquid paraffin or olive oil.

Chronic reversible hepatic damage: Chronic, reversible liver damage can be induced by administering CCl4 (1ml/kg S.C.) twice weekly for a duration of 8 weeks.

Chronic, irreversible hepatic damage: Administration of CCl_4 (1ml/kg S.C.) twice weekly for 12 weeks simulates chronic, irreversible liver damage.

Thioacetamide model

Thioacetamide (100mg/kg s.c.) induces acute hepatic damage after 48 hrs of administration by causing sinusoidal congestion and hydropic swelling with increased mitosis.

D-galactosamine model

D-galactosamine (800mg/kg i.p.) induces acute hepatotoxocity after 48 hrs of administration with diffused necrosis and steatosis.

Paracetamol model

Paracetamol can cause acute liver damage, which varies based on the dosage and the method of administration. Paracetamol administered intraperitoneally at a dose of 800mg/kg causes centrilobular necrosis without steatosis. Paracetamol administered orally at a dosage of 3 grammes per kilogramme of body weight induces severe liver injury. The toxicity can be induced within a period of 48 hours.

Chloroform model

Hepatotoxicity, characterised by widespread central necrosis, fatty transformation, and hepatic cell degeneration and necrosis, can be induced either through inhalation or subcutaneous dose of 0.4-1.5ml/kg.

Ethanol model

Ethanol elicits varying degrees of liposis based on its dosage, method of administration, and duration of use, as outlined below:

A solitary administration of ethanol (1ml/kg) causes fatty deterioration.

The administration of 40%v/v ethanol at a dosage of 2 ml per 100g body weight per day orally for a duration of 21 days results in the development of fatty liver.

Administering country-made liquor at a dosage of 3ml per 100g of body weight per day orally for a period of 21 days leads to the development of liposis.

Hepatoprotective medicaments

Many medications derived from plants have hepatoprotective properties, either through direct or indirect means. The global utilisation of herbal medications for the management of liver ailments has witnessed a surge in recent times. Herbal medications are commonly perceived as safe and devoid of significant harmful effects, as they are derived from natural sources and readily accessible. In addition, modern medicine, including herbal remedies, has a limited number of therapeutic options and often fails to achieve satisfactory results. Recently, numerous researchers have investigated the impact of plants that have been traditionally utilised in folklore remedies. These plant-based therapies have been employed by indigenous healers and herbalists for a considerable period to promote liver health and address liver-related ailments. Research has generally validated traditional experience and wisdom by uncovering the mechanisms and mode of action of these plants, as well as reaffirming the therapeutic efficacy of certain plants or plant extracts in clinical studies. A comprehensive analysis has been conducted on numerous plant species to explore their potential applications in treating a diverse range of liver ailments. Only a small number has been

thoroughly investigated. There are around 600 commercially available herbal preparations that are purported to have hepatoprotective action, and many of these are being sold worldwide. India has around 40 patented poly herbal compositions, which consist of various combinations of 93 plants from 44 different families. According to reports, 160 phytoconstituents found in 101 different plants have been found to have hepatoprotective action. Herbal medications that protect the liver contain a diverse range of chemical components such as phenols, coumarins, lignans, essential oil, monoterpenes, carotenoids, glycosides, flavonoids, organic acids, lipids, alkaloids, and xanthone derivatives. Research conducted in China and Japan led to the discovery of a hepatoprotective lignan called gomishin, which was isolated from the fruits of the Chinese medicinal plant Schizandra chinensis. Gomishin is utilised for the management of chronic hepatitis. Research conducted at the Tropical Botanic Garden and Research Institute (TBGRI) has demonstrated that Trichopus zeylanicus, Phyllanthus maderaspatensis, and P. kozhikodianus exhibit significant efficacy in combating liver damage induced by paracetamol in rats. A new investigation suggests that fumaric acid derived from Sida cordifolia exhibits notable hepatoprotective properties in rats. Ursolic acid, a compound found in various plants, had significant hepatoprotective effects against liver damage induced by paracetamol and CCl4 in rats. Severalcompounds with pharmacologically and therapeutically proven claims can be identified, such as silymarin, catechin, saikosaponins, curcumin, glycyrrhizin, picroside I and II, gomisin, acetyl bergenin, and kolaviron. The herbs most frequently utilised in herbal compositions in India and proven through scientific experimentation on animals include Andrographis paniculata, Boerhaavia diffusa, Eclipta alba, Picrorrhiza kurroa, Cichorium intybus, and Tinospora cordifolia

Antioxidants have the ability to shield both animals and humans against liver damage caused by oxidants. This phenomenon is observable even in specific ubiquitous vitamins, spices, and plants, such as Vitamin-E and turmeric. Several plants have exhibited hepatoprotective effect. A handful of these plants have been evaluated against various experimental models and are mentioned in the table below.

Conclusion

Despite significant advancements in contemporary medicine, liver illness continues to be a global health issue, necessitating continued efforts to discover novel medications. Chinese ethnomedical practice and traditional medicine employ a variety of medicinal plant compositions to treat liver diseases. Several of these medicines function as radical scavengers, while others serve as enzyme inhibitors or mitogens. The hepatoprotective action of the plants is likely attributed to the presence of flavonoids, alkaloids, terpenoids, glycosides, and steroids. Active botanical extracts, individual fractions, or combinations of fractions/extracts derived from plants have the potential to be very efficacious pharmaceuticals. Plant-based medications, whether used alone or in combination, must demonstrate significant effectiveness in treating severe liver diseases resulting from exposure to toxic chemicals, viral infections (such as Hepatitis B and Hepatitis C), excessive alcohol consumption, and repeated use of drugs like paracetamol, Rifampicin, and Isoniazid. One medicine cannot be universally effective in treating all forms of serious liver disorders. It is necessary to create potent formulations by utilising native medicinal herbs, conducting thorough pharmacological investigations, and carrying out clinical trials. Standards of safety and efficacy should regulate the production of plant products.

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