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The effect of functional foods to treat liver disorders: Review study

Yousif Elhassaneen[1*] and Marwa Kamal [1]

[1]Department of Nutrition and Food Science, Faculty of Home Economics, Minoufiya University, Shebin El-Kom, Egypt
*Corresponding author: yousif12@hotmail.com

Abstract: Functional food can be defined as a food that delivers a health benefit beyond basic nutrition and makes a claim about this benefit. Functional foods can be whole foods or foods that naturally contain or have been fortified with nutrients and/or bioactive substances that provide a specific benefit to health. Epidemiological studies randomized clinical trials carried out in different countries have demonstrated numerous health effects related to functional food consumption, such as reduction of cancer risk, improvement of heart health, stimulation of immune system, decrease of menopause symptoms, improvement of gastrointestinal health, maintenance of urinary tract health, anti-inflammatory effects, reduction of blood pressure, maintenance of vision, antibacterial and anti-viral activities, reductions osteoporosis and anti-obese effect. Therefore, this review article will give an idea of functional foods from the chemical and nutritional point of view. Also, the effect of some functional foods such artichoke, milk thistle, turmeric and gum arabic on the prevention and/or treatment of some diseases will be also dicussed.

Keywords: Functional food- artichoke-milk thistle- turmeric- gum arabic- antioxidant enzymes activities

Functional foods

Functional foods are simermilar in appearance to conventional foods; the former being consumed as part of the normal diet. In contrast to conventional foods, functional foods, however, have demonstrated physiological benefits and can reduce the risk of chronic disease beyond basic nutritional functions, including maintenance of gut health. When food is being cooked or prepared using "scientific intelligence" with or

without knowledge of how or why it is being used, the food is called "functional food". Thus, functional food provides the body with the required amount of vitamins, fats, proteins, carbohydrates, etc., needed for its healthy survival (FAO, 2010). Accordinto the definition, functional food is a part of an everyday diet and is demonstrated to offer health benefits and to reduce the risk of chronic disease beyond the widely accepted nutritional effects. The term 'functional foods' was introduced in Japan in mid 1980s. This type of foods is known on the Japanese market as "Foods for Specified Health Use" (FOSHU)(Grajek *et al.*, 2005). Among the functional components, probiotics and prebiotics, soluble fibre, *omega*-3-polyunsaturated fatty acids, conjugated linoleic acid, plant antioxidants, vitamins and minerals, some proteins, peptides and amino-acids, as well as phospholipids are frequently mentioned. Epidemiological studies randomized clinical trials carried out in different countries have demonstrated numerous health effects related to functional food consumption, such as reduction of cancer risk, improvement of heart health, stimulation of immune system, decrease of menopause symptoms, improvement of gastrointestinal health, maintenance of urinary tract health, anti-inflammatory effects, reduction of blood pressure, maintenance of vision, antibacterial and anti-viral activities, reductions of osteoporosis and anti-obese effect (Kris-Etherton *et al.*, 2000).

Artichoke

The globe artichoke (*Cynara cardunculus var. scolymus*), is a perennial, rosette plant, belonging to the family of *Asteraceae*. It is a variety of a species of thistle widely cultivated in the Mediterranean area as a food (Rottenberg and Zohary, 1996). Artichoke is an important component of the Mediterranean diet and it is rich in bioactive polyphenol compounds (mainly cynarin, luteolin and chlorogenic acid), dietary fibers, vitamins and minerals (Lattanzio *et al*., 2009). Leaves and heads of artichoke have been found to be rich in polyphenolic compounds, inulin, fibre and minerals (Macfarlane *et al*., 2006).The main phenolic compounds are the caffeic acid derivatives which include the caffeoylquinic acid derivatives. Using the International Union of Pure and Applied Chemistry (IUPAC) nomenclature, 5-O-caffeoylquinic

acid (chlorogenic acid) is the most abundant single substance (39%), followed by 1,5-O-dicaffeoylquinic acid (21%) and 3,4-O-dicaffeoylquinic acid (11%), based on total caffeoylquinic acid content. Furthermore, the 1,3-O-dicaffeoylquinic acid (cynarin) content in methanolic extracts of artichoke is very low (about 1.5%), the majority of which is located in the pulp of the leaves, although the dried leaves and stems of artichoke also contain this compound (Lattanzio *et al*., 2009). The artichoke flower heads have a high content of vitamin C in fresh weight (10 mg / 100 g FW) and minerals (K 360 mg / 100 g FW; Ca 50 mg / 100 g FW) (Ceccarelli *et al*., 2010).

Effect of artichoke on liver diseases

Gebhardt, (1997) show that aqueous artichoke extracts reduce lipid peroxidation (measured as production of malondialdehyde) and cytotoxicity (measured as lactate dehydrogenase leakage) in cultures of rat primary hepatocytes exposed to tertbutyl hydroperoxide (*t*-BHP). Furthermore, artichoke extracts prevented the corresponding loss of intracellular glutathione caused by t-BHP, which in turn induces lipid peroxidation. When toxins enter the body from the environment they are either stored in tissues or transported to the liver. In the liver, a complex assortment of enzymes attempts to neutralize and excrete them as bile salts. Detoxification programs often stimulate the mobilization of stored toxins into the blood in which they are then transported to the liver. Bile-stimulating compounds like cynarin then facilitate a quicker elimination of toxin-laden bile into the digestive tract where it can be eliminated in the feces (Kewensis, 1992).The hepatoprotective effects of polyphenolic compounds from Cynara scolymus against CCl_4 toxicity in isolated rat hepatocytes is backed by laboratory trials (Gebhardt, 2002). Among different antioxidant enzymes measured (superoxide dismutase, gluthatione peroxidase, gluthatione reductase, and catalase) in erythrocytes, only gluthatione peroxidase activity was elevated in the artichoke group compared to the control group. 2-Aminoadipic semialdehyde, a protein oxidation biomarker, was decreased in plasma proteins and hemoglobin in the artichoke-fed group versus the control group. In conclusion, the in vitro protective activity of artichoke was confirmed in a rat model

(Jiménez-Escrig *et al*., 2003).Artichoke leaves represent a natural source of phenolic acids with dicaffeoylquinic acids, such as cynarin (1,3-dicaffeoylquinic acid), along with its biosynthetic precursor chlorogenic acid (5-caffeoylquinic acid) as the most abundant molecules. Cynara scolymus leaves extracts have long been used in folk medicine for their choleretic and hepatoprotective activities, that are often related to the cynarin content. These therapeutic properties are also attributed to mono-and di-caffeoylquinic acids and since commercial C. *scolymus* preparations can differ for their activities, we studied four extracts to evaluate, if present, a relationship between the hepatobiliary properties of the different preparations and their content in phenolics **(**Moglia *et al*., 2008).Cynareae tribe present in Egypt are known for their efficacy in relieving some liver disorders (El-Sohafy *et al*., 2013). Studies of the secondary metabolites of Cynara spp. have shown that polyphenolic compounds, mainly caffeic acid derivatives, as well as triterpenoid saponins and flavonoids, play an important biological role in the action of these extracts (Jacociunas *et al*., 2014). Recently, our study indicated that artichoke could be exhibited its therapeutic effects in liver through decreasing the enzymatic activity (ALT, AST, ALP, GGT), serum total billirubin, TC, TG, LDL-c, VLDL-c , creatinine, uric acid, fasting serum glucose and MDA increased total protein, albumin, HDL-c, GST and CAT(Figure1, Abd El-Kereim, 2014).

Milk Thistle

Milk thistle (*Silybum marianum* L. Gaert., Asteraceae) seeds have been used for centuries as herbal medicine mainly for the treatment of liver diseases. The common name, milk thistle, is derived from the 'milky white' veins on the leaves, when broken open, yield a milky sap (Evans, 2002).The dried seeds contain 1-4% silymarin flavonoids. Silymarin is a mixture of at least three isomeric flavonolignans, including silybin (silibinin), silidianin, and silychristin. It is the primary active ingredient in milk thistle, and is also found in related species such as artichokes (Schulz *et al*., 1997). Silibinin is the most biologically active. The seeds also contain other flavonolignans, betaine, apigenin, silybonol, proteins, fixed oil and free fatty acids, which may contribute

to the health giving effects of milk thistle seeds (Evans, 2002).Traditional milk thistle extract is made from the seeds, which contain approximately 4–6% silymarin (Greenlee *et al*., 2007). The extract consists of about 65–80% silymarin (a flavonolignan complex) and 20–35% fatty acids, including linoleic acid. Silymarin is a complex mixture of polyphenolic molecules, including seven closely related flavonolignans (silybin A, s–ilybin B, isosilybin A, isosilybin B, silychristin, isosilychristin, silydianin) and one flavonoid (taxifolin). Silibinin, a semipurified fraction of silymarin, is primarily a mixture of 2 diastereoisomers, silybin A and silybin B, in a roughly 1: 1 ratio (Kroll *et al*., 2007 and Hogan *et al*., 2007).

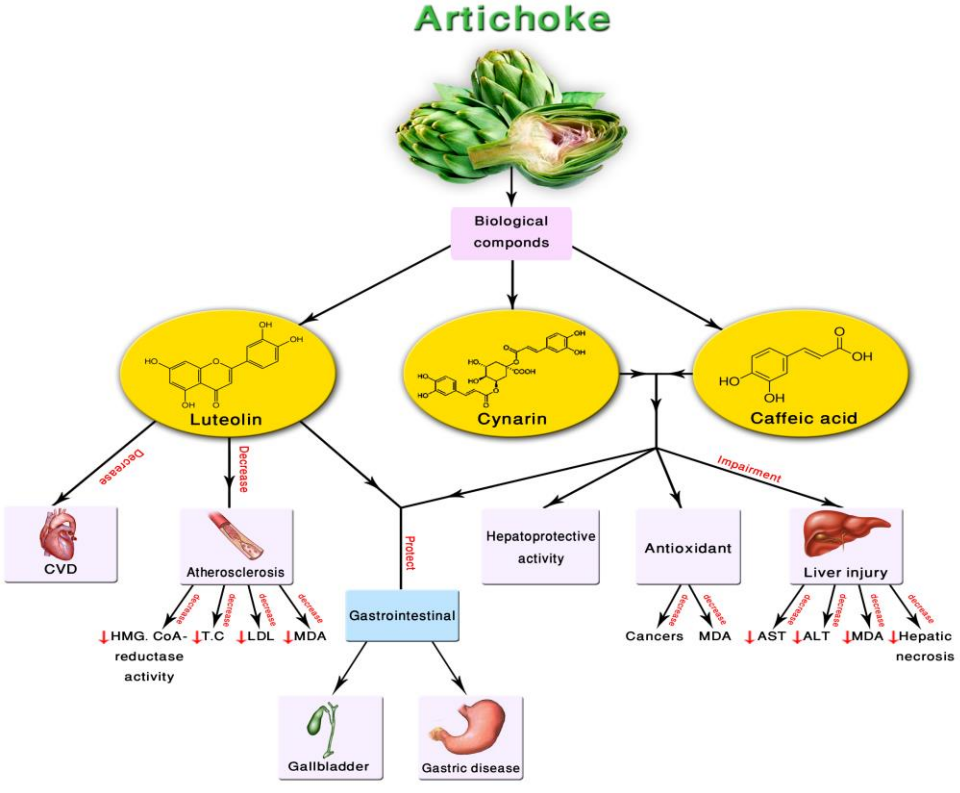

Figure 1. Possible effect of artichoke on liver diseases

Effect of milk thistle on liver diseases

In rats, silymarin and silybin counteracted alcohol toxicity to the liver, as measured by serum gamma glutamyl transpeptidase (GGT), alanine transaminase (ALT), and aspartate transaminase (AST) activity (Miguez *et al*., 1994). In rats with bile duct obstruction, silymarin significantly inhibited hepatic fibrosis (Boigk *et al*., 1997).Milk thistle has been reported to have protective effects on the liver and to greatly improve its function. It is typically used to treat liver cirrhosis, chronic hepatitis (liver inflammation), toxin-induced liver damage (including the prevention of severe liver damage from Amanita phalloides ('death cap'mushroom poisoning), and gallbladder disorders (Tamayo and Diamond, 2007).Silymarin provides heparoprotection against poisoning by halloidin, halothane, thioacetamide, acetaminophen, alcohol, carbon tetrachloride (used widely in the dry-cleaning industry) and the death cap mushroom, which can cause death within a day (Kshirsagar *et al*., 2009).Silibinin treatment has been shown to prevent increases in hepatic enzyme activity and other toxic changes in rats treated with carbon tetrachloride, (Singh *et al*., 2009). In a study evaluating silibinin treatment combined with a hepatotoxic antituberculosis drug, hepatic enzyme activity was significantly decreased (Abenavoli *et al*., 2010).Also has been shown to be protective against radiation-induced hepatic injury, as well as that induced by doxorubicin (Raskovic *et al*., 2011). In addition to being a potent antioxidant, silymarin also has several other actions that make it an important compound for promoting healthy liver function (Loguercio and Festi, 2011). Acute viral hepatitis can be treated with Silymarin (at a dose of 70 mg. three times daily) to lower levels of bilirubin and the transaminases (liver enzymes) (Fraschini *et al*., 2002).In vitro study performed using human hepatocytes, silibinin completely prevented ethanol-induced release of lactate dehydrogenase (Van Pelt *et al*., 2003). Silibinin is well tolerated in human subjects with chronic hepatitis C infection (Gordon *et al*., 2006).Silibinin currently is recommended for use in alcoholic liver disease. Ethanol induces free radical formation through multiple pathways, resulting in steatohepatitis and cirrhosis with chronic use (Saller *et al*., 2008).In patients with chronic hepatitis, silibinin decreases transaminase activity caused by hepatic damage and decreases serum

malondialdehyde concentrations, a marker of oxidative injury. Nonalcoholic fatty liver disease occurs because of metabolic stress or can accompany primary hepatitis, promoting progression of inflammatory disease to fibrosis (Loguercio and Festi, 2011). In a placebo-controlled trial, patients with cirrhosis consuming silibinin had greater total glutathione concentrations and concurrent decreases in Nterminal propeptide of type III collagen, a biomarker for hepatic fibrosis (Lucena *et al*., 2002). Fraschini *et al*. (2002) found that silymarin (at a dose of 140 mg. three times daily), can significantly reduce patient mortality in alcohol-induced liver cirrhosis. Kidd and Head, (2005) found that use of silibinin in hepatic cirrhosis results in improvement in antioxidant status, cytoprotection, reversal of fibrosis, and regeneration. Dose-dependent decreases in hepatic enzyme activity with silibinin treatment. Decreased mortality rates have been documented with silibinin use in randomized controlled trials performed in patients with cirrhosis.90All-cause mortality decreased 4.4% and mortality from liver causes decreased 7.3% in cirrhotic patients (Saller *et al*., 2008).Liver fibrosis can result in remodeling of liver architecture leading to hepatic insufficiency, portal hypotension and hepatic encephalopathy. The conversion of hepatic stellate cells into yofibroblast is condidered as central event in fibrogenesis. Sliymarin treatment markedly inhibits this process in liver fibrosis patients showing antifibrotic potential (Kshirsagar *et al*., 2009). Numerous clinical trials have shown that silymarin protects the liver against various toxins and boasts another remarkable property: namely, by encouraging the regeneration of liver cells, it can actually reverse the damage and help cure the liver. It does this by stimulating protein synthesis [RNA polymerase A], thus activating the liver's ability to regenerate itself through increased formation of new liver cells called hepatocytes (Fraschini *et al*., 2002). Silymarin stimulates liver tissue regeneration by increasing protein synthesis in the injured liver. In vivo and in vitro experiments performed in the liver of rats from which part of the organ (liver) was removed, silibinin produces a significant increase in the formation of ribosomes and in DNA synthesis, as well as an increase in protein synthesis. Interestingly, the increase in protein synthesis is induced by silibinin only in injured livers, not in healthy ones

(Kshirsagar *et al*., 2009). Recently, our study indicated that milk thistle could be exhibited its therapeutic effects in liver through decreasing the enzymatic activity (ALT, AST, ALP, GGT), serum total billirubin, TC, TG, LDL-c, VLDL-c , creatinine, uric acid, fasting serum glucose and MDA increased total protein, albumin, HDL-c, GST and CAT (Figure 2, Abd El-Kereim, 2014).

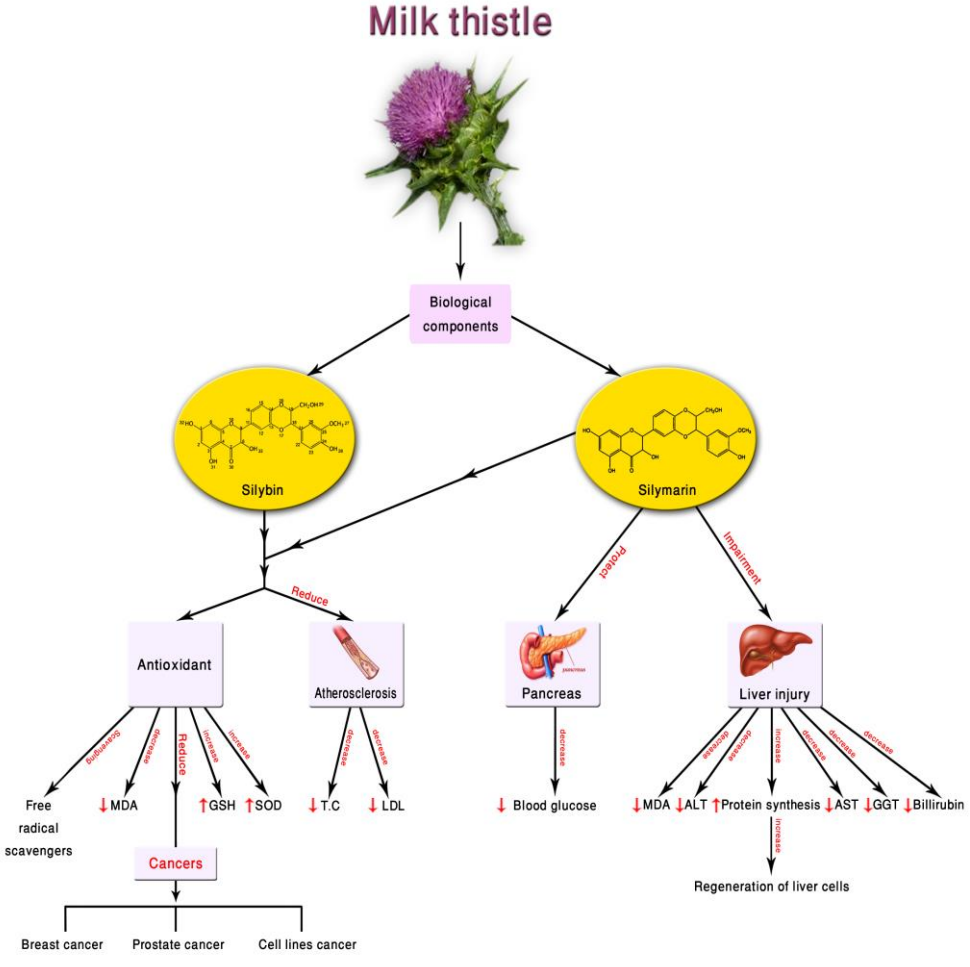

Figure 2. Possible effect of milk thistle on liver diseases

Turmeric

Turmeric (*Curcuma longa*) is a rhizomatous herbaceous perennial plant of the ginger family, *Zingiberaceae*. Turmeric is a perennial herbaceous plant, which reaches up to 1 m tall. Highly branched, yellow to orange, cylindrical, aromatic rhizomes are found (Chan *et al*., 2009). Turmeric contains protein (6.3 %), fat (5.1 %), minerals (3.5 %), carbohydrates (69.4 %) and moisture (13.1 %), the essential oil (5.8 %) obtained by steam distillation of rhizomes has α – phellandrene (1 %) sabinene (0.6 %), cineol (1 %), borneol (0.5 %) zingiberene (25 %) and sesquiterpines (53 %), (Kapoor, 1990). Turmeric rhizomes or root include volatiles and non-volatiles compounds. The non-volatiles compounds of turmeric are the coloring agent and are found to be a rich source of polyphenolic curcuminoids which include: curcumin (diferuloylmethan), demethoxycurcumin, bisdemethoxycurcumin, and cyclocurcumin. The yellow-pigmented curcuminoids represent 2% -5% of the root, typically composed of 85% as curcumin, 10% as demthoxycurcumin and 5% as disdemethoxycurcumin (Jurenka, 2009).

Effect of turmeric on liver diseases

Turmeric has been found to have a hepatoprotective characteristic similar to silymarin. Animal studies have demonstrated turmeric's hepatoprotective effects from a variety of hepatotoxic insults, including carbon tetrachloride (CCl_4), galactosamine, acetaminophen (paracetamol), and Aspergillus aflatoxin (Deshpande *et al*., 1998). Turmeric's hepatoprotective effect is mainly a result of its antioxidant properties, as well as its ability to decrease the formation of pro-inflammatory cytokines. In rats with CCl_4-induced acute and subacute liver injury, curcumin administration significantly decreased liver injury in test animals compared to controls. Turmeric and curcumin also reversed biliary hyperplasia, fatty changes, and necrosis induced by aflatoxin production. Sodium curcuminate, a salt of curcumin, also exerts choleretic effects by increasing biliary excretion of bile salts, cholesterol, and bilirubin, as well as increasing bile solubility, therefore possibly preventing and treating cholelithiasis (Park *et al*., 2000).Curcumin also protects against diethylene to indomethacin and salieylate, has recently been reported (Ishita *et al.,* 2004). Sugiyama *et*

al., (2006) reported that cyte enzyme inactivation in rat liver caused by CCl_4 was inhibited by curcumin. Dietary intake of curcumin may protect against CCl_4–induced hepatic cyte inactivation via its antioxidant properties, without inducing hepatic cyte. Curcumin inhibited the development of TAA (thioacetamide) induced liver cirrhosis mainly due to its anti – inflammatory activities and not by a direct anti – fibrotic effect. As curcumin ingestion is safe in humans, it may be reasonable to assess in clinical studies the beneficial effect of curcumin in slowing the development of liver cirrhosis (Bruck *et al.,* 2007). Curcumin restored the level of biomarker parameters such as AST, ALT and ALP back to normal (Swamy *et al*., 2012). A recent report shows that curcumin suppresses HBV gene expression. It has been further suggested that the combination of curcumin with nucleotide/nucleoside analog can synergistically suppress HBV replication and the risk of escape mutants emerging may be prevented (Rechtman *et al*., 2010). A recent report shows that curcumin inhibits hepatitis C virus replication by suppressing PI3K/Akt-SREBP-1 pathway. It can also be concluded that curcumin may decrease the risk of hepatitis C virus-related hepatocellular carcinoma (HC) through its protective role against hepatitis C virus infection **(**Kim *et al*., 2010). The protective action of curcumin against experimental models of alcoholic liver diseases has been reported previously. It was shown that treatment with dietary curcumin reduced fatty liver, necrosis, and inflammation. Curcumin is also known to inhibit oxidative stress and lipid peroxidation, activation (Nanji *et al*., 1999). Animal studies have further shown that curcumin decreases the ethanol induced increase in malondialdehyde, decreases the levels of LDH and aspartate aminotransferase (AST), and increases the GSH levels (Bao *et al*., 2010). Curcumin inhibits oxidative stress via modification of mitochondrial reactive oxygen species and 8-OH deoxyguanosine levels and modify the abnormal increase in the level of aminotransferases (Ramirez-Tortosa *et al*., 2009).Moreover, curcumin inhibits reactive oxygen species production in myofibroblastic hepatic stellate cells and modifies secretion of tissue inhibitor of metalloprotease-1. In conclusion, curcumin effectively mitigates nonalcoholic fatty liver diseases via its antioxidant and anti-inflammatory actions (Vizzutti *et al*., 2009). Recently, our study

indicated that turmeric could be exhibited its therapeutic effects in liver through decreasing the enzymatic activity (ALT, AST, ALP, GGT), serum total billirubin, TC, TG, LDL-c, VLDL-c , creatinine, uric acid, fasting serum glucose and MDA increased total protein, albumin, HDL-c, GST and CAT (Figure 3, Abd El-Kereim, 2014).

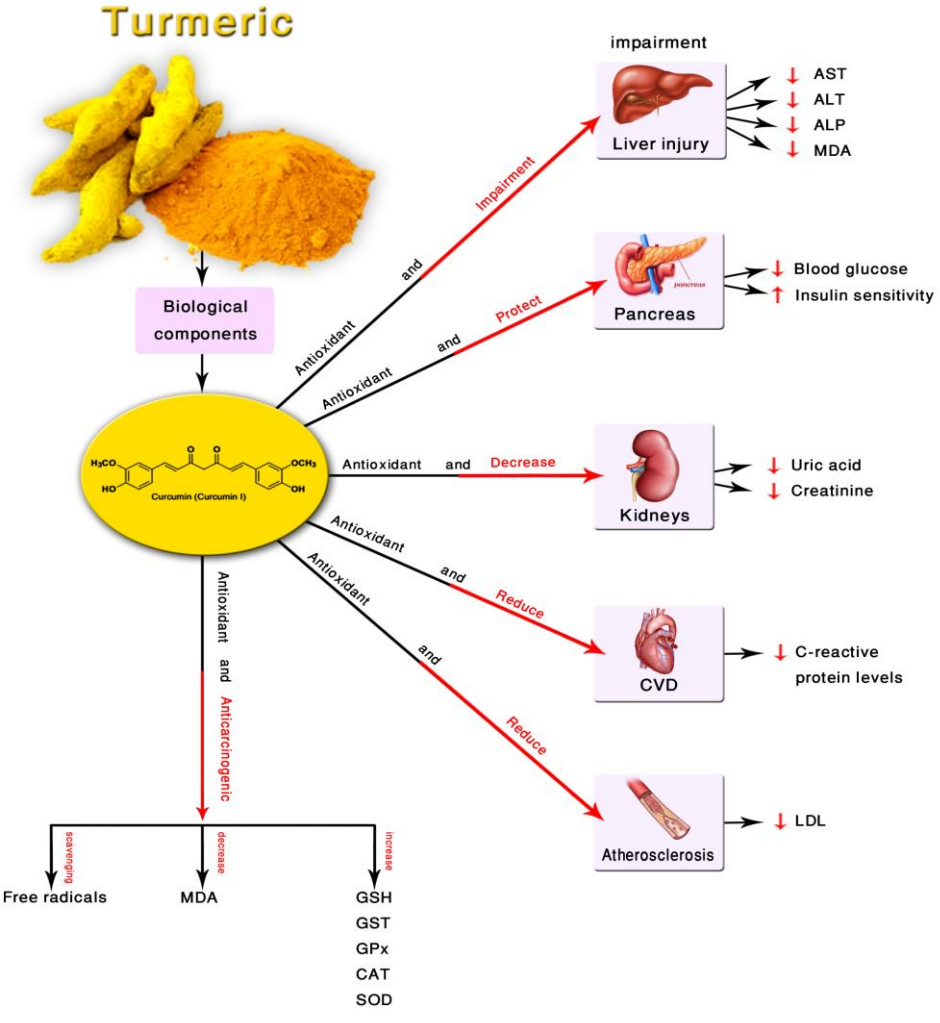

Figure 3. Possible effect of turmeric on liver diseases

Gum Arabic

Common Name(s):Gum arabic, also known as acacia gum, chaar gund, char goond, or meska, Scientific Name(s): *Acacia senegal* (L.) Willd. Family: *Leguminosae*/ *Fabaceae*- Mimosoideae **.** Gum Arabic is defined by the FAO/WHO Joint Expert Committee for Food Additives (JECFA) as a dried exudation obtained from Senegalia (Acacia) senegal and Vachellia (Acacia) seyal (FAO, 1999).Gum Arabic is an edible, dried, gummy exudate from the stems and branches of Acacia Senegal and A. Seyal that is rich in non-viscous soluble fiber (Williams and Phillips, 2000). The chemical composition of GA can vary with its source, the age of the trees from which it was obtained, climatic conditions and soil environment (Idris *et al*., 1998). The gum has a high molecular weight (approx. 350-850 kDa) and has galactose (44 %), rhamnose (13 %), glucuronic acid (16 %) and arabinose (27 %) residues, but also minerals like potassium, magnesium and calcium (Ali *et al*., 2008). GA is a branched-chain, complex polysaccharide, either neutral or slightly acidic, found as a mixed calcium, magnesium and potassium salt of a polysaccharidic acid (arabic acid). The backbone is composed of 1,3-linked b-D-galactopyranosyl units. The side chains are composed of two to five 1,3-linked b-D-galactopyranosyl units, joined to the main chain by 1,6-linkages. Both the main and the side chains contain units of a-L-arabinofuranosyl,a-L-rhamnopyranosyl, b-D-glucuronopyranosyl and 4-O-methyl-b-D-glucuronopyranosyl, the last two mostly as end units (Verbeken *et al*., 2003).

Effect of GA on chronic liver disease:

Fujiwara *et al*. (1995) inferred that such effects of GA would merit consideration in the therapy for chronic liver disease, as deranged function of Kupffer cells (KC) and hepatic macrophages occurs in this disease and is involved in its complications, such as endotoxemia because of GA has free radical (nitric oxide) scavenging properties and macrophage inhibition functions. Macrophages play an important role in the regulation of immunological process in rats. Mochida *et al*. (1996) studied the effect of GA on macrophage activation by their ability to produce superoxide anions in vitro, and found that GA was found to

blocking function hepatic macrophage to prevent release nitric oxide in vitro. Such effects of Arabic gum would merit consideration in the therapy for chronic liver disease, as deranged function of Kupffer cells and hepatic macrophages occurs in this disease and is involved in its complications, such as endotoxemia. Rehman *et al*. (2001) suggest that GA reduce the damage of hepatic tissue take place due to have ability to scavenging nitric oxide in order to blocking oxidative stress. Gum Arabic is considered as acytoprotetive agent have demonstrated its usefulness agent a number of drugs like cisplatin induced nephrotoxicity and ccl4 induced hepatoxicity in mice (Al-Majed *et al*., 2003). Gamal El-din *et al*., (2003) examine the effects of arabic gum (AG), which is commonly used in processed foods, on acetaminophen-induced hepatotoxicity in mice. Mice were given arabic gum orally (100 g l^{-1}) 5 days before a hepatotoxic dose of acetaminophen (500 mg kg^{-1}) intraperitoneally. Arabic gum administration dramatically reduced acetaminophen-induced hepatotoxicity as evidenced by reduced serum alanine (ALT) and aspartate aminotransferase (AST) activities. Acetaminophen-induced hepatic lipid peroxidation was reduced significantly by arabic gum pretreatment. The protection offered by arabic gum does not appear to be caused by a decrease in the formation of toxic acetaminophen metabolites, which consumes glutathione, because arabic gum did not alter acetaminophen-induced hepatic glutathione depletion. Acetaminophen increased nitric oxide synthesis as measured by serum nitrate plus nitrite at 4 and 6 h after administration and arabic gum pretreatment significantly reduced their formation. GA may induce the immune system of liver (innate and adaptive) particularly KC for clearance of particulates and soluble molecules and also induction of tolerance to food antigens derived from gastrointestinal tracts. For this tolerance, including apoptosis (Thames, 2004). So GA has strong antioxidant properties and major mechanism for the induction of these toxicities is the generation of free radicals (Ali *et al*., 2008). Al-Kenanny *et al*., (2012) studied that received of GA orally to mice at concentration 10gm /kg/for eight days, have ability as antioxidant because have a significantly ameliorating hepatotoxicity by increase the level of GSH and reduction MDA in addition to enzymatic level ALT and AST in serum

of mice although it haven't reach to normal value. Rishi *et al*. (2014) indicate that the hydro alcoholic extract of Acacia senegal has significant hepatoprotective activity. This may be probably due to the higher content of flavonoids. The earlier investigators have screened the hepatoprotective activity of the flavonoids, which is also claimed to have free radical scavenging property and it inhibits the lipid peroxidation against CCl_4 induced hepatic toxicity. Antioxidant potential, free radical scavenging and excellent total antioxidant capacity of Acacia senegal pod-HA in-vitro system and screening of the pharmacological action against the liver damage is being investigated. Recently, our study indicated that gum arabic could be exhibited its therapeutic effects in liver through decreasing the enzymatic activity (ALT, AST, ALP, GGT), serum total billirubin, TC, TG, LDL-c, VLDL-c , creatinine, uric acid, fasting serum glucose and MDA increased total protein, albumin, HDL-c and antioxidant enzymes activities (GST &CAT) (Figure 4, Abd El-Kereim, 2014).

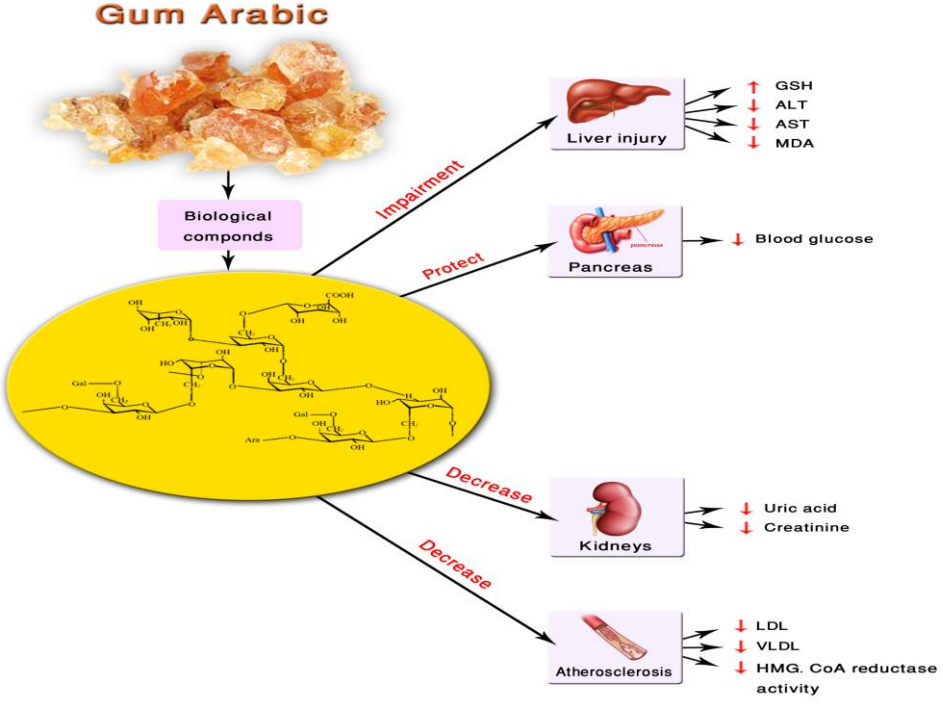

Figure 4. Possible effect of gum Arabic (GA) on liver diseases

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تأثير الأغذية الوظيفية على معالجة إضطرابات الكبد : دراسة مرجعية

يوسف عبد العزيز الحسانين ، مروة كمال

قسم التغذية وعلوم الأطعمة- كلية الاقتصاد المنزلى- جامعة المنوفية – شبين الكوم - مصر

تعرف الأغذية الوظيفية على أنها المواد الغذائية التي تقدم فوائد صحية بجانب التغذية الأساسية، لذلك يمكن أن تشتمل تلك جميع الأغذية أو الأغذية ذات المحتوى الطبيعي أو المدعمة بمواد غذائية أو مواد نشطة بيولوجيا والتي تمدنا بفوائد صحية خاصة. وقد أثبتت دراسات وبائية عشوائية أجريت في بلدان مختلفة الآثار الصحية المرتبطة باستهلاك الأغذية الوظيفية , مثل الحد من مخاطر الإصابة بالسرطان وتحسين صحة القلب وتحفيز جهاز المناعة وتقليل أعراض انقطاع الطمث وتحسين صحة الجهاز الهضمي و الحفاظ على صحة الجهاز البولي والتأثيرات المضادة للالتهابات وتقليل ضغط الدم المرتفع والحفاظ على الرؤية والأنشطة المضادة للبكتريا والفيروسات وتقليل هشاشة العظام والتأثير المضاد للسمنة. لذلك تهدف هذه الدراسة إلى دراسة تأثير أجزاء بعض النباتات تشمل الخرشوف, شوكة الجمل ,الكركم والصمغ العربي كأغذية وظيفية فى الوقاية وعلاج بعض الأمراض.

الكلمات المفتاحية: الأغذية الوظيفية – الخرشوف - شوكة الجمل- الكركم -الصمغ العربي- نشاط الانزيمات المضادة للأكسدة