

Effect of Some Herbal Plants on Liver Function of Rats Treated with Trichloroethylene

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Abstract

Background & Aims: Curcumin (CCM), Cinnamon (CNN) and Ginger (GE) had been considered to possess antioxidant activities. This study aimed to investigate their protection effect against trichloroethylene (TCE)-induced hepatotoxicity and to demonstrate its possible mode of action.

Methods: Rats were fed CCM, CNN and GE singly with or without 5mg/l of TCE in drinking water. The rat were killed after treatment period of 8 weeks, and the serum levels of alanine aminotransferase (ALAT), aspartate aminotransferase (ASAT), bilirubin, proteins, glucose, albumin and triglycerides were determined.

Results: TCE- receiving rat exhibited significant changes in biomarkers of liver function to indicate liver injury. The CCM containing diet significantly ameliorated the serum aminotransferases, especially ALAT, total protein and albumin. The CNN containing diet significantly ameliorated the total protein and albumin. The GE containing diet significantly ameliorated the serum aminotransferases, especially ALAT.

In conclusion, this work indicated that CCM is the most herbal plant which can protected and alleviated the liver function.

Key Words: Trichloroethylene - Curcumin - Cinnamon - Ginger - Rats

Introduction

Nowadays, there is an increasing interest in the protective function of dietary antioxidants, which play an important role in the protection of liver. CCM (diferuloyl methane) is a phenolic compound present in large quantities in the root of plant *curcuma longa*. It has been widely used as a spice and coloring agent in food. Recently, CCM has been considered to possess anti-inflammatory and antioxidant activities (Anto *et al.*, 2000). The ability of CCM to prevent tumor formation in the skin , forestomach, duodenum, and colon of mice and in the tongue, colon, mammary glands, and sebaceous glands of rats has been well

documented (sharma *et al.*, 2001).CCM has been also shown to inhibit lipid peroxidation caused by many toxic agents in hepatocytes either *in vitro or in vivo* (Ramirez Bosca *et al.*,1995; Devasena *et al.*, 2002). On the other hand, no treatment-related toxicity was observed up to an oral dose of 8000 mg/day for 3 month in mice (Chuang *et al.*, 2000). This non toxic nature of CCM, as well as its multiple beneficial clinical effects, has made it one of the most attractive antioxidants.

CNN (Cinnamon), also known by Cassia, Sweet Wood, and Gui Zhi, is traditionally harvested in Asian

countries (Leung, 1996; Toriizuka, 1998). Many studies have shown the diverse biological functions of cinnamon including anti-diabetic effects (Qin *et al.*, 2003), anti-inflammatory (Lee *et al.*, 2005), An antitumor effect of cinnamon was previously suggested *in vitro* (Schoene *et al.*, 2005) without *in vivo* evidence or a working mechanism, and anti-microbial (Matan *et al.*, 2006) and anti-oxidant (Singh *et al.*, 2007).

In addition, Phenol compounds present in (cinnamon) spices that show natural anti-oxidant properties have been studied for substitution of synthetic anti-oxidants, due to possible side effects of synthetic anti-oxidants which may in some circumstances act deleterious to animal organisms (Pratt, 1992).

GE (Ginger), is one of the most widely used spices around the world and is a common condiment for a variety of compounded foods and beverages (Gupta, 2008). Ginger (*Zingiber officinale Roscoe*) has been used as a spice for over 2000 years (Bartley and Jacobs, 2000). Recently, ginger has received increasing attention because of its pronounced anti-oxidant (El- Ghorab *et al.*, 2010), anti-inflammatory (Minghetti *et al.*, 2007), anti-diabetic (Afshari *et al.*, 2007) anticancer activities (Shukla and Singh, 2007), anti-emetic, anxyolytic, anti-thrombotic, anti-pyretic, analgesic, and a treatment for toothache, insomnia, baldness, urinary tract infections, and as therapy for various gastrointestinal disorders (Balch, 1996), arthritis, rheumatism (Dedov *et al.*, 2002; Wang and Wang, 2005; Tapsell *et al.*, 2006), sprains, muscular aches, pains, sore throats, cramps, constipation, indigestion, vomiting, hypertension, dementia, fever, infectious diseases and helminthiasis (Ali *et al.*, 2008), and cardiovascular effects (Kaul and Joshi, 2001; Afzal *et al.*, 2001).

The present study was thus designed to investigate the protective

activity of CCM, CNN and GE against TCE-induced liver injury.

Selected routine work biochemical markers of liver function tests were chosen to express the effect.

Material and methods

Chemicals: Pure TCE was purchased from Sigma Chem. Co.(St. Louis, MO, USA). Powdered CCM, CNN and GE was purchased from Libya spice market. Kits for biochemical measurements were supplied from Randox Laboratories Ltd., UK.

Diets: Standard diet was prepared from Casein (20%), Starch (32%), Sucrose(33%), Cellulose(5%) , Corn oil(5%) and Vitamin / Mineral(5%) ,(Abd-Allah ,2003).

Animals and Treatments:

Twenty five male albino Wistar rats (weight range 58 – 160 g) were used for the experimental study. Animals were obtained from hellwan animal station, Ministry of Heath, Egypt . The experimental rats were housed in the animal house in zoology Department, Faculty of Science, Damietta Branch, Mansoura University, New Damietta, Egypt . They were divided into 8 groups of 5 rats each.

Group 1: (Normal) rats were given basal diet and water for 8 weeks.

Group 2: (Trichloroethylene) rats were given 5% TCE in drinking water for 8 Weeks.

Group 3: (Curcumin) rats were given Curcumin at 1g /kg diet for 8 weeks.

Group 4:(Trichloroethylene + Curcumin) rats were given 5% Trichloroethylene in drinking water and Curcumin at 1g /kg diet for 8 weeks.

Group 5:(Cinnamon) rats were given Cinnamon at 1g /kg diet for 8 weeks.

Group 6:(Trichloroethylene + Cinnamon) rats were given 5% Trichloroethylene in drinking water and Cinnamon at 1g /kg diet for 8 weeks.

Group 7 : (Ginger) rats were given Ginger at 1g /kg diet for 8 weeks.

Group 8 : (Trichloroethylene + Ginger) rats were given 5% Trichloroethylene in drinking water and Ginger at 1g /kg diet for 8 weeks.

Sampling and Measurements:

After 8 weeks of treatments, rats were killed by Anesthesia and the blood was withdrawn by syringe in to blood centrifuge tubes. Blood collected was allowed to coagulate at 37c° for 30 minutes. Serum was separated by centrifugation at 4000 rpm and used for the estimations of ALAT(Reitman and Frankel 1957), ASAT(Reitman and Frankel 1957) , total Bilirubin (Walloy and Gerade 1970), albumin (Doumas *et al.*, 1971), triglycerides (Vassault(1986), total protein (Gornal *et al.*,1949) and Glucose Trinder-reaction (1969)..

Statistics: Student's *t* test was used for statistical analysis of the data. The significant levels were considered as: $p \leq 0.05$, $p \leq 0.01$ and $p \leq 0.001$, (Millar 2001).

Results

Serum Aminotransferases:

Table (1) presents the results of serum ALAT and ASAT of rats treated with TCE with or without plant herbs.

Means of serum ALAT and serum ASAT were significantly higher in rats treated with TCE than that of normal animals. On the other, serum ALAT and ASAT did not changed in animals fed on herbs alone than that of normal ones.

On the effect of herbs the mean of serum ALAT was completely improved in rats treated with TCE /CCM, but no changes were observed with CNN and GE. No alleviation was resulted in serum ASAT of rats fed on all expremintel herbs.

Liver function tests:

Table (2) presents the results of serum concentrations of total protein, albumin

and bilirubin of rats treated with TCE with or without plant herbs.

Means of serum total protein was significantly lower in rats treated with TCE than that of normal animals. On the other, serum total protein did not changed in animals fed on CCM and CNN alone than that of normal ones. On adding of Herbal plants to TCE treatment serum total protein was partially ameliorated with CCM and CNN .On the other hand, no significant changes were observed in rats treated with TCE/GE.

In similar, means of serum albumin was significantly lower in rats treated with TCE than that of normal animals. On the other, serum albumin did not changed in animals fed on CCM alone than that of normal ones. On adding of herbal plants to TCE treatment serum albumin was partially ameliorated with CCM and GE . On the other hand, no significant changes were observed in rats treated with TCE/CNN.

Means of serum bilirubin was significantly higher in rats treated with TCE than that of normal animals. On the other, serum bilirubin did not changed in animals fed on herbs alone than that of normal ones. On adding of herbal plants to TCE treatment serum bilirubin was partially ameliorated with CCM and CNN . On the contrary, no improvement was observed on adding GE to TCE treatment.

Serum Metabolites:

Table (3) presents the results of serum concentrations of Triglycerides and Glucose of rats treated with TCE with or without plant herbs.

Means of serum triglycerides and serum glucose were significantly higher in rats treated with TCE than that of normal animals. On the other, serum triglycerides and glucose did not changed in animals fed on herbs alone than that of normal ones.

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On adding herbs to diet the mean of serum triglycerides was partially ameliorated in rats treated with TCE /CCM, but no changes were observed with

CNN and GE. No alleviation was resulted in serum Glucose of rats fed on all tested herbs.

Table (1): Activities of serum aminotransferases in rats treated with CCM, CNN and GE with TCE.

Parameters	C	TCE	CCM	TCE+CCM	CNN	TCE+CNN	±GE	TCE+GE
Alanine aminotransferase(IU/I)	16.0± 2.12	28.6± 2.19 **	15.4± 1.34 NS	16.6± 1.34 ***	15.4± 1.34 NS	29.4± 2.19 ***	15.4± 2.50 NS	23.8± 1.78 **
Mean ± SD P-value vs. control P-value vs. TCE		**	NS	***	NS	***	NS	**
Aspartate aminotransferase(IU/I)	16.0± 2.12	32.2± 3.83 **	15.4± 1.34 NS	32.2± 3.83 ***	17.2± 1.64 NS	34.0± 2.74 ***	19.4± 3.51 NS	29.4± 2.19 **
Mean ± SD P-value vs. control P-value vs. TCE		**	NS	***	NS	***	NS	**

Number of animals per group=5 *= significant , **and***= significant higher , ****= significant very higher , NS = Non- significant value

Table (2): Serum Concentrations of serum metabolites in rats treated with CCM, CNN and GE with TCE.

Parameters	C	TCE	CCM	TCE+CCM	CNN	TCE+CNN	GE	TCE+GE
Total protein (g/dl)	5.82± 0.26	5.46± 0.15 *	7.01± 0.65 *	6.23± 0.18 * **	6.21± 0.43 NS	6.68± 0.66 * **	5.36± 0.70 *	5.42± 0.49 NS
Mean ± SD P-value vs. control P-value vs. TCE		*	*	**	NS	**	*	
Albumin (g/dl)	3.75± 0.01	3.13± 0.02 ****	3.83± 0.03 **	3.59± 0.03 *** ****	3.38± 0.10 ***	3.18± 0.03 **** *	3.17± 0.02 ****	3.38± 0.02 **** ****
Mean ± SD P-value vs. control P-value vs. TCE		****	**	****	***	*	****	****
Bilirubin (mg/dl)	0.22± 0.06	0.65± 0.11 **	0.26± 0.02 NS	0.63± 0.03 ****	0.31± 0.05 *	0.58± 0.04 ****	0.25± 0.04 NS	0.74± 0.14 **
Mean ± SD P-value vs. control P-value vs. TCE		**	NS	****	*	****	NS	**

Number of animals per group=5 *= significant , **and***= significant higher , ****= significant very higher , NS = Non- significant value

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Table (3): Serum Concentrations of serum metabolites in rats treated with CCM, CNN and GE with TCE.

Parameters	C	TCE	CCM	TCE+CCM	CNN	TCE+CNN	GE	TCE+GE
Triglycerides (g/dl)	70.5	180.4	65.6	159.1± 11.96	73.6±	184.4±	70.9±	178.5±
Mean ± SD	±4.66	±18.48	±7.05	***	8.96	10.56	9.37	3.12
P-value vs. control		***	*	*	NS	****	NS	****
P-value vs. TCE								
Glucose (mg/dl)	56.07±	102.79±	52.41±	107.24± 1.40	55.66±	104.24±	55.24±	105.08±
Mean ± SD	0.56	2.77	0.68	****	1.09	2.43	0.69	1.29
P-value vs. control		****	***	*	NS	****	NS	****
P-value vs. TCE						*		

Number of animals per group=5 * = significant , **and*** = significant higher , **** = significant very higher , NS = Non-significant value

Discussion

The toxicity of TCE is dependent on its metabolism, which occurs by either cytochrome p-450 (p-450) –dependent oxidation or glutathione (GSH) conjugation. Metabolites derived from p-450 metabolism, including trichloroacetate (TCA) and dichloroacetate (DCA), have been associated with the hepatotoxicity (Davidson and Beliles, 1991; Lash et al., 1999).

The effect of TCE was agreed to that reported by (Maiti *et al.*, 2004; Maiti *et al.*, 2005 and Beppu *et al.*, 2006). Many medicinal plants are used today in therapy of different diseases (Mascolo *et al.*, 1989).

Curcumin Over the years, a number of studies have tried addressing the pharmacokinetics of that is poorly absorbed from intestine after oral administration of different doses of 3H-**curcumin** in rats (Ravindranath and Chandrasekhara, 1980, Ravindranath and Chandrasekhara, 1981 and Ravindranath and Chandrasekhara, 1982). It was shown that oral consumption of **curcumin** in rats resulted in approximately 75% being

excreted in the feces and only traces appeared in the urine (Wahlstrom and Blennow, 1978), converted to monoglucuronide conjugates (Pan *et al.*, 1999).

In the present work, adding CCM to TCE treated rats was completely improved the results of ALAT and albumin, but the other parameters, total protein, bilirubin and Triglycerides, were partially alleviated. These results are more or less agreed to that obtained by Abd-Allah (2003). Because the measurement of serum ALAT and albumin are more from the intestine. Numerous studies have suggested presence of different metabolites of **curcumin**. It has been shown to be bio-transformed to 11% found in bile (Holder *et al.*, 1978) ,suggesting poor absorption of **curcumin** of fecal excretion of **curcumin** with only dihydrocurcumin and tetrahydrocurcumin. the absolute abolishment of their increase closely indicate the ability of CCM to minimize the TCE-induced liver injury in the present study. It may be argued that the ability of CCM to scavenge free radicals

might have been responsible for its antioxidative activity. Moreover, it is also with the hepatic metabolism of TCE though the modulation of liver drug metabolizing enzymes involved in its biotransformation, i.e cytochrome P450 and glutathione-s- transferase (Sharma *et al.*, 2001; Thapliyal and Maru, 2001).

Similarly, the interaction between TCE and CCM effects on the metabolism of proteins, carbohydrates and lipid may be attributed to the same issue or to the lipid-lowering potent activity *in vivo* by the dietary curcuminoid (Asia and Miyazawa, 2001) However, this point is still unclear and needs further investigations to be resolved and cleared up.

CNN is a popular flavoring ingredient, widely used in food products. It has exhibited beneficial properties to health, such as antimicrobial activity, for controlling glucose intolerance and diabetes, inhibiting the proliferation of various cancer cell lines, and for treating the common cold (Anderson & Broadhurst, 2004; Murcia *et al.*, 2004). CNN extracts can reduce lipid peroxidation in the (3-carotene-linoleic acid system (Mancini-Filho & Van-Koij, 1998). CNN extracts exhibit a protective capacity against irradiation induced lipid peroxidation in liposomes, and quench hydroxyl radicals and hydrogen peroxide (Murcia *et al.*, 2004). Extracts on lard and vegetable oils demonstrated that they could stabilize lard against oxidation and showed antioxidative properties when tested on vegetable oils during storage or frying conditions (Vekiari, & Thomopoulos, 1993).

In the present work ,CNN was partially ameliorated the effect of TCE on the liver most selected biochemical markers, although total protein was showed completely improvement. Moselhy and Junbi (2010) observed that the elevated serum ASAT and ALAT enzymatic activities induced by CCl₄ were restored

possible that CCM might have interfered towards normalization significantly by orally administrated 200 mg/kg CNN of either extracts once daily for 7 days as compared to non treated rats. The results obtained indicated that CNN extract have potent hepatoprotective action against CCl₄ by lowering the MDA level and elevate the antioxidants enzymes activities. GE (Zingiber officinale Roscoe) is example of botanicals which is gaining popularity amongst modern physicians and its underground rhizomes are the medicinally useful part (Mascolo *et al.*, 1989). Many studies were carried out on ginger and its pungent constituents, fresh and dried rhizome. Among the pharmacological effects demonstrated is antiplatelet, antioxidant, anti-tumour, anti-rhinoviral, antihepatotoxicity immunomodulatory, anti-tumorigenic, anti-inflammatory, anti-apoptotic, anti-hyperglycemic, anti-lipidemic anti-emetic actions and anti arthritic effect (Fisher-Rasmussen *et al.*, 1991; Sharma *et al.*, 1994 and Kamtchouing *et al.*, 2002). Ginger is a strong anti-oxidant substance and may either mitigate or prevent generation of free radicals. It is considered a safe herbal medicine with only few and insignificant adverse/side effects (Ali *et al.*, 2007).

In the present work ,GE is partially ameliorated ALAT, ASAT, Triglycerides and albumin, but other biochemical markers did not showed changes.

There results are similar to the result of Sakr (2007), who observed that the Treating animals with ginger led to significant decrease in ALT and AST activity in albino rats. El-Sharaky et al., (2009) reported that the results showed significant lowering of serum ASAT, ALAT that Ginger extract was found to have a protective effect on CCl₄ and acetaminophen-induced damage as confirmed by histopathological

examination of the liver in rats (Yemitan and Izeqbu, 2006).

In conclusion, the present results showed that selected plant herbs induced either complete improvement or partial

amelioration on the effect of TCE on the liver. By comparison the CCM was the most potent whit normalize of most tested biochemical markers.

References

- Anto R J, Maliekal T T, and Karunagaran D (2000):** 1- 929 Cells harboring ectopically expressed relA resist curcumin-induced apoptosis. *J. Biol. Chem.*, 275 (21): 15601-15604.
- Sharma R A, Ireson C R, Verschoyle R D, Hill K A, Williams M L, Leuratti C, Manson M M, Marnett L J, Steward W P, and Gescher A (2001):** Effects of dietary curcumin on glutathione S-transferase and malondialdehyde-DNA adducts in rat liver and colon mucosa: relationship with drug levels. *Gin.Cancer Res.*, 7: 1452-8.
- Devasena T, Rajasekaran K N, and Menon P V (2002):** Bis- 1,7- (2-hydroxyphenyl)-hepta-1,6-diene -3,5-Dione (a curcumin analog) ameliorates dmh-induced hepatic oxidative stress during colon carcinogenesis. *Pharmacol.Res.*, 46 (1): 39-45.
- Chuang S E, Kuo M I, Hsu C H, Chen Cr, Lin L k, Lai G M, Hsieh C Y, and Cheng A L (2000):** Curcumin-containing diet inhibits diethylnitrosamine-induced murine hepatocarcinogenesis. *Carcinogenesis.*, 21(2): 331-335.
- Pratt D E (1992):** Natural antioxidants from plant material. In: HO, C.I., Lee, C.Y., Huang, M.I. (Eds.), Phenolic Compounds in Food and their Effects on Health, Vol. 507. ACS Symposium Series American Chemical Society, Washington., 54-71.
- Gupta R K (2008):** Ginger – a wonderful spice: An overview. *Vegetos.*, 21: 1-10.
- Minghetti P, Sosaz S, Cilurzo F, Casiraghi A, Alberti E, and Tubaro A (2007):** Evaluation of the topical anti-inflammatory activity of ginger dry extracts from solutions and plasters. *Planta Medica.*, 73: 1525-1530.
- Shukla Y, and Singh M (2007):** Cancer preventive properties of ginger: A brief review. *Food and Chemical Toxicology.*, 45: 683-690.
- Balch J F, and Balch PA (1996):** Prescription for nutritional healing: A practical A-Z reference to drug-free remedies using vitamins, minerals, herbs & food supplements. *New York: Avery Publishing Group.*
- Dedov V N, Tran V H, Duke C C, Connor M, Christie M J, Mandadi S, and Roufogalis B D (2002):** Gingerols: a novel class of vanilloid receptor (VR1) agonists. *British Journal of Pharmacology.*, 137 (6): 793-798.
- Wang W H, Wang Z M (2005):** Studies of commonly used traditional medicine ginger. *Journal of China Chinese Medicine.*, (30): 1569-1573.
- Tapsell L C, Hemphill I, Cobiac L, Patch C S, Sullivan D R, Fenech M, Roodenrys S, Keogh J B, Clifton P M, Williams P G, Fazio V A, and Inge K E (2006):** Health benefits of herbs and spices: the past, the present, the future. *Medical Journal of Australia.*, 185 (4): 4-24.
- Ali B H, Blunden G, Tanira M O, and Nemmar A (2008):** Some phytochemical, pharmacological and toxicological properties of ginger (*Zingiber officinale* Roscoe): A review of recent research. *Food and Chemical Toxicology.*, 46: 409-420.
- Kaul P N, Joshi B S (2001):** Alternative medicine: Herbal drugs and their critical appraisal. *Birkhauser Verlag, Basel (Switzerland).*
- Afzal M, Al-Hadidi D, Menon M, Pesek J, and Dhama M S (2001):** Ginger: an ethnomedical, chemical and pharmacological review. *Drug Metab. Drug Interact.*, 18: 159-190.
- Davidson I W F, and Beliles R P (1991):** Consideration of the target organ toxicity of trichloroethylene in terms of metabolite toxicity and pharmacokinetics. *Drug Metab. Rev.*, 23: 493-499.
- Lash L H, Lipscomb J C, Putt D A, and Parker J C (1999):** Glutathione conjugation of trichloroethylene in human liver and kidney: kinetics and individual variation. *Drug Metab. Dispos.*, 27(3): 35 1-359.
- McAuff M A, Omoruy F O, Morrison E Y, and Asemota H N (2003):** Lipid management among coronary artery disease patients in diabetes mellitus or advanced age. *Am. J. Cardiol.*, 87: 646-664.
- Maiti R, Jana D, Das U K, and Ghosh D (2004):** Antidiabetic effect of aqueous extract of seed of *Tamarindus indica* in streptozotocin-induced diabetic rats. *J. Ethnopharmacol.*, 92: 85-91.

- Maiti R, Das U K, and Ghosh D (2005):** Attenuation of hyperglycemia and hyperlipidemia in streptozotocin induced diabetic rats by aqueous extract of seed of *Tamarindus indica*. *Biol. Pharm. Bull.*, 28: 1172–1176.
- Beppu H, Shimpo K, Chihara T, Kaneko T, Tamai I, Yamaji S, Ozaki S, Kuzuya H, and Sonoda S (2006):** Antidiabetic effects of dietary administration of *Aloe arborescens* Miller components in multiple low-dose streptozotocin-induced diabetes in mice: Investigation on hypoglycemic action and systemic absorption dynamics of aloe components. *J. Ethnopharmacol.*, 103: 468–477.
- Mascolo N, Jain R, Jain S C, and Capasso F (1989):** Ethnopharmacologic investigation of ginger (*Zingiber officinale*). *J. Ethnopharmacol.*, 27: 129–140.
- Afshari A T, Shirpoor A, Farshid A, Saadatian R, Rasmi Y, and Saboory E (2007):** The effect of ginger on diabetic nephropathy, plasma antioxidant capacity and lipid peroxidation in rats. *Food Chemistry.*, 101: 148–153.
- Dufour D R, Lott J A Nolte F S, Gretch D R, Koff R S, and Seefe L B (2000):** Diagnosis and monitoring of hepatic injury. 1. Performance characteristics of laboratory tests. *Clin. Chem.*, 46(12): 2027-2049.
- Thapliyal, R. and Maru, G. B. (2001):** Inhibition of cytochrome P450 isozymes by curcumins in vitro and in vivo. *Food Chem. Toxicol.*, 39(6): 541-547.
- Asai, A. and Miyazawa, T. (2001):** Dietary curcuminoids prevent high-fat diet-induced lipid accumulation in rat liver and epididymal adipose tissue. *J. Nutr.*, 131 (11) :2932-2935.
- Anderson R A, Broadhurst C L, Polansky M M, Schmidt W F, Khan A, and Flanagan V P (2004):** Isolation and characterization of polyphenol type-A polymers from cinnamon with insulin-like biological activity. *J Agric Food Chem.*, 52:65-70.
- El-Ghorab A H, Nauman M, Anjum F M, Hussain S, and Nadeem M (2010):** A comparative study on chemical composition and antioxidant activity of ginger (*Zingiber officinale*) and cumin (*Cuminum cyminum*). *Journal of Agricultural and Food Chemistry.*, 58: 8231–8237.
- Ramirez Bosca A, Soler A, Gutierrez M A C, Alvarez J L, and Almagro E Q (1995):** Antioxidant *Curcuma* extracts decrease the blood lipid levels of human subjects. *Age.*, 18:167-9.
- Fisher-Rasmussen W, Kjaer S K, Dahl C, and Asping U (1991):** Ginger treatment of *Hyperemesis gravidarum*. *Eur. J. Obstet. Gynecol. Rep. Biol.*, 38 (1): 19-24.
- Sharma J N, Srivastava K C, and Gan E K (1994):** Suppressive effects of eugenol and ginger oil on arthritic rats. *Pharmacol.*, 49 (5): 314-318.
- Kamtchouing P, Mbongue Fandio G Y, Dimo T, and Jatsa H B (2002):** Evaluation of androgenic activity of *Zingiber officinale* and *Pentadiplandra brazzeana* in male rats. *Asian J. Androl.*, 4 (4): 299-301.
- Murcia M A, Egea I, Romojaro F, Parras P, Jiménez A M, and Martínez-Tomé M (2004):** Antioxidant evaluation in dessert spices compared with common food additives. Influence of irradiation procedure. *Journal of Agricultural and Food Chemistry.*, 52:1872-1881.
- Mancini-Filho J, and Van-Koij A (1998):** Antioxidant activity of cinnamon (*Cinnamomum Zeylanicum*, Breyne) extracts. *Bollettino Chimico Farmaceutico.*, 137:443-447.
- Vekiari S A, Oreopoulou V, Tzia C, and Thomopoulos C D (1993):** Oregano flavonoids as lipid antioxidants. *Journal of the American Oil Chemists Society.*, 70:483-487.
- Ravindranath, V and Chandrasekhara N (1980):** Absorption and tissue distribution of curcumin in rats, *Toxicology.*, 16 : 259–265.
- Ravindranath V, and Chandrasekhara N (1981):** In vitro studies on the intestinal absorption of curcumin in rats, *Toxicology.*, 20 : 251–257.
- Ravindranath V, and Chandrasekhara N (1982):** Metabolism of curcumin-studies with [3H] curcumin, *Toxicology.*, 22: 337–344.
- Wahlstrom B O, and Blennow G (1978):** A study on the fate of curcumin in the rat, *Acta Pharmacologica et Toxicologica.*, 43: 86–92.
- Holder G M, Plummer J L, and Ryan A J (1978):** The metabolism and excretion of curcumin (1,7-bis-(4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione) in the rat, *Xenobiotica.*, 8: 761–776.
- Pan M H, Huang T M, and Lin J K, (1999):** Biotransformation of curcumin through reduction and glucuronidation in mice, *Drug Metabolism and Disposition.*, 27:486–494.
- Yemitan O K and Izeqbu M C (2006):** Protective effects of *Zingiber officinale* (*Zingiberaceae*) against carbon tetrachloride and acetaminophen-induced hepatotoxicity in rats. *Phytother. Res.*, 20(11):997-1002.
- Leung F S, A Y (1996):** in: Encyclopedia of common natural ingredients used in foods, drugs, and cosmetics, *second ed.*, Wiley, New York., 168–170.

- Toriizuka K (1998):** Basic lecture of Kampo medicine: pharmacological effect of cinnamon. *Kampo Med.*, 11: 431–436.
- Lee S H, Lee S Y, Son D J, Lee H, Yoo H S, Song S, Oh K W, Han D C, Kwon B M, and Hong J T (2005):** Inhibitory effect of 20-hydroxycinnamaldehyde on nitric oxide production through inhibition of NF-[kappa]B activation in RAW 264.7 cells, *Biochem. Pharmacol.*, 69: 791– 799.
- Singh G, Maurya S, deLampasona M P, and Catalan C A N (2007):** A comparison of chemical, antioxidant and antimicrobial studies of cinnamon leaf and bark volatile oils, oleoresins and their constituents, *Food Chem. Toxicol.*, 45: 1650–1661.
- Matan N, Rimkeeree H, Mawson A J, Chompreeda P, Haruthaithanasan V, Parker M (2006):** Antimicrobial activity of cinnamon and clove oils under modified atmosphere conditions, *Int. J. Food Microbiol.*, 107: 180–185.
- Qin B, Nagasaki M, Ren M, Bajotto G, Oshida Y and Sato Y (2003):** Cinnamon extract (traditional herb) potentiates in vivo insulin-regulated glucose utilization via enhancing insulin signaling in rats, *Diab. Res. Clin. Pract.*, 62 : 139–148.
- Schoene N W, Kelly M A, Polansky M M, and Anderson R A (2005):** Watersoluble polymeric polyphenols from cinnamon inhibit proliferation and alter cell cycle distribution patterns of hematologic tumor cell lines, *Cancer Lett.*, 230: 134–140.
- Sakr A (2007):** Ameliorative Effect of Ginger (*Zingiber officinale*) on Mancozeb Fungicide Induced Liver Injury in Albino Rats, *Australian Journal of Basic and Applied Sciences.*, 4: 650-656.
- El-Sharaky A S, Newairy A A, Kamel M A, and Eweda S M (2009):** Protective effect of ginger extract against bromobenzene-induced hepatotoxicity in male rats, *Food and Chemical Toxicology.*,47: 1584-1590.
- Abd-Allah, G A (2003):**Curcumin counteracts trichloroethylene induced liver injury in mice. *J. Egypt. Ger. Soc. Zool.*, 40 (A): 19-32.
- Moselhy S, Junbi H (2010):** Antioxidant properties of ethanolic and aqueous Cinnamon extracts against liver injury in rats.*International Journal of Advances in Pharmaceutical Sciences.*, (1): 151-155.
- Ali T A, Alireza S, Amirabbas F, Yousef R.; Ehsan S, Behrooz L, and Abdolamir A (2007):** The effect of ginger on diabetic nephropathy, plasma antioxidant capacity and lipid peroxidation in rats, *Food Chemistry.*, 101: 148–153.
- Walters M I, Gerarde H (1970):***Microchem J.*, 15:(231).
- Vassault A (1986):** *Ann.Biol.Clin.*, 44:(686).
- Reitman S, and Frankel S (1957):** A colorimetric method for the determination of serum glutamate oxaloacetic and glutamate pyruvic transaminases *Am.J.Clin. pathol.*, 28 (1): 56-63.
- Trinder, P.(1969):** Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor.*Ann Clin. Biochem.*, 6:24-27.
- Gornal A C, Bardawill C J, and David M M (1949):** Determination of serum proteins by means of the biuret method.*J.Bio.Chem.*,177:571.
- Doumas B T, Watson W A, and Bigge H G (1971):**Automated determination of serum albumin with Bromocersol Green. *Clin.Chem.Acta.*, 31: 78-96.
- Millar N (2001):** Biology statistics made simple using Excel. *School Sci. Rev.*,83(303): 23-34.

تأثير بعض النباتات العشبية على وظائف الكبد فى الفأران المعالجة بتراي كلوروايثيلين

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الكرم والقرفة والزنجبيل هي مركبات فينولية من أصل نباتي تستخدم بكثرة كتوابل للطعام . وقد وجد حديثاً أن للكرم والقرفة والزنجبيل خصائص مضادة للأكسدة . وتهدف الدراسة الحالية لفحص ما اذا كان الكرم والقرفة والزنجبيل تقى من تلف الكبد المستحدث بتراي كلورو ايثيلين , وكذلك لاطهار آلية نشاطه المحتملة .

الطرق البحثية:

تم استخدام أبعون من ذكور الجرذان البيضاء تتراوح أوزانهم بين 85 – 160 جم في الدراسة التجريبية . تم تقسيم حيوانات التجارب الى ثمانية مجموعات خمس جرذان في كل مجموعة, المجموعة الأولى (جرذان طبيعية) تناولت حمية وماء حسب الرغبة, المجموعة الثانية (تراي كلوروايثيلين) تناولت 5% من تراي كلوروايثيلين مذابة في, المجموعة الثالثة (الكرم) تناولت وجبة تحتوى على الكرم 1 جرام لكل كيلو جرام, المجموعة الرابعة (الكرم و تراي كلوروايثيلين) تناولت 5% من تراي كلوروايثيلين مذابة في مياه شرب متزامنة مع اعطاء وجبة تحتوى على الكرم 1 جرام لكل كيلو جرام, المجموعة الخامسة (القرفة) تناولت وجبة تحتوى على القرفة 1 جرام لكل كيلو جرام , المجموعة السادسة (القرفة و تراي كلوروايثيلين) تناولت 5% من تراي كلوروايثيلين مذابة في مياه شرب متزامنة مع اعطاء وجبة تحتوى على القرفة 1 جرام لكل كيلو جرام, المجموعة السابعة (الزنجبيل) تناولت وجبة تحتوى على 52جرام من الزنجبيل , المجموعة الثامنة (الزنجبيل و تراي كلوروايثيلين) تناولت 5% من تراي كلوروايثيلين مذابة في مياه شرب متزامنة مع اعطاء وجبة تحتوى على الزنجبيل 1 جرام لكل كيلو جرام.

النتائج:

أظهرت مجموعة الفئران المعاملة بتراي كلورو ايثيلين نموذج لوطأة الاكسدة فى الكبد (الاكسدة الفوقية فى الدهون)والتي تمثلت فى زيادة مستويات المصل من أنزيمات نقل الامين .وقد أكدت الزيادة فى مستويات الأنزيمات الناقلة للأمين حدوث تلف فى خلايا الكبد أثناء وطأة الاكسدة المستحثة بتراي كلوروايثيلين.ولكن الوجبة المزودة بالكرم قد قمعت معنويا الزيادة فى , الأنزيمات الناقلة للأمين, والبورتين الكلى والايبومين.والوجبة المزودة بالقرفة حسنت البروتين الكلى والايبومين . والوجبة المزودة بالزنجبيل حسنت وبشكل كبير أنزيمات نقل الأمين.

الخلاصة:

أظهرت الدراسة الحالية الى ان الاعشاب المختبرة أدت اما الى تحسن كامل أو جزئى على تأثير بتراي كلورو ايثيلين على الكبد.
وعلى سبيل المقارنة كان الكرم أكثر قوة فى التحسين الى الطبيعى , من قبيل الاعشاب المختبرة.