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ANTIOXIDANT EFFECTS OF GARLIC AND *Panax ginseng* AGAINST PCBS-INDUCED OXIDATIVE STRESS IN RATS

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ABSTRACT

The aim of the current study was to evaluate the protective role of garlic and Panax ginseng extract against polychlorinated biphenyls (PCBs)-induced oxidative stress in rats. Eighty female Sprague- Dawley rats were randomly divided into eight groups including the control group, the rat group given garlic alone (5 mg/kg b.w), the rat group administrated Panax ginseng extract alone (20 mg/kg b.w), the rat group given garlic plus *Panax ginseng* extract and the other 4 groups administrated PCBs (250 µg/kg b.w) alone or with either garlic, *Panax ginseng* extract, or with garlic and *Panax ginseng* extract. The results indicated that rats administrated PCBs caused a significant increase in the activities of ALT, AST, ALP and the levels of uric acid, creatinine, triglycerides and lipid peroxidation whereas it decreased significantly SOD and GPX. Histological examinations of the liver and kidney tissues showed severe histopathological and histochemical changes. Animals treated with garlic, *Panax ginseng*, garlic and *Panax ginseng* led to a significant elimination of the harmful effects of PCBs in all the biochemical, antioxidant parameters and the histological picture of the liver and kidney. Moreover, the treatment of garlic was found to be more effective than ginseng meanwhile, the combined treatment of garlic and *Panax ginseng* extract showed a synergistic protective effect than the individual treatment. It could be concluded that both garlic and *Panax ginseng* extract have a protective role against the PCBs-induced toxicity and these agents may be used pharmaceutically in areas with highly PCBs contamination.

Keywords: Polychlorinated biphenyls, antioxidants, garlic, ginseng

INTRODUCTION

Polychlorinated biphenyls (PCBs), members of the halogenated aromatic groups, are wide-spread, persistent environmental toxicants that have been associated, particularly during development, with behavioral deficits in both humans and animals (Seegal, *et al.*, 2002). Residues are found at different levels in the food chain and significant levels of these compounds have been found in human tissue. Being lipophilic and resistant to metabolism, PCBs accumulate and persist in adipose tissue and breast milk (Kuriyama *et al.*, 2003; Minh *et al.*, 2004). Exposure to these compounds results in various harmful effects including reproductive toxicity, immune suppression, birth defects, cancer and developmental and behavioral changes (Fadhel *et al.*, 2002).

There are 209 congeners of PCBs differing in the number and position of chlorine atoms on the two coupled biphenyl rings (Storelli *et al.*, 2003) possessing a wide spectrum of toxic effects. The number and location of chlorine atoms introduced into the biphenyl molecule determine the potency and nature of toxicity of each PCBs (Robertson and Hansen, 2001). PCBs have been widely used for various industrial applications; in plastics, electrical equipment, lubricants, hydraulic system, copying paper and adhesives, and as pesticides and fire retardants (Fadhel *et al.*, 2002). The toxicity of PCBs has been extensively investigated in recent years, but the mechanism by which the PCBs exert its effects is still unknown (Robertson and Hansen, 2001).

MacLellan *et al.*, (1994) and Gilroy *et al.*, (1998) reported that the hepatotoxicity of PCBs includes ultrastructural changes in the rough endoplasmic reticulum, smooth endoplasmic reticulum, mitochondria, plasma membrane and the cytoplasm. One mechanism by which PCBs may induce or promote hepatic tumors is by increasing hepatic oxidative stress. It has been suggested that some of the toxic manifestations of PCBs may be based, at least in part, upon an oxidative stress which results in enhanced lipid peroxidation leading to cell membrane damage, DNA damage, cellular apoptosis or necrosis, and tumorigenesis (Robertson and Hansen, 2001).

PCBs can be metabolized to catechols and hydroquinones. Further oxidation/reduction of these compounds can lead to the generation of semiquinones and quinines with the production of reactive oxygen species and hence resulting in cellular damage (Oakley *et al.* 1996; McLean *et al.* 2000; Srinivasan *et al.* 2001). Moreover, exposure to Aroclor 1254, a PCB member, may generate reactive oxygen species (ROS) and decrease the activity of epididymal sperm antioxidant systems in adult rats (Dhanammal, 2002).

There is growing evidence in the literature that some plant extracts possess an array of interesting pharmacological effects. Ginseng is a traditional herbal remedy used in Chinese medicine for thousands years (Loo *et al.*, 2004). One of the most commonly used and researched of ginsengs is *Panax ginseng* (Kakizoe, 2000). The main active components of *Panax ginseng* are ginsenosides, which have been shown to have a variety of beneficial effects, including anti-inflammatory, antioxidant, and anticancer effects (Kakizoe, 2000; Kampen *et al.*, 2003; Abdel-Wahhab and Ahmed, 2004; Loo *et al.*, 2004).

Garlic (*Allium sativum*) is a common spicy flavouring agent used since ancient times. It has been shown to posses many medicinal properties including bactericidal, hypolipidemic, hypocholestrolemic, antineoplastic, anticancer effects (Jonkers *et al.*, 1999; Duncan, 1999; Singh and Shukla, 1998; Siegers *et al.*, 1999) and antioxidant properties against different toxic agents e. g., aflatoxin (Abdel-Wahhab and Aly, 2003). Dwivedi *et al.* (1998) reported that oil soluble organosulfur compounds present in garlic induced an antiperoxidant effect. Moreover, Sumioka *et al.* (1998) stated that S-allylmercaptocysteine (SAMC), one of the water-soluble organosulfur compounds in ethanol extracts of *Allium sativum* protects mice against acetaminophen that induce liver injury. Furthermore, the protective role of

garlic extracts against the toxic effect of aflatoxin was studied by Abdel-Wahhab and Aly (2003). They reported that treatment with garlic extract plus aflatoxin resulted in a significant improvement in the different biochemical and antioxidant parameters affected by aflatoxin treatment. Garlic extract was also found to prevent the maternal and developmental toxicity and lipid peroxidation in pregnant rats treated with fumonisin mycotoxin (Abdel-Wahhab *et al.*, 2004).

The objectives of the current study were to evaluate the protective role of garlic and *Panax ginseng* extract alone and together against the toxicological effects of PCBs.

MATERIALS AND METHODS

Chemicals:

DCMA Polychlorinated biphenyls (PCBs) mixtures were purchased from Supelco Company (Supelco Park, Bellefonte, PA. U.S.A). The chemical composition of the PCBs mixture is depicted in Table (1). Other chemicals were of the highest purity commercially available.

Kits:

Kits for determination of alanine aminotransferase (ALT) and aspartate aminotransferase (AST), alkaline phosphatase (ALP), triglycerides, uric acid and creatinine kits were obtained from Biomerieux, Laboratory of Reagents and Products (Marcy 1 'Etoile, France). Kits for determination of glutathione peroxidase (GPX) and superoxide dismutase (SOD) were obtained from Randox Laboratories Co. Ardmore, Diamond Road, Crumlin,Co. Antrim, BT294 QY (UK).

Panax ginseng

The standarized *Panax ginseng* extract EFLA400 (*Phoenix ginseng*) (Batch No. 303298) of *Panax ginseng* C. A. Mayer was supplied from Lotte Group R&D Center (Seoul, Korea). The content of ginsenoside Rg3, a pharmacologically active ingredient of *Phoenix ginseng* was 3.6 % (w/w) as determined by HPLC (Panwar *et al.*, 2005).

Preparation of garlic powder

Fresh garlic bulbs were purchased from the local market at Cairo, homogenized and freeze dried using a freeze dryer system (Dura-Dry Freeze Drye, Model PAC-TC-V4; FTS system, Inc. Stone Ridge, NY. USA). The dried garlic powder was kept at - 20 °C until used.

Experimental animals

Three months old, sexually mature female Sprague- Dawley rats (130-150 g) were purchased from the Animal House Colony, Giza, Egypt. The animals were maintained on a standard diet (protein: 16.04%; fat: 3.63%; fiber: 4.1%, and metabolic energy: 2887 Kcal, Kg) and water was available *ad libitum* at the Animal House Lab., National Research Centre, Cairo, Egypt. After an acclimatization period of 1 week, animals were divided into eight groups (10 rats/ group) and housed in filter-top polycarbonate cages housed in a temperature controlled and artificially illuminated room free from any source of chemical contamination.

Experimental design:

Animals within treatment groups were intragastically treated daily for two weeks as follows: group 1, untreated control; group 2, treated with garlic powder (5 mg/kg b.w) suspended in corn oil; group 3, treated with *Panax ginseng* extract (20 mg/kg b.w) suspended in corn oil; group 4, treated orally with garlic powder and *Panax ginseng* extract suspended in corn oil; group 5, treated with PCBs (250 µg/kg b.w) dissolved in corn oil.; group 6, treated with garlic and PCBs in corn oil; group 7, treated with *Phoenix ginseng* and PCBs in corn oil; and group 8, treated with garlic and *Panax ginseng* extract and PCBs in corn oil.

At the end of the experimental period, blood samples were collected from all animals from the retro-orbital venous plexus after being fasting for 12 h for different biochemical analysis. After the collection of blood samples, all animals were sacrificed and dissected. The activities of ALT and AST were determined according to the method recommended by The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology (1974). Triglycerides were determined in serum according to Wahlefeld (1974). Alkaline phosphatase activity (ALP) was determined by kinetic spectroscopy according to the method of Roy (1970). Creatinine and Uric acid were determined in serum according to the methods of Bartles *et al.* (1972) and Haisman and Muller (1977) respectively. Samples of the livers were homogenized in phosphate buffer (pH 7.4) to give 20 % w/v homogenate (Lin *et al.*, 1998). The homogenate was centrifuged at 1700 rpm at 4 °C for 10 min and the supernatant was stored at –20 °C until analysis at the next day. The supernatant (20%) was used for the determination of lipid peroxidation in the liver tissue and it was further diluted with phosphate buffer solution to give 2 % and 0.5 % dilutions for the determination of glutathione peroxidase (2 %) and superoxide dismutase (0.5 %) activities in the liver tissues.

Hepatic lipid peroxidation was estimated by the measurement of malondialdehyde (MDA) by spectrophotometeric method (Esterbauer *et al.*, 1991) using Oxos Research™ Co. kit (USA). Hepatic glutathione peroxidase activity was determined by spectrophotemetric method using reduced glutathione and cumen hydroperoxide as a substrate and 20 µl diluted liver homogenate according to the modified method of Paglia and Valentine (1967) using a Ransel kit obtained from Randox Laboratories Co. Ardmore, Diamond Road, Crumlin,Co. Antrim, BT294 QY (UK). Hepatic superoxide dismutase activity was assayed spectrophometerically by a red formazan dye reduction procedure (Suttle, 1986) using 50 µl diluted liver homogenate and the Ransel kit from Randox Laboratories Co. (UK). The activity of hepatic glutathione peroxidase, and superoxide dismutase was expressed as unit/g liver.

Other samples from the livers and kidneys were excised and fixed in 10% neutral formalin for histopathological studies. The tissue samples were dehydrated in ascending grades of ethanol, clean in xylene and embeded in paraffin. Sections (8um) from all organs were cut and stained with hematoxylin and eosin (H&E) (Drury and Wallington, 1980). For the

histochemical investigations Crossman's stain was carried out for demonstration of connective tissue in liver (Culling, 1963).

Statistical analyses:

All data were subjected to statistical analyses using the General Linear Models (GLM) Procedure of the Statistical Analysis System (SAS, 1982). The significance of the differences among treatment rat groups with variable means was determined by Waller-Duncan k-ratio T test (Waller and Duncan, 1969). All statements of significance were based on a probability level of $P \leq 0.05$.

Table (1): Composition and concentrations of PCBs standard mixture

Individual PCB	Concentration µg/ml
2 chlorobiphenyls	100
3,3'- Dichlorobiphenyls	100
2,4,5- Trichlorobiphenyls	10
2,2',4,4'-Tetrachlorobiphenyls	10
2,3,4,5,6- Pentachlorobiphenyls	10
2,2'3,3',6,6'- Hexachlorobiphenyls	10
2,2', 3,4,5,5',6- Heptachlorobiphenyls	5
2,2', 3,3',4,4',5,5',- Octachlorobiphenyls	5
2,2', 3,3',4,4',5,5',6- Nonachlorobiphenyls	5
2,2', 3,3',4,4',5,5',6,6'- Decachlorobiphenyls	5

RESULTS

The results of the present study revealed that PCBs induced severe toxicological effects on serum biochemical parameters tested. Animals feeding on a control diet and orally given PCBs showed a significant increase in ALT, AST, ALP, uric acid, creatinine and triglycerides. The biochemical parameters in animals feeding on a control diet and orally given *Panax ginseng* extract and/or garlic for 2 weeks were comparable to the controls except uric acid level which was significantly lowered from 4.44 mg/dl for the control group to 3.00 mg/dl in the group treated with *Panax ginseng* extract plus garlic. Animals feeding on a control diet and orally given *Panax ginseng* extract and/or garlic for 2 weeks in the presence of PCBs significantly eliminated the deleterious effect of PCBs on ALT, AST, uric acid, creatinine, triglycerides although feeding rats on a control diet and orally given *Panax ginseng* in the presence of PCBs did not alleviate the harmful effect on alkaline phosphatase (ALP). However, feeding rats on a control diet and orally administrated garlic in the presence of PCBs for 2 weeks significantly eliminate the harmful effect of PCBs on ALP (Table 2).

The effects of different treatment on the activity of glutathione peroxidase (GPX), superoxide dismutase (SOD) and lipid peroxidation (LPO) in liver of rats are depicted in table (3). It is clearly indicated that PCBs induced a significant decrease in both GPX and SOD activity whereas, it induced a significant increase in LPO. Animals feeding on a control diet and

Table (2): Effect of garlic and *Panax ginseng* extract on serum biochemical parameters in rats orally administrated PCBs for two weeks.

orally given garlic alone, *Panax ginseng* extract alone or in combination increased the antioxidant capacity of the liver since these treatments succeeded to increase both SOD and GPX and decrease LPO. The

Treatment	ALT (U/ml)	AST (U/ml)	ALP (IU/l)	Uric Acid (mg/dl)	Creatinine (mg/dl)	Triglycerides (mg/dl)
Control	26.25^{b} ± 4.03	28.67^{b} ± 3.48	124.46^{b} ± 13.78	4.44^{bc} ± 0.18	0.42^{b} ± 0.01	127.16^{b} ± 13.01
PCBs	87.75^{a} ± 10.16	79.00^{a} ± 12.47	190.20^{a} ± 5.85	6.47^{a} ± 0.39	0.65^{a} ± 0.04	166.80^{a} ± 7.86
Ginseng	26.33^{b} ± 3.76	28.00^{b} ± 3.81	124.34^{b} ±11.37	3.72^{cd} ± 0.28	0.44^{b} ± 0.05	125.67^{b} ± 12.95
Garlic	27.33^{b} ± 2.67	29.00^{b} ± 1.35	123.55^{b} ±12.93	4.29^{bc} ± 0.28	0.45^{b} ± 0.06	124.66^{b} ± 1.61
Ginseng + Garlic	26.75^{b} ± 5.27	29.25^{b} ± 4.07	126.09^{b} ± 9.91	3.00^{d} ± 0.07	0.44^{b} ± 0.05	100.50^{b} ± 12.13
PCBs + Ginseng	36.75^{b} ± 3.59	31.25^{b} ± 4.07	156.76^{a} ±14.44	4.24^{bc} ± 0.19	0.46^{b} ± 0.06	112.34^{b} ± 11.55
PCBs + Garlic	32.00^{b} ± 4.42	36.33^{b} ± 2.96	111.90^{b} ± 7.48	4.86^{b} ± 0.37	0.40^{b} ± 0.04	123.34^{b} ± 10.48
PCBs + Ginseng + Garlic	40.50^{b} ± 3.30	40.25^{b} ± 3.73	125.45^{b} ± 5.54	4.79^{b} ± 0.22	0.43^{b} ± 0.02	115.73^{b} ± 11.94

treatment with PCBs plus *Panax ginseng* extract, garlic or *Panax ginseng* extract plus garlic resulted in a significant improvement of the antioxidant parameters towards the normal level of the control.

Within each column, means superscripted with different letters are significantly different (P<0.05)

Table (3): Effect of garlic and *Panax ginseng* on lipid peroxidation, in rats orally given PCBs for two weeks.

Treatment	Lipid peroxidation (nmol/mg liver)	Glutathione peroxidase (u/g liver)	Superoxide dismutase (u/g liver)
Control	175.99^{bc} ± 24.03	56.75^{c} ± 2.36	1130.0^{b} ± 65.74
PCBs	312.82^{a} ± 52.22	44.34^{d} ± 1.26	627.08^{a} ± 98.97
Ginseng	103.09^{d} ± 17.09	64.52^{a} ± 1.01	1134.38^{b} ± 24.67
Garlic	169.02^{cd} ± 14.10	64.84^{a} ± 1.92	1175.0^{b} ± 13.11
Ginseng + Garlic	166.32^{bc} ± 15.81	65.69^{a} ± 1.19	1200.0^{b} ± 13.69
PCBs + Ginseng	241.41^{b} ± 13.93	58.55^{bc} ± 1.54	1107.5^{b} ± 28.67
PCBs + Garlic	146.15^{cd} ± 17.05	62.04^{ab} ± 2.18	1121.88^{b} ± 83.91
PCBs + Ginseng + Garlic	171.02^{cd} ± 12.43	66.03^{a} ± 1.07	1057.5^{b} ± 79.21

Within each column, means superscripted with different letters are significantly different (P<0.05)

The histopathological results showed normal structure of the liver in the control group or those treated with *Panax ginseng* extract, garlic and *Panax ginseng* extract plus garlic (Fig.1a). Livers of rats administrated PCBs

showed thickening of the interlobular connective tissue and massive inflammatory infiltration, congested and dilated veins in the portal areas. The hepatocytes that surrounded the portal areas showed necrosis associated with inflammatory infiltration. Focal necrosis of the hepatocytes and increasing of the binucleated cells was also seen (Fig 1b,c,d). On the other hand, the liver of rats fed on control diet and orally given PCBs and *Panax ginseng* extract, PCBs and garlic, PCBs plus garlic and *Panax ginseng* appeared more or less as control rats (Fig.1e). The histochemical examinations of the liver sections of the rat control group revealed normal distribution of Crossman's stain in the portal areas and the septum between the hepatic lobules (Fig.2a). Rats administrated PCBs alone showed an increase of the connective tissue in the portal areas with increased thickness of the interlobular connective tissue (Fig. 2b,c). Whereas, animals given PCBs and *Panax ginseng* extract, PCBs and garlic and PCBs plus garlic and *Panax ginseng* extract showed a decrease in the connective tissue and appeared more or less as the control (Fig. 2d).

Microscopic examinations of kidney sections of control rats and the groups given *Panax ginseng* extract, garlic or *Panax ginseng* extract plus garlic showed normal structure of the renal tissue (Fig. 3a). On the other hand, kidney of the animals administrated PCBs showed desquamation of the tubular epithelial cells. The glomeruli showed hypercellularity associated with dilatation of the urinary space and some glomeruli showed complete degeneration (Fig.3b). Kidney tissues of rats given PCBs and *Panax ginseng* extract showed haemorrhagic areas in the interstitium and inflammatory infiltration with cloudy swelling in the tubules (Fig.3c). Whereas, kidney of rats fed on a control diet and orally administrated PCBs and garlic exhibited normal structure of both the renal corpuscles and tubules (Fig.3d) although a cloudy swelling in the renal tubules was found in some cases and the glomeruli exhibited segmented form and wide urinary space (Fig 3e). Kidneys of rats given a control diet and orally administrated PCBs plus garlic and *Panax ginseng* extract exhibited fibrous tissue in the interstitium that was associated with inflammatory infiltration (Fig. 3f).

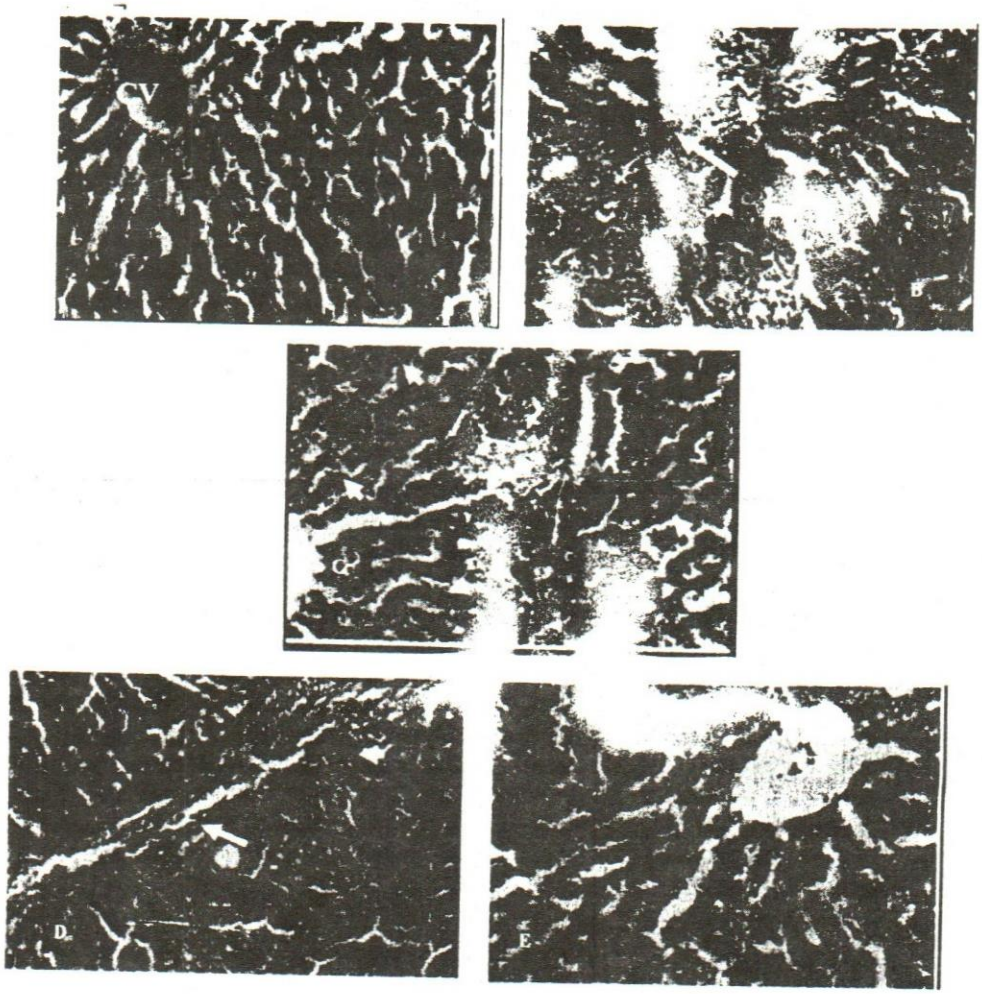

Fig. (1): Liver of rat (A) control showing the normal structure, (B) rats given orally PCBs showing thickening of the interlobular connective tissue (arrow), the portal area with dilated and congested vein (arrowhead), the hepatocytes that surround the portal area exhibit necrosis associated with inflammatory infiltration, (C) rats administrated PCBs showing focal necrosis (*) of the hepatocytes and little inflammatory infiltration. Notice the increase of binucleated cells, (arrowhead), (D) administrated PCBs showing the thickening of the interlobular connective tissue (arrow) and massive inflammatory infiltration (arrowhead)., (E) administrated PCBs plus garlic, *ginseng* or garlic plus *ginseng* showing the central vein and hepatocytes similar to that of control (H& E X 300).

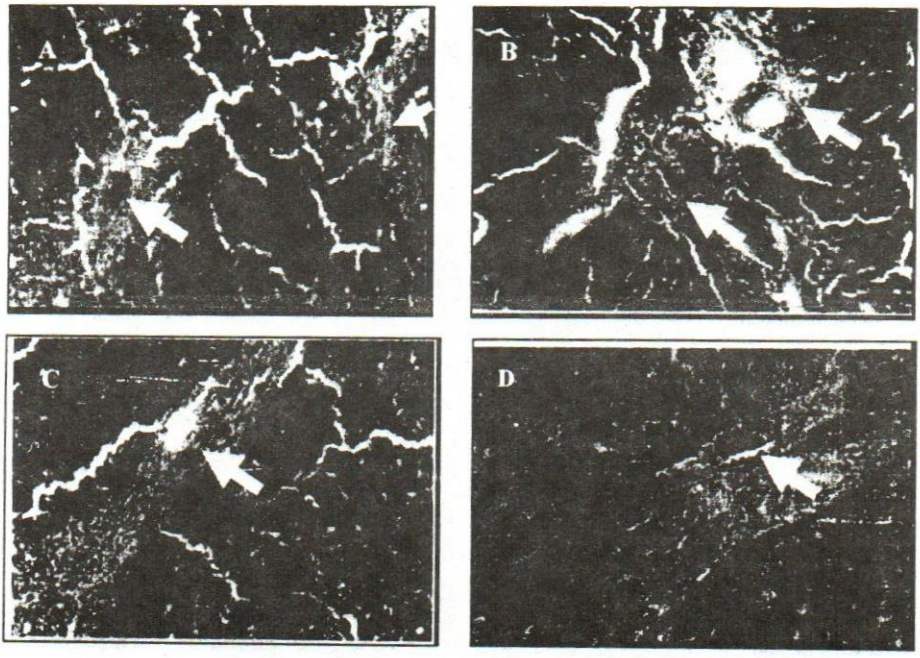

Fig. (2): Photographs of liver sections (A) control rats showing the connective tissue of the portal area and the septum between the hepatic lobules (arrow), (B) rat administrated PCBs showing connective tissue growing in the portal area (arrows), (C) Rats given PCBs showing the thickening of the interlobular connective tissue (arrow), and (D) rats given PCBs plus garlic, *Panax ginseng* or garlic plus *Panax ginseng* showing the connective tissues that exhibited normal distribution (Crossman's stain X 150).

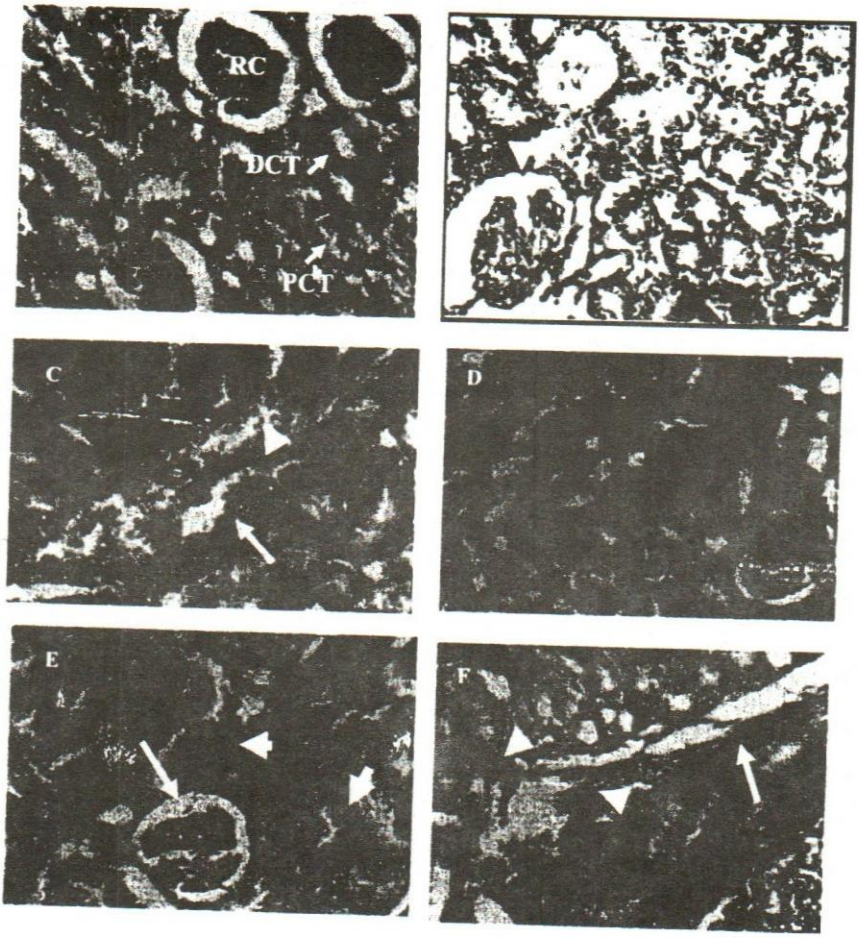

Fig. (3): (A) Photographs of kidney sections (A) control rats showing a renal corpuscle (RC), proximal convoluted tubules (PCT) and distal convoluted tubules (DCT), (B) Rats administrated PCBs showing dosquamation of the tubular epithelial cells (arrow). The glomerulus shows hepercellularity associated with dilatation of the nary space (arrowhead). Another glomerulus shows complete degeneration (*), (C) rats given PCBs plus *Panax ginseng* showing haemorrhagic areas (arrow) in the interstitium and inflammatory infiltration (arrowhead). The tubules show cloudy swelling, (D) rats administrated PCBs plus garlic showing the normal structure of both the renal corpuscles and tubules, (E) Rats given PCBs plus garlic and *Panax ginseng* showing cloudy swelling of the renal tubules (arrowhead). The glomerulus exhibits a segmented form and wide urinary space (arrow) and (F) treated with PCBs plus garlic and *Panax ginseng* showing fibrous tissue in the interstitium (arrow) which is associated with inflammatory infiltration (arrowhead) (H& E X 300).

DISCUSSION

Previous studies indicated that each PCB congener has specific toxic criteria, for example, PCB (3) is a protype of lower halogenated biphenyl and is rapidly metabolized (MacLellan *et al.*, 1994) whereas, PCB (38) metabolite is a potent inducer of oxidative DNA damage (Oakley *et al.*, 1996), PCB (77) is an aryl hydrocarbon receptor (AhR) agonist (Parkinson *et al.*, 1983), and PCB (153) is a higher chlorinated biphenyl that induces cytochrome P450 (Twaroski *et al.*, 2001).

In the current study, mixture of PCBs was used to induce toxicity in rats. Our data revealed that PCBs induce a significant increase in ALT, AST and ALP activities, uric acid, creatinine and triglyceride levels. The activity of ALT and AST are sensitive indicators of acute hepatic necrosis (Kaplan, 1987; Abdel-Wahhab, *et al.*, 2002; Abdel-Wahhab and Aly, 2005). Consequently, these results may indicate degenerative changes of liver and kidneys (Abdel-Wahhab and Aly 2003; Farombi *et al.*, 2005). The increased levels of uric acid reported herein may indicate protein catabolism and/or kidney dysfunction (Abdel-Wahhab *et al.*, 1998, 1999, 2002). These results clearly showed that PCBs possessed harmful and stressful influence on the hepatic and renal tissue and consistent with those reported by Robertson and Hansen (2001). Similar to our observations, Twaroski *et al.* (2001) stated that PCBs mixture can diminish liver activity in rats and supported the hypothesis that rodent liver is a target of PCBs toxicity.

Another mechanism by which PCBs may induce or promote hepatic toxicity is through the increase of hepatic oxidative stress (Ramadass *et al.*, 2003). This oxidative stress results in enhanced lipid peroxidation (LPO) leading to cell membrane damage, DNA damage, cellular apoptosis or necrosis and tumorigenesis (Robertson and Hansen, 2001; Abdel-Wahhab *et al.*, 2005; Ahmed *et al.*, 2005). LPO is one of the main manifestations of oxidative damage and has been found to play an important role in the toxicity and carcinogenesis of many carcinogens (Rastogi *et al.*, 2001; Abdel-Wahhab, *et al.*, 2006). The hepatic antioxidants represent the major defense against toxic liver injury, and they act as anti-apoptosis. In the current study, the levels of oxidative stress indice, LPO was significantly increased while the levels of antioxidants GPx and SOD in PCBs-treated group were significantly decreased compared to their levels in the controls. The increased level of LPO with the decreased level of GPx and SOD indicated that LPO is one of the most pronounced factors in PCBs-toxicity and carcinogenicity (Hassan *et al.*, 2005). Moreover, Abdel-Wahhab and Aly (2005) stated that both GPx and SOD are considered to be enzymatic free-radical scavengers in cells. Thus, the decrease of both enzymes leads to an indirect increase in oxidative DNA damage and suggesting that SOD plays a role in the suppression of oxygen free-radical formation and the decrease of NO generation. In this regards, Cheung *et al.*, (2002) proposed that chlorinated hydrocarbons, which caused oxidative stress, could inhibit the activities of protective enzymatic antioxidants. Casillas *et al.* (1996) also reported a decrease in the activities of antioxidant enzymes in mussels

exposed to PCB contaminated waters. These results were in agreement with those reported by Glauert *et al.* (2001); Abdel-Wahhab and Aly (2005). Moreover, Azevedo-Martins *et al.* (2003) stated that SOD play a role in the suppression of oxygen free radical formation and decrease NO generation.

It is well known that the antioxidant enzymes play a substantial role in protecting organisms against oxidative stress. In the absence of these antioxidant enzymes, hydroxyl radicals, the causative agent of LPO, attack polyunsaturated fatty acids to produce lipid peroxides in the presence of transition metals such as iron which coupled with a redox system and oxygen under appropriate conditions. The current results coincided with those of Fadhel *et al.*, (2002) who stated that PCBs may increase the hepatic LPO by lowering cellular antioxidant defenses. In addition, Goeptar *et al.*, (1995) reported that PCBs decrease hepatic glutathione peroxidase activity and vitamine E concentrations and stated that active oxygen in the form of superoxide or hydrogen peroxide can be released as a by-product from cytochrome P450 and could contribute to LPO. Other sources of oxidative stress include many enzymes induce, the suppression of antioxidants or antioxidant enzymes (Pelissier *et al.*, 1992), or different metabolic events (Perg *et al.*, 2001). Moreover, the induction of cytochrome P450 by PCBs increase free radical formation. Reaction of free radicals with cellular molecules can lead to the initiation of LPO (Fadhel *et al.*, 2002).

The changes in the serum biochemical parameters reported in the current study were confirmed by the histological and histochemical examination of liver and the histological examination of the kidney. Treatment with PCBs alone resulted in severe pathological changes in liver and kidney tissues and indicated that PCBs mixture is a potent hepatonephrotoxicant. In this regard, Fadhel *et al.* (2002) reported that PCBs induced ultrastructural changes in the rough and smooth endoplasmic reticulum, mitochondria, plasma membrane and the cytoplasm. Moreover, Glauert *et al.*, (2005) and McGavin *et al.*, (2001) reported that PCBs induced necrosis and the presence of hyaline droplets in the renal tubules. It was previously reported that the kidney in rats is a target for PCBs (McGavin *et al.*, 2001). The histopathological changes found in the present study provide additional support for conclusion that the kidney in rats is a primary target for PCBs.

In the present study a protective effect of garlic and *Panax ginseng* extract against the toxic hazards of PCBs was observed. The treatment with garlic or *Panax ginseng* extract in the presence of PCBs led to the lowering of the elevated levels of the biochemical parameters (ALT, AST, ALP, creatinine, uric acid, triglycerides and LPO) and normalized the histological picture of the investigated organs. Recently, organosulfur compounds of garlic have been shown to scavenger the oxygen free radicals and possessed antioxidant properties (Imai *et al.*, 1994; Ide *et al.*, 1996; Abdel-Wahhab and Aly, 2003; Abdel-Wahhab *et al.*, 2004). Ide *et al.*, (1996) stated that garlic had the highest antioxidant activity against peroxyl radical and suggested that garlic may be an effective antioxidant in preventing or treating disorders related to endothelial cell injury associated with free radicals.

Another mechanism which may be involved in the protection of garlic against PCBs-induced liver and kidney injury is the P450 enzymes. Several

P450 enzymes have been reported to play important roles in the bioactivation of PCBs to reactive metabolites (Ramadass, *et al.*, 2003) and P450 inhibitors, such as diallyl sulfide present in garlic have been shown to protect the liver against PCBs-induced liver injury. Furthermore, the increased level of antioxidant enzymes (GPX and SOD) and the decreased level of LPO in rats fed on a diet comprised garlic may be another pathway in the protection against PCBs toxicity (Ramadass *et al.*, 2003; Glauert *et al.*, 2005; Abdel-Wahhab and Aly, 2005).

In a previous work, Panwar *et al.* (2005) reported that *Phoenix ginseng* is a 3 % ginsenoside Rg3 standardized *Panax ginseng* extract. Three percent means that more than 95% of protoPanaxadiol ginsenosides in natural *Panax ginseng* are converted into ginsenoside Rg3 and Rg5. Other protopanaxatriol ginsenosides are also converted into their congeners. This conversion makes *Phoenix ginseng* has 8 times more potent antioxidant action. Moreover, Kim *et al.* (1997) reported that ginseng has a potent protective action against CCL_4-induced toxicity and it showed inhibitory effect on cytochrome P450- associated monoxygenase activities. Therefore, it is suggested that the protective effect of *Phoenix ginseng* is attributable to its free radical scavenging activity (Abdel-Wahhab and Ahmed, 2004; Mannaa *et al.*, 2006).

In the present study, the histological examination of liver and kidney of rats fed on a diet containing PCBs combined with garlic alone, *Panax ginseng* alone or with *Panax ginseng* plus garlic revealed marked elimination of the adverse effects of PCBs in the histopathological and histochemical picture. *Ginseng* extracts increase the biosynthesis of proteins and nucleic acids, metabolize the carbohydrates and lipids, enhance the reduction and elimination of the toxic effects as well as stimulate regeneration of cells and improve inflammation (Kim *et al.*, 1997; Abdel-Wahhab and Ahmed, 2004). It was found that, the non-saponin components of Korean red *ginseng* suppressed the harmful effects of free oxygen radicals (O_2, H_2O_2, OH_2), which exercise an important role in tissue degeneration (Kim *et al.*, 1997). Moreover, Zhang *et al.* (1996) showed that hydroxyl radical formed by the Fenton reaction were completely inhibited by *ginseng* extract. This antioxidant effect of *ginseng* may be responsible for its wide pharmacological actions in clinical practice by a free radical reaction-inhibition mechanism. Therefore, the protective effects of *Panax ginseng* or garlic may be related to their antioxidant properties. The earlier studies demonstrated that garlic protects against the genotoxic effects of carcinogens by modulating LPO and enhancing GSH-dependent antioxidants (Arivazhagan *et al.*, 2000, 2001; Chandra Mohan *et al.*, 2003; Abdel-Wahhab and Aly, 2003; Abdel-Wahhab *et al.*, 2002, 2004). Furthermore, the possible reason for the apparent synergistic effects of *Panax ginseng* and garlic combination may be due to the presence of several phytochemicals which are reported to display both complementary and overlapping mechanism of actions, including induction of detoxification enzymes and antioxidants (Kik *et al.*, 2001; Weisburger, 2002).

In conclusion, the current study revealed that PCBs induced severe toxic effects on liver and kidney as indicated by the elevation of serum biochemical parameters and LPO in liver accompanied with the decrease in antioxidant enzyme activities. Both garlic and *Panax ginseng* extract exhibit potential protective effects against PCBs-induced stress. Moreover the combined treatment with PCBs plus garlic and *Panax ginseng* was more effective than the individual treatment. The protective effects of both garlic and *Panax ginseng* extract may be due to their ability to scavenge free radicals, decrease LPO and lead to higher antioxidant enzyme activities that reflect high potency as antioxidant agent.

REFERENCES

Abdel-Wahhab, M. A., and Ahmed, H. H. (2004). Protective effects of Korean *panax ginseng* against chromium VI toxicity and free radical generation in rats. J. Ginseng Res. 28 (1), 11-17.

Abdel-Wahhab, M. A. and Aly, S. E. (2003). Antioxidants and radical scavenging properties of vegetable extracts in rats fed aflatoxin-contaminated diet. J. Agric. Food. Chem.*51*, 2409-2414.

Abdel-Wahhab, M. A., and Aly, S. E. (2005). Antioxidant property of *Nagilia Sativa* (Black cumin) and *Syzygium Aromaticum* (Clove) in rats during Aflatoxicosis. J. Appl. Toxicol. *25*, 218-223

Abdel-Wahhab, M. A., Abdel-Galil, M. M., and Ellithey, M. M. (2005). Melatonin counteracts oxidative stress in rats fed Ochratoxin A-contaminated diet. J. Pineal Res. *38*, 130-135.

Abdel-Wahhab, M. A, Ahmed, H. H., and Hagazi, M. M. (2006). Prevention of aflatoxin B_1-initiated hepatotoxicity in rat by marine algae extracts. J Appl. Toxicol. *26*, 229-238.

Abdel-Wahhab, M. A., Hassan, A. M., Amer, H. A., and Naguib, Kh. M. (2004). Prevention of fumonisin-induced maternal and developmental toxicity in rats by certain plant extracts. J. Appl. Toxicol. *24*, 469 -474.

Abdel-Wahhab, M. A., Nada, S. A., and Arbid, M. S. (1999). Ochratoxicosis: Prevention of Developmental Toxicity by L-methionine in the rats. Ibid. *19*, 7-12.

Abdel-Wahhab, M. A., Nada, S. A.; and Khalil, F. A. (2002). Physiological and toxicological responses in rats fed aflatoxin-contaminated diet with or without sorbent materials. Animal Feed Sci. and Technol.,10740,1-11.

Abdel-Wahhab, M. A., Nada, S. A., Farag, I. M., Abbas, N. F., and Amra, H. A. (1998). Potential Protective effect of HSCAS and Bentonite against dietary aflatoxicosis in rat: with special reference to chromosomal aberrations. Nat. Toxins 6: 211-218.

Ahmed H. H., Mannaa, F. A., Fyiad, A. A., and Abdel-Wahhab, M. A. (2005). Dimethyl diphenyl bicarboxylate (DDB) reduces the burden of alcohol-induced liver toxicity. J. Egypt. Soc. Toxicol. *33*, 9-17.

Arivazhagan, S.,Balasenthil, S.and Nagini, S.(2000).Modulatory effects of garlic and neem leaf extract on N-methyl-NV-nitro-N-nitrosoguanidine induced oxidative stress in Wistar rats.Cell Biochem. and Fun.*18*, 17–21.

Azeredo-Martins, A. K, lortz, S., Lenzen, S., Curi, R., Eizirik, D. L, and Tiedge, M. (2003). Improvement of the mitochondrial antioxidant defense status prevents cytokine-induced nuclear factor-kappa B activation in insulin-producing cells. Diabetes *52*, 93-101.

Bartles, H.; Bohmer, M.; Heirli, C. (1972). Serum creatinine determination without protein precipitation. Clin. Chem. Acta. *37*,193- 197.

Casillas, E.; Krishnakumar, P. K., Snider, R. G., and Varanasi, U.(1996): Effects of chemical contaminants on subcellular changes in digestive cells of the marine bivalve, from Puget Sound, Washington, Marine Environ. Res. *42*, 107-111.

Chandra Mohan, K. V. P., Bhuvaneswari, V., Abraham, S. K., and Nagini, S. (2003). Dose-dependent protection by tomato against 7, 12-dimethylbenz[a]anthracene-induced genotoxicity and oxidative stress in mice. J Med Food. *6*, 169–173.

Cheung, C. C. C.; Zheng, G. J.; Lam, P. K. S.; and Richardson, B. J. (2002). Relationships between tissue concentrations of chlorinated hydrocarbons (polychlorinated bipenyls and chlorinated pesticides) and antioxidative responses of marine mussels, *Perna viridis*. Marine Pollution. *45*, 181-191.

Culling, C. F.A (1963). Handbook of histopathological technique, 2nd Ed. Butterworths, London.

Dhanammal, S. (2002). Effects of vitamin C, vitamin E and quercetin on Aroclor 1254 induced oxidative stress in epididymis of adult albino rats. M. Phil. Dissertaion, University of Madras, Chennai, India.

Drury, R. A. B. and Wallington, E. A. (1980). Carleton's histological technique 4th Ed. Oxford, New York, Toronto, Oxford University Press.

Duncan, M. G. (1999). The effects of nutritional supplements on treatment of depression diabetes and hypercholesterolemia in the renal patient. J. Ren. Nutr. *9*:58-62.

Dwivedi, C., John, L. M., Schmidt, D. S., and Engineer, F. N. (1998). Effects of oil-soluble organosulfur compounds from garlic on doxorubicin-induced lipid peroxidation. Anti Cancer Drugs. 9 (3), 291- 294.

Esterbauer, H., Schaure, R. J., and Zollner, H. (1991). Chemistry and biochemistry of 40 hydroxy-nonenal malonaldehyde and related aldehydes. Free Rad. Biol. Med. *11*, 81- 128.

Fadhel, Z.; Robertson, L.W., and Glauert, H.P.(2002). Effect of 3,3',4,4'-tetrachlorobiphenyl on the induction of hepatic lipid peroxidation and cytochrome P-450 associated enzyme activities in rats, Toxicol. *175*: 15-25.

Farombi, E. O., Adepoju, B. F., Ola-Davies, O. E., and Emerole, G. O. (2005). Chemoprevention of aflatoxin B1-induced genotoxicity and hepatic oxidative damage in rats by kolaviron, a natural bioflavonoid of Garcinia kola seeds. Eur J Cancer Prev. 14 (3), 207-214.

Gilroy, C., Connell, B. J., Singh, A., Suidgeest, P., and Chu, I. (1998). PCB congener 77- induced ultrastructural alterations in the rat liver: a quantitative study. Toxicol. *127*, 179-185.

[redacted], H.P., Robertson, L.W., and Silberhorn, E.M. (2001). PCBs and tumor promotion. In: Robertson, L.W., Hansen, L.G. (Eds.), PCBs: recent advances in environmental toxicology & health effects. University press of Kentucky, lexinglon,ky Ky, pp.355-371.

Goeptar, A. R., Scheerens, H., and Vermeulen, N. P. E. (1995). Oxygen and xenobiotic reductase activities of cytochrome P450. Crit. Rev. Toxicol. 25: 25-65.

Haisman, P., and Muller, B. R. (1977). Glossary of clinical chemistry terms. Butterworth, London,p.p.126.

Hassan, A. M., Nada, S. A., Hassan, N. S., El- Kady, A. A., and Abdel–Wahhab, M. A. (2005). Protective role of fennel oil against polychlorinated biphenyls (PCBs) induced oxidative stress in rats. Zagazig J. of Forn. Med. & Toxicol.*7*, 95-111.

Ide, N., Matsuura, H., and Itakura, Y. (1996). Scavenging effect of aged garlic extract and its constituents on active oxygen species. Phytother Res. *10*, 340-341.

Imai, J., Ide, N., Nagae, S., Moriguchi, T., Matsuura, H., and Itakura, Y. (1994). Antioxidant and radicai scavening effects of aged garlic extract and its constituents. Planta Med. *60*, 417-420.

Jonkers, D., Van-den-broek, E., Van-dooren, I., Thijs, C., Dorant, E., Hageman, G., and Stobberingh, E. (1999). Antibacterial effect of garlic and omeprazole on Helicobacter pylori. J. Antimicrob. Chemother. *43*,837- 839.

Kakizoe, T (2000). Asian studies of cancer chemoprevention: latest clinical results. Eurcpean J. of Cancer. *36*,1303-1309.

Kampen, J. V.; Robertson, H.; Hagg, T. and Drobitch, R. (2003). Neuroprotective actions of the ginseng extract G115 in two rodent models of Parkinson's disease. Experimental Neuology. *184*, 512-529.

Kaplan, M. M. (1987). Laboratory tests In: *Diseases of the liver*. Schiff L.; E. R. Schiff, E. R. Eds. J. B. Lippincott Co., Philadelphia pp 219-237.

Kik, C., Kahane, R., and Gebhardt, R. (2001). Garlic and health. Nutritional Metabolism and Cardiovascular Disease *11*, 57–65.

Kim, H. J., Chun, Y. J., Park, J. D, Kim, S. I., Roh, J. K., and Jeong, T. C. (1997). Protection of rat liver microsomes against carbon tetrachloride-induced lipid peroxidation by red ginseng saponin through cytochrome P450 inhibition. Planta Med 63(5), 415-418.

Kuriyama, S.; Fidalgo-Neto, A. and Chahoud, I. (2003). Effect of low dose mono-ortho 2,3′, 4, 4′, 5 pentachlorobiphenyl on thyroid hormone status and EROD activity in rat offspring: consequences for risk assessment, Toxicol. *186*,11-20.

Lin, C. C., Hsu, Y. F., Lin, T. C., Hsu, F. L., and Hsu, H. Y. (1998). Antioxidant and hepatoprotective activity of punicalgin and punicalin and carbon tetrachloride- induced liver damage in rats. J. Pharm. Pharmacol. *50*, 789-794.

Loo, W. T. Y., Cheung, M. N. B. and Chow, L. W. C. (2004). The inhibitory effect of a herbal formula comprising ginseng and carthamus tinctorius on breast cancer. Life Sci. *76*, 191-200.

Maclean, M. R., Twaroski, T. P. and Robertson, L. W. (2000). Redox cycling of 2- (x'-mono, -di, -trichlorophenyl)- 1,4 –benzoquinones, oxidation products of polychlorinated biphenyls. Arch. Biochem. Bioohys. *376*, 449- 455.

MacLellan, K., Singh, A., Chu, I.; and Villeneuve, D. C. (1994). Subchronic toxicity of 3,3',4,4'-tetrachlorobiphenyl in the rat liver: an electron microscope study. Histopathol. 9: 453-459.

Mannaa, F., Abdel-Wahhab, M. A., Ahmed, H. H. and Park, M. H. (2006). Protective role of *Panax ginseng extract* standardized with ginsenoside Rg3 against acrylamide-induced neurotoxicity in rats. J. Appl. Toxicol. *26*, 198-206.

McGavin, D.M.,Carlton, W.W. and Zchary, J.F.(2001). Special veterinary Pathology. Mosby,Inc. aharcourt health science company 11830 Westline industrial drive st. Louis. Missouri 63146.

Minh, N. H., Someya, M., Minh, Tu B., Kunisue, T., Iwata, H., Watanabe, M., Tanabe, S., Viet, P. H. and Tuyen, B. C. (2004). Persistent organochlorine residues in human breast milk from Hanoi and Hochiminh city, Vietnam: contamination, accumulation kinetics and risk assessment for infants. Environ. Poll. *129*, 431- 441.

Oakley. G. G., Devanaboyina, U., Robertson, L. W. and Gupta, R. C. (1996). Oxidative DNA damage induced by activation of polychlorinated biphenyls (PCBs): implications for PCB-induced oxidative stress in breast cancer. Chem. Res. Toxicol. *9*, 1285-1292.

Paglia D. E. and Valentine, W. N. (1967). Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. J. Lab. Clin. Med. *70*, 158-169.

Panwar, M., Kumar, M., Samarth, R., and Kumar, A. (2005). Evaluation of chemopreventive action and antimutagenic effect of the standardized *Panax ginseng* extract, EFLA400, in swiss albino mice. Phytother. Res. *19*, 65-71.

Parkinson, A., Safe, S. H., Robertson, L. W., Thomas, P. E. and Ryan, D. E. (1983). Immunochemical quantitation of cytochrome P-450 isozymes and epoxide hydrolase in liver microsomes from polychlorinated or polybrominated biphenyl-treated rats, A study of structure activity relationships, J. Biol. Chem. *258*, 5967-76.

Pelissier, M. A., Frayssinet, C., Boisset, M. and Albrecht, R. (1992). Effect of phenoclor DP6on enzyme-altered foci and lipid peroxidation in livers of aflatoxin B_1- initiated rats, Food Chem. Toxicol., *30*, 133-137.

Perg, D., Tampal, N., Espandiari, P. and Robertson, L. W. (2001). Distribution and macromolecular binding of benzo (α) pyrene and two polychlorinated biphenyl congenersin female mice, Chem. Biol. Interact., *137*, 243-258.

Ramadass, P., Meerarani, P., Toborek, M., Robertson, L. W. and Hennig, B. (2003). Dietary Flavonoids Modulate PCB-induced oxidative stress, CYP1A1 induction, and AhR-DNA binding activity in vascular endothelial cells. Toxicol. Sci. *76*, 212 –219

Rastogi, R., Srivastava, A. K. and Rastogi, A. K. (2001). Long term effect of aflatoxin B_1 on lipid peroxidation in rat liver and kidney: effect of picroliv and silymarin. Phytother Res. *15*, 307-10.

Robertson, L. W. and Hansen, L. G. (2001). PCBs: Recent advances in environmental toxicology and health effects. University Press of Kentucky, Lexington, KY.

Roy, A. V. (1970). Rapid method for determining alkaline phosphatase activity in serum with thymolphthalin. J. Clin. Chem. *16*, 431-436.

Safe, S. H. (1994). Polychlorinated piphenyls (PCBs). environmental impact, biochemical and toxic responses and implications for risk assessment. Crit. Rev. Toxicol., *24*, 87-149

SAS Institute (1982). *SAS User's Guide*: Statistics. 1982 Edition, SAS Institute Inc., Cary, NC.

Seegal, R.F., Okoniewiski, R.J. and Bemis, J.C. (2002). Polychlorinated Biphenyls Alter Extraneuronal but not tissue Dopamine concentrations in adult rat striatum : An *in vivo* Microdialysis study Environ . Health Pers. *110*, 113 -117.

Siegers, C. P., Stefen, B., Robke, A. and Pentz, R. (1999). The effects of garlic preparations against human tumor cell proliferation. Phytomedicine. *6*, 7-11.

Singh, A. and Shukla, Y. (1998). Antitumour activity of diallyl sulphide in two-stage mouse skin model of carcinogenesis. Biomed. Environ. Sci. *11*, 258-263.

Srinivasan, A., Lehmler, H. J., Robertson, L. W. and Ludewig, G. (2001). Production of DNA strand breaks in vitro and reactive oxygen species *in vitro* and in HL-60 cells by PCB metabolites. Toxicol. Sci. *60*, 92-102.

Storelli, M. M., Giacominelli-Stuffler, R., Storelli, A. and Marcotrigiano, G. O. (2003). Polychlorinated biphenyls in seafood: contamination levels and human dietary exposure. Food Chem. *82*, 491- 496.

Sumioka, I., Matsura, T., Kasuga, S., Itakura, Y. and Yamada, K. (1998). Mechanisms of protection by S-allylmercaptocystiene against acetaminophen-induced liver injury in mice. Jpn. J. Pharmacol. *78*, 199-207.

Suttle, N. F. (1986). Copper deficiency in ruminants, recent developments. The Veterinary Record *119*, 519-522.

The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical
Physiology (1974). Recommended methods for the determination of four enzymes in blood. Scand. J. Clin. Lab. Invest. 33, 291 – 306.

Twaroski, T. P., OBrien, L. and Robertson, L.W. (2001). Effects of selected polychlorinated biphenyl (PCB) Cogeners on hepatic glutathione, glutathione related enzymes, and selenium status implications for oxidative stress, Biochem. Pharma., 62, 273 -281

Wahlefeld, A. W. (1974). Method of enzymatic analysis. H. U. Bergmeyer, Ed. New York, NY: Academic Press: 1831-1835.

Waller, R. A. and Duncan, D. B. (1969). A Bayes rule for the symmetric multiple comparison problems. I. Am. Stat Assoc. 64, 1484-1503.

Weisburger, J. H. (2002). Lycopene and tomato products in health promotion. Exper Biol Med 227, 924–927.

Zhang, D., Yasuda, T., Yu, Y., Zheng, P., Kawabata, T., Ma, Y., Okada, S. (1996). Ginseng extract scavenges hydroxyl radical and protects unsaturated fatty acids from decomposition caused by iron-mediated lipid peroxidation. Free Radical Biol Med 20 (1), 145-150.

تأثير الثوم ومستخلص الجينسينج ضد الضغط التأكسدى الناتج عن المركبات ثنائية الفينيل عديدة الكلور PCBs في الفئران

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تعتبر مركبات ثنائية الفينيل عديدة الكلور (PCBs) من الملوثات البيئية عالية الثبات والتي كانت تستخدم علي نطاق واسع في كثير من التطبيقات الصناعية المختلفة. تهدف هذه الدراسة الى تقييم الثوم ومستخلص الجينسينج الواقي ضد الإجهاد التأكسدى الناتج عن المعاملة بمركبات ثنائية الفينيل عديدة الكلور. استخدمت في هذه الدراسة ٨٠ من إناث الفئران قسمت الى ثمان مجموعات شملت المجموعة المقارنة والمجموعة المعاملة بمركبات ثنائية الفينيل عديدة الكلور (٢٥٠ ميكروجرام/كجم من وزن الجسم) والمجموعات المعاملة بمستخلصات الثوم (٥مجم/كجم من وزن الجسم) والجينسنج (٢٠ مجم/ كجم من وزن الجسم) بمفرديهما أو في مخلوط مع أو بدون مركبات ثنائية الفينيل عديدة الكلور لمدة ١٥ يوما.

في نهاية التجربة تم جمع عينات دم للدراسات البيوكيميائية وكذلك تم أخذ عينات من الكبد والكلي لتقدير الأنزيمات المضادة للأكسدة وتأكسد الدهون وكذلك الدراسات الهستولوجية.

أوضحت النتائج ان المعاملة بمركبات ثنائية الفينيل عديدة الكلور أدت الى زيادة معنوية في مستوي إنزيمات ووظائف الكبد والكلي (الأنزيمات الناقلة لمجموعة الأمين والفوسفاتيز القلوي وتركيزات حمض اليوريك والكرياتينين والجلسريدات الثلاثية) في الدم وكذلك مستوى تأكسد الدهون في الكبد. كما أدت المعاملة إلي تغيير معنوي في مستوي الأنزيمات المضادة للأكسدة (SOD, GPX) مع حدوث تغيرات باثولوجية في أنسجة الكبد والكلي. وأدت المعاملة بالمستخلصات إلي حدوث تحسنا معنويا في كل القياسات البيوكيميائية والصورة الهستولوجية في الكبد والكلي. نستخلص من هذه الدراسة ان كلا من الثوم ومستخلص الجينسينج لهما القدرة علي الحماية من التأثيرات الضارة للمركبات ثنائية الفينيل عديدة الكلور كما ان مخلوط الثوم والجينسنج كان أكثر فعالية من الثوم ومستخلص الجينسينج بمفرديهما.