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Therapeutic potential of *Urtica dioica* L.: A comprehensive review of its hepatoprotective activity

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Abstract
Liver is a vital organ involved in metabolism, detoxification and homeostasis. However, the continuous exposure to toxins such as drugs, alcohol, heavy metals, environmental pollutants cause hepatotoxicity, frequently leads to oxidative stress and hepatocellular damage. Many synthetic drugs are available for the treatment of hepatic diseases, but they have adverse effects. Therefore, the use of herbal medicines has expanded and their popularity has increased. Herbal medicines have long been used to treat liver diseases. *Urtica dioica* commonly called as stinging nettle is a widely distributed herb traditionally utilized in numerous cultures for its various medicinal benefits, including its effects on liver health. This comprehensive review evaluates the available scientific literature concerning the hepatoprotective effects of *Urtica dioica* and analysis of *in vitro*, *in vivo*, and clinical studies investigating its efficacy against different forms of liver injury. The phytochemical constituents are known to exhibit hepatoprotective effects against hepatotoxins by reducing lipid peroxidation, enhancing activity of antioxidant enzyme, modulating inflammatory responses, stabilizing cell membrane modulating key enzymes involved in detoxification and metabolism. Experimental studies including *in vivo* and *in vitro* showed that the plant extract reduces hepatic injury induced by toxins such as Carbon tetrachloride, acetaminophen, alcohol, thioacetamide, erythromycin *etc. Urtica dioica* exhibits substantial potential as a natural hepatoprotective agent, providing a preventative strategy for liver diseases.

Keywords: *Urtica dioica,* hepatotoxicity, hepatotoxin agents, hepatoprotective

Introduction

Liver is a largest and key organ of human body which plays diverse functions in the body such as synthesis, biotransformation of food and medicine, secretion, metabolism, detoxification, maintaining homeostasis, storage and removal of xenobiotics (Ingawale *et al*., 2014) [14]. The organ is involved in essential life processes such as growth, nutrient supply, energy production, metabolism of carbohydrates, proteins, fats and stores vitamins, proteins, glycogen (Maqbool *et al*., 2019) [27]. Liver carries out its digestive function by the production of bile and emulsification of oils, fats and Vitamins A, D, E and K (Okaiyeto *et al*., 2018) [31].

Liver disorders are a major global health issue, especially in developing countries. Liver injury also known as dysfunctioning of liver is generally induced by alcohol, xenobiotics (CCL_4, chlorinated hydrocarbons, CO_2, O_2,), immunosuppressants (cyclosporine), analgesics, anti-inflammatory drugs (PCM), anti-tubercular drugs (rifampicin, isoniazid), heavy metals (Cd, As), mycotoxin (aflatoxin), galactosamine, lipopolysaccharides, toxin compounds (thioacetamide, t-BuOOH), antibiotics (erythromycin) (Ingawale *et al*., 2014; Iqubal *et al*., 2016) [14, 15]. The toxins are transformed into intermediate reactive radicals which are responsible for hepatotoxicity (Kanter *et al*., 2003) [23]. Most of the hepatocytes either die or get converted to a fibrotic condition in acute liver injury. Excessive oxidative stress generated by over production of free radicals plays role in the formation of disease and behave as a prognostic factor in liver damage (Zhu *et al*., 2012) [42]. Symptoms may include jaundice, skin, eye, severe abdominal pain, weakness, extreme fatigue, persistent bleeding, and rapid weight gain (Maqbool *et al*., 2019) [27]. Liver diseases are caused due to alteration in the equilibrium of oxidative defence (Joshi *et al*., 2015) [20]. Glutathione (GSH), superoxide dismutase (SOD), ceruloplasmin, glutathione peroxidase (GPx), catalase and Vitamins A, C & E are the antioxidant systems that shield the cells against lipid peroxidation (Kanter *et al*., 2003) [23]. So, it is essential to maintain the equilibrium between antioxidant systems to avoid the oxidative stress and lipid peroxidation. The mechanism of hepatic damage by lipid peroxidation and oxidative stress is shown in Fig 1.

Urtica dioica also known as nettle or stinging nettle of Urticaceae family is a perennial plant growing in temperate and tropical regions throughout the world. Since ancient period, it has been used as a traditional medicinal herb in various countries (Kk and Parsuraman, 2014) [24]. The leaves of are used to treat asthma, diabetes, rheumatism, tuberculosis, scurvy, allergic rhinitis, fever, hypertension, cough, arthritis and snake bites (Dhouibi *et al*., 2020) [12]. It has anti-inflammatory, antioxidant, antiproliferative, hepatoprotective, analgesic, anti-infectious, allelopathic, hypotensive and anti-ulcer properties because of the presence of various phytoconstituents like polyphenols, terpenoids, volatile compounds, tannins, alkaloids etc. (Bhusal *et al*., 2022 [7]; Kumar *et al.,* 2025; Ajah *et al*., 2024 [4, 25]; Chira *et al*., 2025) [9].

The hepatoprotective effects of *U. dioica* are due to the presence of hepatoprotective agents which protects from hepatic injury by reducing lipid peroxidation and boosting

the antioxidant enzymes (Kanter *et al*., 2005) [22]. Quercetin (QR) is a naturally occurring flavonoid which protects the cells from CCl_4-associated liver injury, by reducing the effect of oxidative stress and inflammation. It is an excellent hepatoprotective agent with good pharmacological effect against fatty liver, hepatic fibrosis, hepatic steatosis and hepatic cancer (Jain *et al*., 2020; Zhao *et al*., 2021) [17, 41]. QR exhibit hepatoprotective effects (Fig 2) through various mechanisms, including the reduction of hepatic inflammation through NF-kB/TLR/NLRP3 signalling pathways. Furthermore, it inhibits the production of apoptotic proteins linked to the development of hepatic diseases, controls mTOR activation in autophagy, and reduces oxidative stress via the PI3K/Nrf2 pathway (Zhao *et al*., 2021) [41]. QR has been shown to promote apoptosis in hepatic cancer cells by regulating various signalling pathways, such as downregulating Akt, cyclin-A, cyclin-B1, CDC-2, CDK-2, PLK1, and Bcl-2 and upregulating p21, caspase-3 and Bax. It also influences important signalling pathways such as MAPK/ERK and PI3K/Akt, which contributes to its apoptotic effects. QR regulates cell death by altering the balance between pro-apoptotic and anti-apoptotic proteins, producing oxidative stress, interfering with mitochondrial function and causing endoplasmic reticulum stress (Xiong *et al*., 2024) [39]. QR shows a potential hepatoprotective activity by reducing DNA damage caused by acrylamide (ACR). It scavenges free radical production and prevents liver tissues from being damaged by ACR (Ansar *et al*., 2016) [6]. QR shields the liver from damage caused by CCl_4 and is effective against tert-butyl hydroperoxide (t-BHP) induced liver injury, thereby acting as a hepatoprotective agent (Kalantari *et al*., 2018) [21].

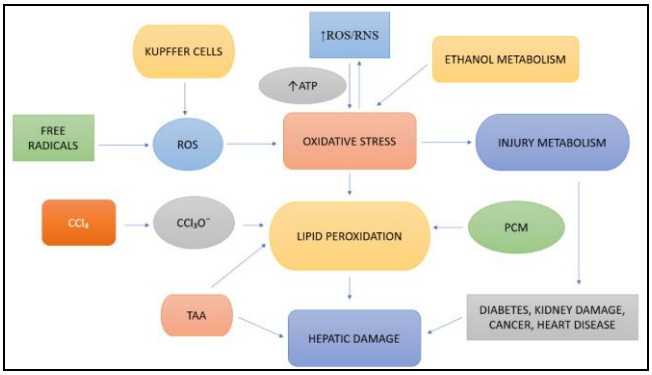

Fig 1: Mechanism of hepatic damage

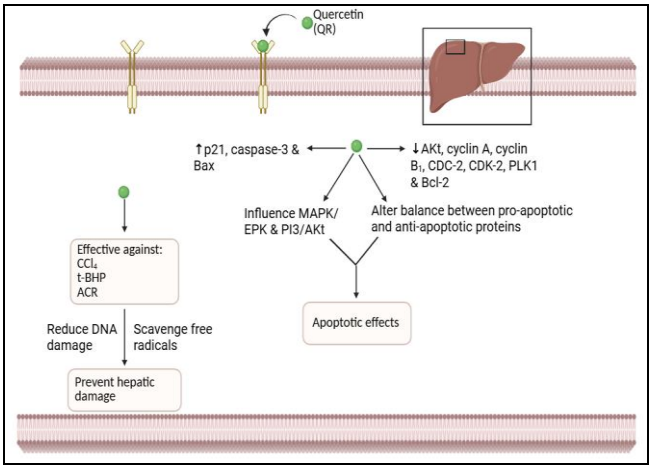

Fig 2: Hepatoprotective effect of quercetin

Hepatotoxicants and Mode of Action

Hepatotoxicants (Fig 3) are the agents or molecules which induced hepatotoxicity in the body of an organism. They are the harmful exogenous agents which cause various hepatic diseases and affecting the whole body of the organism (Maqbool *et al*., 2019) [27]. Direct hepatotoxicants include carbon tetrachloride, acetaminophen, thioacetamide, galactosamine, fulvin, ethyl alcohol, phalloidin, and aflatoxin. Indirect hepatotoxicants include methyltestosterone, tetracycline, chlorpropamide halothane, phenytoin sulfonamides, and rifampicin. It can cause cytotoxic effects like apoptosis, fibrosis, steatosis, necrosis, cholestasis, hepatitis, cirrhosis, and liver cancer.

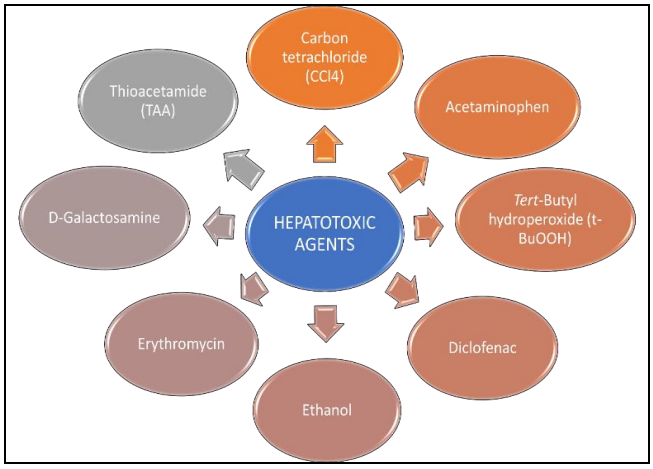

Fig 3: Hepatotoxic agents

Carbon tetrachloride

Carbon tetrachloride (CCl_4) is a selective hepatotoxin which generates reactive free radical (CCl_3) due to its reductive metabolism by hepatic cytochrome P450. The reactive free radical starts cell injury by covalently binding to membrane protein and cause lipid peroxidation (Kanter *et al*., 2005) [22]. The cytochrome P-450 and CYP2E1 are the two enzymes present in endoplasmic reticulum and mitochondria metabolising the CCl_4. The reactive oxygen species (ROS, CCl_3O^-) initiates a chain reaction that may lead to lipid peroxidation and causes damage to liver cells. The production of trichloromethyl radical (CCl_3) is highly reactive which results in hypertoxicity (Okaiyeto *et al*., 2018) [31]. The mechanism of CCl_4 induced hepatotoxicity is shown in Fig 4.

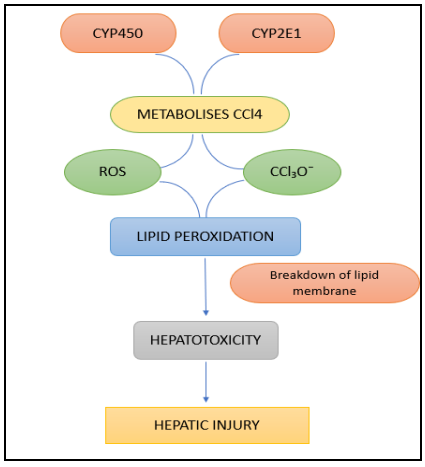

Fig 4: Mechanism of CCl_4 induced hepatotoxicity

Thioacetamide (TAA)

Thioacetamide (TAA) is an organic compound that obstructs the movement of RNA between the nucleus and cytoplasm. It causes either chronic or acute liver injuries and affects the formation of DNA, RNA and proteins (Jannu *et al*., 2012 [19]; Delgado-Montemayor *et al*., 2015) [10]. It has the potential to causes cholestasis and decline the number of liver cells and oxygen uptake (Iqubal *et al*., 2016) [15]. The enzymes CYP450 and monooxygenases (containing flavin) are responsible for the bioactivation of TAA. TAA is converted into TAA sulfine (highly reactive) and sulfone-type compounds by oxidation and cause severe damage to hepatocytes (Akhtar and Sheikh, 2013) [5]. The mechanism of TAA associated hepatotoxicity shown in Fig 5.

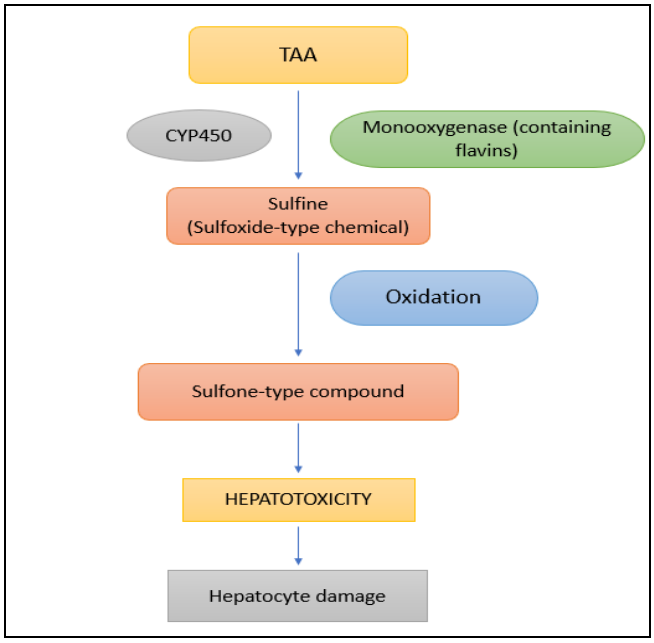

Fig 5: Mechanism of TAA associated hepatotoxicity

Acetaminophen

Acetaminophen also known as paracetamol (PCM) is the most extensively prescribed antipyretic and analgesic medication. It is usually considered safe when taken in proper amount or doses. The over dose or high consumption causes necrosis of hepatocytes which leads to acute liver damage (Iqubal *et al*., 2016) [15]. At normal doses, PCM is metabolised in liver by CYP450 into N-acetyl-P-benzoquinone imine (oxidative product). The detoxification of NAPQI metabolite which is highly reactive is done by GSH. At high doses, the excess amount of NAPQI is formed in the body results into depletion of GSH. This toxic NAPQI attaches covalently with sulphydryl groups of protein leading to lipid peroxidation, deprivation of GSH level and hence induce the liver necrosis (Iqubal *et al*., 2016; Jannu *et al*., 2012; Ibrahim *et al*., 2013) [13, 15, 19]. The mechanism of hepatotoxicity caused by PCM is shown in Fig 6.

Erythromycin

Erythromycin is an antibiotic which is usually consumed by the people, but it's over consumption can lead to liver injury. The metabolite of erythromycin produces free radicals that induce hepatotoxicity and even death (Pari and Murugan, 2004) [32]. Although acute hepatic damage from erythromycin is rare, its extensive usage has been a prevalent source of hepatic injury caused by drug. The enzyme CYP3A4 of the CYP450 enzyme family metabolises the erythromycin (Shafia *et al*., 2016) [36]. Prolonged administration of erythromycin can lead to the formation ROS which plays important role in hepatotoxicity (Fig 7) (Jamshed *et al*., 2022) [18]. Erythromycin estolate was initially considered to be the primary cause for hepatotoxicity, but recently it has been recognized that all types of erythromycin have the potential to cause hepatic injury. It may lead to cholestatic hepatitis, the most prevalent form of hepatic damage, in which the flow of bile from the liver is blocked.

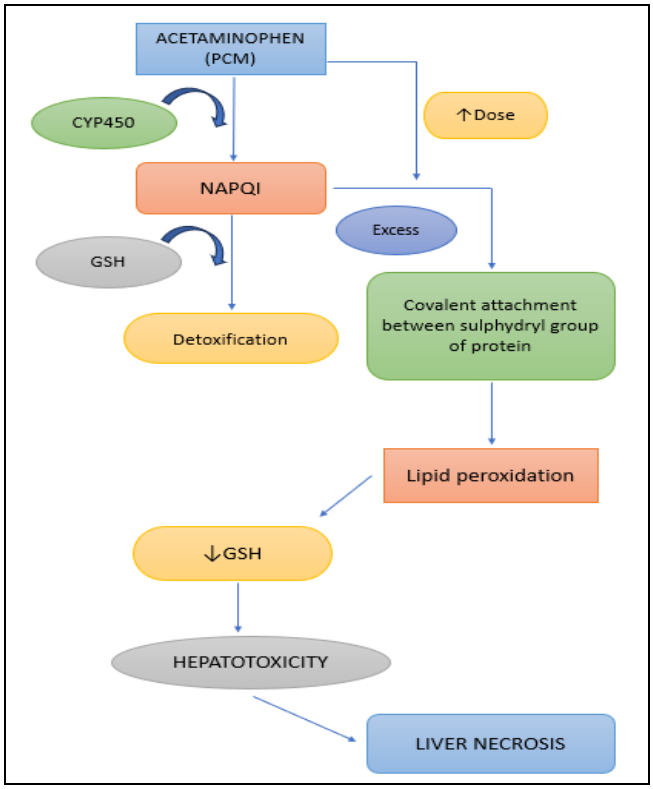

Fig 6: Mechanism of hepatotoxicity by PCM

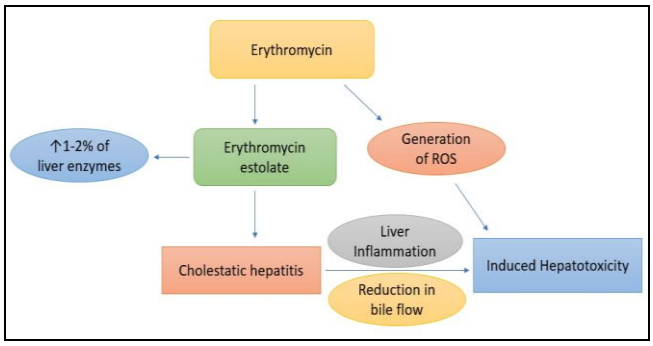

Fig 7: Hepatotoxicity by erythromycin

Diclofenac

Diclofenac is a non-steroidal anti-inflammatory medicine used to cure acute muscle pain, fever, rheumatoid arthritis, and osteoarthritis. Though the consumption of diclofenac in appropriate amount is normally safe but it can induce severe hepatic injury in rare cases (Aithal *et al*., 2004; Boelsterli, 2003) [3, 8]. Diclofenac may produce oxidative stress and dysfunctioning of mitochondria which can lead to liver cell damage (Fig 8) (Ponsoda *et al*., 1995) [34]. The clinical use of diclofenac is limited due to its potential for idiosyncratic liver injury (Aithal, 2011) [2].

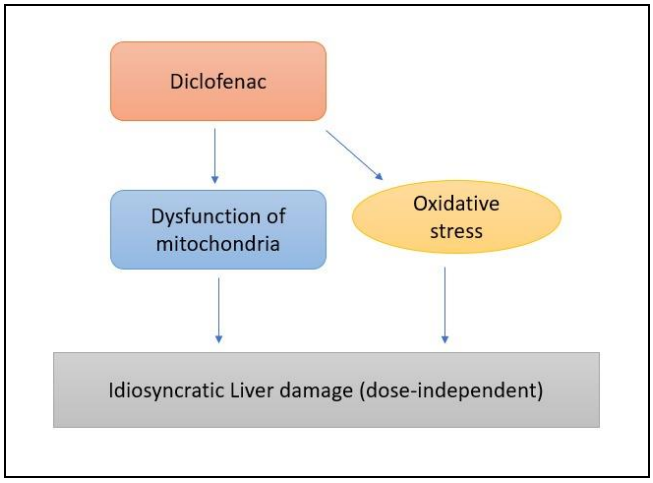

Fig 8: Hepatotoxicity by diclofenac

D-Galactosamine (D-Gal)

D-Galactosamine is a hepatotoxicant harming the liver by specifically reducing uridine nucleotides in liver cells. This result in deficit of RNA due to prohibition of RNA synthesis and affects the production of proteins (Lian *et al*., 2010; Iqubal *et al*., 2016) [15, 26]. One dosage of D-Gal may result fatty liver and liver cell infection (Delgado-Montemayor *et al*., 2015) [10]. D-Gal produces damage that is same to that produced by viral hepatitis in terms of morphological and functional features. The mechanism of hepatotoxicity caused by D-Gal is shown in Fig 9.

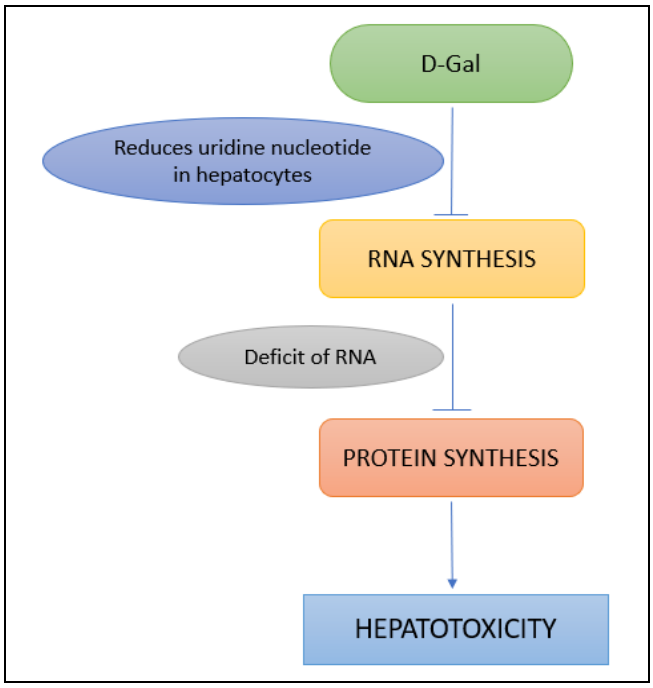

Fig 9: Hepatotoxicity mechanism produced by D-Gal

Tert-Butyl hydroperoxide (t-BuOOH)

t-BuOOH is an inducer of oxidative stress and generate lipid peroxidation which affects cell integrity resulting in cell damage (Oh *et al*., 2012). It get converted into free radical intermediates, like methyl and t-butoxyl radicals due to the enzyme CYP450 (in liver cells) or haemoglobin (in RBCs) which lead to lipid peroxidation, haemolysis and oxidative denaturation of Hb, glutathione depletion, permeabilization of cellular membranes and DNA injury resulting in cellular damage. This damage is comparable to oxidative stress produced in cells or tissue. Glutathione Peroxidase (GPx) transforms the t-butyl hydroperoxide into t-butyl alcohol (Oh *et al*., 2012; Wang *et al*., 2000) [38]. Glutathione (GSH) is used as a cofactor by GPx enzymes to convert a variety of peroxides, including t-BuOOH, to their respective alcohols. Protecting cells from oxidative damage produced by reactive oxygen species is an essential component of cellular antioxidant defense mechanisms (Pei *et al*., 2023) [33]. The mechanism of hepatotoxicity caused by t-BuOOH is shown in Fig 10.

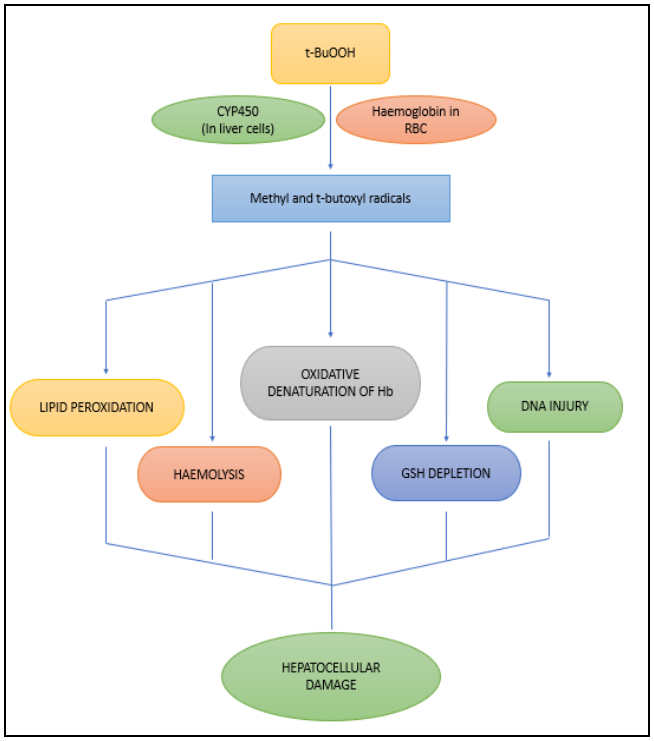

Fig 10: Mechanism of t-BuOOH inducing hepatotoxicity

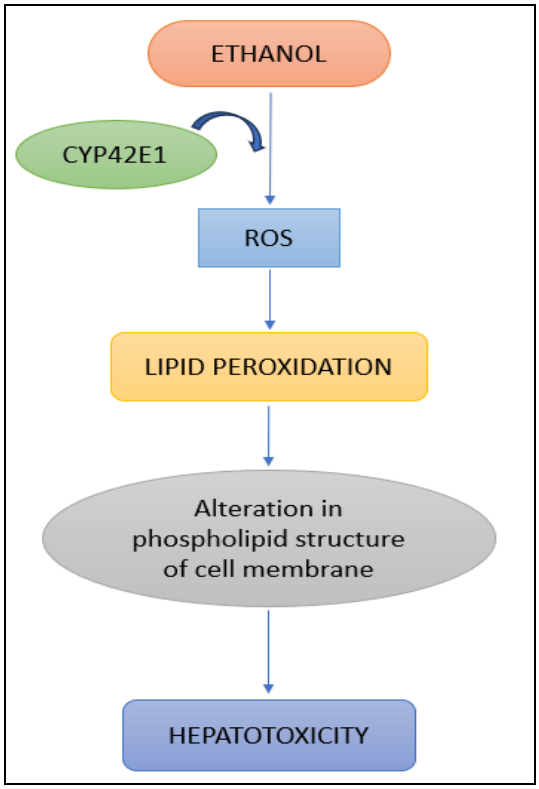

Fig 11: Hepatotoxicity mechanism caused by alcohol

Alcohol

Excessive consumption of alcohol has become one of the major primary cause of liver damage. Ethanol is a powerful inducer of ROS production in the body and oxidative stress play an important role in the hepatotoxicity (Mollazadeh and Hosseinzadeh, 2014) [28]. Alcohol-mediated hepatotoxicity (Fig 11) induced by the generation of lipid radical, lipid peroxidation, protein conjugation and glutathione depletion leads to hepatic diseases like cirrhosis and hepatitis (Mollazadeh and Hosseinzadeh, 2014) [28]. The metabolism of alcohol occurs in liver which disturbs lipoprotein and lipid metabolism (Okaiyeto *et al*., 2018) [31]. Ethanol is metabolised by the isoform of the enzyme CYP50 i.e. CYP2E1, which generate oxidative stress with the production of reactive species of oxygen (ROS), results in enhancement of lipid peroxidation, and leads to the change in the structure of phospholipid of the cell membrane (Jaeschke *et al*., 2002) [16].

Hepatoprotective activities

The dried root extract of *U. dioica* was evaluated for hepatoprotective property in albino rats. Rats intoxicated with CCl_4 showed higher serum levels of alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and bilirubin than the normal control group. Treatment with the plant extract restored the enzyme activity and was found to be effective in preventing cirrhosis and liver fibrosis. The results suggest that root extract improve hepatic marker functions by aiding in detoxification and free radical scavenging. The histological findings confirmed that the extract shows hepatoprotective effect against liver damage caused by CCl_4 (Shawagfeh, 2017) [37]. The chronic liver damage was caused by CCl_4 in male Sprague Dawley rats and *U. dioica* at 200 and 400 mg/kg doses, significantly inhibited CCl_4-induced hepatic encephalopathy due to the reductions in AST, ALT, and GGT level. *U. dioica* effectively reduced liver damage and enhanced liver function, and provide protection against CCl_4-induced brain toxicity (Deniz, 2018) [11]. The liver marker enzymes are elevated and released into the bloodstream because of the CCl_4 induced liver damage. The *in-vivo* studies showed that the serum levels of SGOT, ALP and SGPT decreased toward their respective normal values after treatment with *U. dioica* ethyl acetate fraction. The oxidative stress was mitigated by restoring GSH and catalase and lowering lipid peroxidation and nitrite level. The plasma membrane was stabilized and the toxicant-induced damage to the liver tissue has been repaired. The hepatoprotective effect was caused by the presence of antioxidant phenolic components such as ferulic acid quantified by HPTLC (Joshi *et al*., 2015) [20]. Hepatotoxicity was induced in female *Mus musculus* mice by intraperitoneal injection of N-Nitroso methyl urea (NMU). The extract when administered after NMU exposure restores the liver damage caused by carcinogen- and inhibited the carcinogenic process (Abir *et al*., 2017) [1]. A significant increase in lipid peroxidation, liver enzymes, and decreased antioxidant enzyme levels confirmed CCl4-induced toxicity in Wistar rats. *U. dioica* treatment significantly reduced the elevated liver enzymes (ALT, ALP), and bilirubin in the CCl_4 and *U. dioica* treated group. *U. dioica* reduced the hepatotoxic effects of CCl_4 by raising the antioxidant enzymes (catalase, superoxide dismutase, glutathione) level and significantly reducing lipid peroxidation levels (Naz and Mahboob, 2014) [29]. The methanolic extract of leaves was tested for hepatoprotective activity against CCl4 induced liver damage in HepG2 cell line. The extract exhibited highest hepatoprotection at a dose of 110µg/ml (Priyachitra *et al*., 2023) [35]. The protective effect of seed extract was investigated against radiation-induced liver injury in albino rats. Radiation has been reported to enhance oxidant molecule, AST, ALT, and MDA levels while reducing antioxidant enzymes such as SOD, CAT, and Glutathione peroxidase (GSH-Px). These results indicate that *U. dioica* in combination with radiotherapy decreases the harmful effects of irradiation in liver (Yıldızhan *et al*., 2020).

Conclusion

U. dioica exhibits the significant hepatoprotective potential and can be used as a natural agent with multifaceted hepatoprotective properties. The experimental studies demonstrate its efficacy against various forms of liver injury caused by drugs, toxins, and metabolic disorders. Preclinical studies showed hepatoprotective effects by minimizing the hepatic injury induced by hepatotoxic agents such as CCl_4, alcohol, drugs, environmental pollutants *etc.* The phytoconstituents exhibits significant hepatoprotective effects against hepatotoxicants and play a crucial role in scavenging oxidative stress, reducing lipid peroxidation, stabilizing the cellular membrane, modulating inflammatory pathways and enhancing the antioxidant enzyme activity, thereby preventing or reducing hepatic injury.

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