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Evaluation of Hepatotoxicity Effects on Fruits and Vegetables

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Abstract – Liver toxicity in world health problems is a major challenge. Food and Drug Administration (FDA), which causes induced hepatotoxicity, is withdrawing many of the drugs from the market. In this study, in vitro hepatotoxicity, enhancing activity of methanoic extracts from different parts (peel, pulp and seed) of carbon-tetrachloride exotic fruits and vegetables (CCl4) was studied. For lipid peroxidation (LPO), a free radical scavenging and sodium dismutase (SOD) test, chicken liver cell suspension treated with CCl4 was employed. Different extracts from exotic fruits and common vegetables have shown a powerful antioxidant effect. Methanoic extract from some unused parts of vegetables and fruits (peel and seed) even showed hepatoprotective action but the remarkable hepatoprotective action was shown among all these fibergine pulp (vegetables) and passion fruit pulp (exotic fruit) Thus we conclude that there was notable protective activity in CCl4 induced by the methanoic extracts of exotic fruits and vegetables.

Key Words – Hepatotoxicity; Liver Injury; Paracetamol; Carbon Tetrachloride (Ccl4); Thioacetamide (TAA); Hepatoprotective Agents.

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INTRODUCTION

India has cultural diversity and traditions. Specific dietary and medical practices are being followed in the region. A wide range of traditional medicine practices, such as folk medicines and tribe medicine, Ayurveda, Siddha, Unani etc., are practiced in India. However, it is also noted that most of the food-producing plants and their parts, such as fruits, seeds, leaves and rooted substances of medicinal value like antioxidants, flavonoids, tannins and other phenolic compounds [1].

The liver is the largest glandular and main organ in the body to maintain the inner body. Almost every organ in the body is supported in some facet by the liver and vital for survival. Some of these major functions include protein, fat and carbohydrate, detoxification, bile secretion, and vitamin storage. The system performs several critical functions, which ensure that the body is purified by filtering toxins and many medicines from the blood. Maintaining a healthy liver is therefore an important factor in human health and well-being as a whole. Although the liver has tremendous capacity for regeneration, the disease of the liver has increased strongly. Drug-induced liver injury is prominent among all reasons for hepatotoxicity. It has an annual incidence of 10 to 15 to 100,000 people exposed to prescription drugs. Approx. 20 percent of the ALF in children and a higher percentage of the ALF in adults are induced by a drug induced by acute liver failure (ALF). Hepatotoxicity is therefore one of the leading safety reasons for drug withdrawal and is one of the major pharmaco-awareness concerns. The model substances that cause in vivo or in vitro experimental hepatocyte injury often use chemical toxins (including acetaminophen, carbon tetrachloride, galactosamine and thioacetamide). Most of the hepatotoxic chemicals cause lipid peroxidation damage to the hepatocytes. Maybe the best studied model for cirrhosis of the liver is the cirrhosis induced by CCl_4. CCl_4 was the first toxin to demonstrate that the injury generated by a free-radical mechanism is mediated in large part or entirely. The cytochrome P450 system metabolizes CCl_4 to give a radical, carbon-centered trichloromethyl. The initial stage in a chain of events leading finally to membranous lipid peroxidation (LPO) and finally to cell apoptosis and necrosis of trichloromethyl free radicals is considered to be the covalent binding of cell proteins. High levels of reactive oxygen cell damage (ROS), including liver cirrhosis and fibrosis, involve several human pathology. Reactive aldehydes, malondialdehyde (MDA), and 4-hydroxynonenal, which easily bind to functional protein groups, are part of the degradation products. Cellular disturbance is most likely the result of increased membrane permeability of these reactive acid thiobarbiturates (TBARS), lactate dehydrogenase (LDH) leakage, cell protection loss,

GSH depletion, and as a result of any of these changes – cell death. The second phase of hepatotoxicity-induced CCl_4 involves activating the Kupffer cells and producing pro-inflammatory mediators. Hence hepatoprotection can be demonstrated by extracts with high free radical breaking activity/antioxidant. Free radicals are also transformed into waste by-products and eventually removed from the body. They can also repair cell damage beforehand. However, consumption of fruits and vegetables is known to lower the risk of several diseases, and such health benefits are mainly imposed due to the presence of phytochemicals, such as polyphenols, carotenoids and Vitamin E and C.

Hepatoprotective Activity of Some commonly consumed vegetables (in the form of seeds, roots, leaves and fruits)

In Indian traditional medical practice a variety of food significant plants are used as preventive substances for diverse ailments. Many vegetables are consumed without the realization of the medicinal values. However, a list of plants reported to have significant hepatoprotective activity, in alphabetical order of their family, together with their scientific names, plant part consumed, type of extract used in assay, Hepatotoxicity inducer, biochemical parameters studied and references.

HEPATOTOXIC CHEMICALS PARAMETERS STUDIED

Most of the hepatotoxic chemicals damage liver cells mainly by inducing lipid peroxidation and other oxidative damages in liver. Estimation of the alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin (TB), total protein (TP), Total Glyceride (TG), albumin (ALB), glutathione (GSH), Gamma-glutamate transpeptidase (GGTP), malondialdehyde (MDA), Serum glutamate oxaloacetate transaminase (SGOT) and Serum glutamate pyruvate transaminase (SGPT), Reactive Oxygen species (ROS) such as superoxide dismutase, catalase with the levels of control animals and micrographs on histopathological changes were used in general as diagnostic tools [3].

Allium cepa L

The bulbs and their extracts were tested against Cl4, Ethyl Acetate and paracetamol, and induced liver toxicity in wistar albino rats for their hepatoprotective activities by Rawat et.al.[4]. Paracetamolic or acetaminophen produces liver necrosis at a larger dose by cytochrome P450 monoooxygenase following bioactivation with toxic electrophilic, N-acetyl-p-benzoquinone-imine (NAPQI) (a microsomal enzyme). This changes the SGOT & SGPT levels. Tetrachloride carbon administration has significantly raised the SGOT, SGPT, ALP, and bili rrubin serum levels, altering the liver cell structure and function due to its enzymatic activation of a free radical CCl3. In their research, they observed that high levels of biochematic parameters have been substantially decreased due to their interference with cytochromome P450 monooxygenase by the treatment of alcohol extract (AEAC) and aqueous extract (AQEAC) because they contain saponins, carbonates, steoids and flavonoids, which reducing hepatotoxic free radicals. The hepatoprotective effect of these extracts has been shown too in the liver histopathology.

The allium-cetoaqueous extract (ACE) preventive effect against cadmium-induced hepatotoxicity has been reported by Ige et. [5] using male Wistar rats as a model [6-7]. They showed that cumulative oxidative damage is the hepatotoxic effect of Cd. The concentration in the livers of all Cd-exposed group of rats is observed as cadmium decreases superoxide dismutase (SOD) and increases malondialdehyde (MDA). Aminotransferase levels of serum aspartate [8]. ALT, AST and ALP are the most common enzymes used for hepatocellular damage. Liver damage leads to the enzymes' increased plasma activity. The mechanism through which AcE improves the hepatotoxicity of Cd is its ability to preserve the integrity of hepatocytes and scavenging ROS.

Allium sativum L

Ajayi et.al and Senapati et.al. [9-10] have studied the supplementary diet of garlic with vitamin C and its hepatoprotective effect on lead-induced liver toxicity. Due to the lead therapy they found significant increases in the ALT and ALP enzyme levels as well as decreases in AST. A. sativum and vitamin C post-lead treatment considerably reduced ALT and ALP activities and increased plasma AST activity, such as control activity. Body is well known to link cysteine-containing sulfhydryl groups of enzymes that form amino acids and protein complexes. Because ALT is the liver enzyme, lead changes the tissue level of ALT activity by interrupting its membrane and therefore transferring to plasma in the blood[11-12]. The reduced serum ALT and ALP activities can usually be attributed to a reduced production of these sources of enzymes, which therefore indicates the reverse effect of the toxicity of lead. Ebenyi et al. (14] on Paracetamol, Md. Asaduzzaman and al. [15] on Islandazid, and other studies were carried out in a similar manner [13] on Paracetamol.

Amorphophallus paeoniifolius

Pramod J Hurkadale et al. [16] investigated the methanol and aqueous extracts of elephant foot yam tuber and the hepatoprotective act on males albino-wistar rats induced by paracetamol. The hepatotoxicity of paracetamol is caused by the

oxidative stress- and lutathium depletion reaction metabolite N-acetyl-p-benzoquinoneimine (NAPQI). It is a renowned analgesic and antipyretic agent that produces liver necrosis at higher doses. Before the administration of paracetamol the pretreatment of rats with methanol and aqueous mineral was almost comparable with the silymarin and the Liv-52 in the form of SGOT, SGPT, SALP and SB (P<0.01). A histopathologic test of the controlling liver tissue and amorphophallus extract treated animals was confirmation of the hepatoprotective action. The report demonstrates that flavonoids and steroids are responsible for hepatoprotection, as the major chemical constituents of the tubers are carbohydrates, sitosterol, stigmasterol, thiamines and riboflavins.

Benincasa hispida

Das, S.K, and C. Roy have reported hepatic damage against diclofenac sodium caused by aqueous extract of winter melon (Benincasa sespida = BH) on male albino rats. It was observed that the hepatoprotective effect was caused by antioxidant - mediated mechanism, alteration of Serum GLOT, serum glutamate pyruvate transaminase (SGPT), alkaline phosphatase (ALP), SOD (SUD) and catalase (CAT), reduction of glutathyonics (GSH) and lipid peroxidation (LPO). Vitamin E, beta-carotene, flavonoids and flavonols are contained in the BH pulp. Therefore, the results of SGPT, SGOT, ALP, LPO, CAT, SOD and GSH, potentially vitamin E, beta carotene, flavonols and flavonoids present in the pulp of BH, may be found to protect rat liver against oxidative stress. The hepatoprotective effect of BH against Nimesulide also reported by Das and Roy.

Beta vulgaris

The presence of flavonoids, carbohydrates, betainas and anthocyanin pigments is shown in phytochemical studies of the beetroot. The effect from the CCl4-induced hepatic damage in rats of ethanolic extract Beta vulgaris (EEBV) root. Estimates of serum enzyme levels of aspartate aminotransferase (AST), alanine amine transferase (ALT," alkaline phosphatase (ALP), total protein and bilirubin were investigated into EEBV's hepatoprotective activity. Treatment with EEBV (P<0,01) is observed to reduce the elevated serum levels of enzyme activity induced by CCl4 and the increase by the total protein, AST, ALT and ALP of parallel significant (P<0,01) bilirubin, indicating an extract that could maintain the normal functional status of the headache. Histology of the liver sections of the extract treated animals was also observed, which demonstrated further hepatoprotective activity by the presence of normal hepatic cords, the lack of necrosis or fatty infiltration. It can be because of its chemical content using Beta vulgaris leaves that it has hepatoprotective effect.

FRUIT AND VEGETABLE EXTRACT PREPARATION:

Fresh exotic fruit (Table 1a) and vegetables (Table 1b) from hop comes were bought and washed with running pumpkin water and separated from them their peel, pulp and grain. For more than a month, the shade was dried separately. A mechanical grinder was used for the dried fruit parts. Each sample was weighed approximately and extracted by cold maceration at room temperature with 80 percent methanol. The 80 percent methanol could be drenched for 72 hours with macerate. The samples have been filtered with regular filter paper after 72 hours. The methanolic extracts thus obtained was collected in the vials and stored at 4^{o}C.

Table 1a: Exotic Fruits Used In Experiment

Exotic fruits	Scientific Name
Date Palm	*Phoenix Dactylifera*
Grape Vine	*Vitis Vinifera*
Passion Fruit	*Passiflora Edulis*
California Wild Grape	*Vitis Californica*
Kiwi Fruit	*Actinidia Deliciosa*
Wood Apple	*Limonia Acidissima*
Indian Jujube	*Ziziphus Mauritiana*
Avocado	*Persea Americana*
Litchi	*Litchi Chinensis*
Java Plum/Black Palm	*Syzygium Cumini*
Rose Apple	*Syzygium Jambos*
Soursop	*Annona Muricata*

Table 1 b: Common Vegetables Used In Experiment

Common Vegetables	Scientific Name
Eggplant	*Solanum melongena*
Bitter Gourd	*Momordica charantia*
Ivy Gourd	*Coccinia grandis*
Tomato	*Solanum lycopersicum*
Green chilli	*Capsicum frutescens*
Ladies finger	*Abelmoschus esculentus*
Cucumber	*Cucumis sativus*
Bottle Gourd	*Lagenaria siceraria*
Bell peppers	*Capsicum annuum*
Snake Gourd	*Trichosanthes cucumerina*

CONCLUSION:

This paper describes the use, by hepatoprotection, of entire plant extracts (peel, pulp, seed) from common vegetables and exotic fruit. All the extracts with powerful hepatotoxicity actions have been shown. This was concluded by the above results, which showed a significant anti-hepatotoxic effect even in unusing parts for CCl_4 induced liver toxicity in in vitro conditions of exotic fruit and common vegetable mechanics extracts.

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