

Evaluation of radio protective effects of Coriander (*Coriandrum sativum* L.) in male rats

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ABSTRACT

Radiation is one of the most widespread sources of environmental stress in living environment which cause oxidative stress and metabolic changes. The basic purpose of this work was to determine the radio protective ability of Coriander (*Coriandrum sativum* L.) seeds against whole body gamma irradiation of rats. The study was conducted on thirty two male rats which were classified into four equal groups. Control group: (normal, untreated). Coriander aqueous extract group (C.E.): rats received orally by gavage the aqueous extract of Coriander seed powder (300 mg/ kg b. wt. / day for 42 days). Irradiated group: rats were subjected to whole body γ -irradiation at dose of 4 Gy delivered as a single exposure dose. Combined treatment group: rats received orally C.E. (300 mg/ kg b. wt. / day) for 42 days? at day 35 of C.E. treatment the rats were irradiated at dose level of 4 Gy. The animals exposed to gamma radiation showed a significant increase in serum aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP) and lactate dehydrogenase (LDH), urea (U), creatinine (Cr), total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C) and tissue thiobarbituric acid reactive substance (TBARS). On the other hand, a significant decrease was recorded in serum total protein (T.P), albumin (Alb), high density lipoprotein cholesterol (HDL-C). A decrease of liver and kidney reduced glutathione (GSH) content, superoxides dismutase (SOD) and catalase (CAT) activities were reported. Treatment of rats with C.E. significantly reduced the radiation-induced serum biochemical disorders which was associated with significant amelioration in the oxidant / antioxidant status of liver and kidney tissues. It could be concluded that C.E. might protect from radiation induced damage due to its ability to scavenge free radicals.

*Key words: Gamma Irradiation/ Coriander / Liver Function/ Kidney Function/
Oxidative stress.*

INTRODUCTION

Ionizing radiation is used in a lot of various purposes including therapeutic, industrial and other applications apart from for generation of nuclear power and developing new high-yielding varieties of new crops and enhancing storage-period of food materials ^(1 and 2). Moreover, Radiation is one of the most severe causes of oxidative stress mediated by free radical flux. This flux interferes with oxidation/reduction-based physiological mechanisms inside the mammalian body system. Radiation protection is an area of great significance due to its possible applications in planned radiotherapy as well as unplanned radiation exposure ^(3 and 4). Research in the development of protectors worldwide has focused on screening a variety of chemical and biological compounds. Various drugs from natural or synthetic origin have been evaluated extensively for their radioprotective potentials in both *in vitro*

and *in vivo* models ^(3 and 5). However, the fact that remains that there is not a single radioprotective drug available which meets all the prerequisites of an ideal radioprotector, i. e., produces no cumulative or irreversible toxicity provides effective long-term protection, remains stable for a number of years without losing shelf life, and can be easily administrated ⁽⁶⁾. In view of this, the search for less toxic and more potent radioprotector drugs continues.

Herbal drugs have been utilized since ancient times for curing various diseases and other disorders. Even today, more than 70% of the world's population still depends on plant-based remedies to meet their health care needs ⁽⁷⁾. Plants constitute an important source of natural products which differ widely in their structures, biological properties and mechanism of action. Various phytochemical components especially polyphenols, flavonoids, phenolic acids etc. are responsible for the free radical scavenging and antioxidant activity of the plants. Polyphenols possess many biological affects, mainly attributed to their antioxidant activities in scavenging free radicals, inhibition of peroxidation and chelation of transition metals ⁽⁸⁾.

It is, therefore, logical to expect that plants may protect against radiation-induced reactive oxygen species (ROS) and reactive nitrogen species (RNS) mediated damage ⁽⁶⁾. *Coriandrum sativum* L. is an important spice crop and occupies a prime position in flavoring substances. The seeds are used in medicine as a carminative, diuretic and also used in the preparation of many house hold medicines to cure bed cold, seasonal fever, nausea and stomach disorders. Coriander oil is used in baked foods, condiments and also functions as an essential ingredient in curry mixes ⁽⁹⁾.

The study of antioxidants that are ubiquitously present in spices is gaining momentum in human health, as these are easily absorbable in human system. Among such plants, *Coriandrum sativum* is well known for its antioxidant properties. Phytochemical constituents of CS seeds have been studied extensively and its analysis has revealed the presence of polyphenols (rutin, caffeic acid derivatives, ferulic acid, gallic acid, and chlorogenic acid), flavonoids (quercetin and isoquercetin) and β carotenoids ⁽¹⁰⁾. Most of these compounds are known to inhibit free radicals generated in the cellular system, when they are obtained through the diet.

In the light of aforementioned medical properties of coriander, this study was carried out to investigate the possible protective properties of coriander extract against oxidative stress and related biochemical parameters in liver and kidney of gamma irradiated rats.

MATERIALS AND METHODS

Materials:

Coriander seeds were purchased from local herbal market (Cairo, Egypt).

Preparation of C.E.:

Dried coriander seeds were ground to a fine powder, of which 10 g were added to 500 ml distilled water. After 24 h maceration was done at room temperature (37 °C), the mixture was then heated for 30 min in the water bath at 65 °C. The extract was filtered and stored at 4 °C then used to treat animals as needed.

Radiation facility:

Source of irradiation was Canadian gamma cell-40, (¹³⁷Cs). Whole body gamma irradiation was performed at NCRRT, Cairo, Egypt. Animals were irradiated at an acute single dose level of 4 Gy delivered at a dose rate of 0.42 Gy/ min at the time of the experiment.

Experimental animals:

Adult male albino rats weighing 140 - 150 g were used in this study. The animals were housed in plastic cages under standard laboratory conditions including all hygienic measures with constant illumination and ventilation and normal conditions of temperature and humidity. They were maintained on a standard commercial rodent chow containing all nutritive elements and have free access to tap water.

Experimental design:

The animals were randomly assigned into 4 groups (8 rats for each group). Control group (normal, untreated), received distilled water. C.E. group: rats administered orally by gavage C.E. at a dose of 300 mg/kg b. wt. / day⁽¹¹⁾ for six weeks. Irradiated group: rats were subjected to whole body γ - irradiation at dose 4 Gy delivered as single exposure dose. Combined treatment group: rats administered with C.E. at a dose of 300 mg/ kg b. wt. by oral gavage once daily for 42 days. At day 35 of C.E. treatment rats were irradiated at dose level of 4Gy.

Sample Collection:

Animals were fasted overnight prior to sacrificing. Rats from different groups were sacrificed at the 7th day after irradiation. Blood samples were collected and serum was separated. Liver and kidney were rapidly excised and homogenized in physiological saline solution. The homogenates were centrifuged at 3000 rpm for 15 min and the supernatants were used for the biochemical analysis.

Biochemical parameters:

The activity of serum AST and ALT was assayed by the method of Reitman and Frankel⁽¹²⁾. ALP activity was estimated according to the method of Kind and King⁽¹³⁾, as well as serum LDH was assessed according to Rosalk⁽¹⁴⁾. Serum level of TC, TG and HDL-C was determined according to Allain *et al.*⁽¹⁵⁾, Fossati and Principe⁽¹⁶⁾ and Demacker *et al.*⁽¹⁷⁾, respectively. LDL-C was evaluated according to Friedewald *et al.*⁽¹⁸⁾ formula, by the following equation: $LDL-C (mg/dl) = TC - (TG/5 + HDL-C)$. Serum U and Cr were estimated according to Patton and Crouch⁽¹⁹⁾ and Henery⁽²⁰⁾, respectively. T.P and Alb concentrations were measured using the methods of Lowry *et al.*⁽²¹⁾ and Doumas *et al.*⁽²²⁾, respectively. Lipid peroxidation content was determined by quantifying TBARS in tissue homogenates according to the colorimetric method described by Yoshioka *et al.*⁽²³⁾ SOD activity was measured by the method of Minami and Yoshikawa⁽²⁴⁾, CAT activity was determined according to the method described by Aebi⁽²⁵⁾. Determination of GSH content was performed according to Beutler *et al.*⁽²⁶⁾.

Statistical analysis:

Analysis of variance (ANOVA) was conducted for all data using the general linear model (GLM) (SAS Institute²⁷). Duncan's multiple-range test was used for comparison between treatments⁽²⁸⁾. Data were presented as means \pm standard error. A value at $P < 0.05$ was taken as criterion of significance.

RESULTS

Effect of gamma rays alone and ameliorating effect of CS extract prior radiation exposure on some hepatic biochemical variables of various groups were assessed and are presented in Table (1). It is clear from the results that treatment of rats with gamma rays showed a significant elevation in AST, ALT, ALP and LDH activities compared to control group animals. The liver marker enzymes were significantly suppressed by administration of C.E. ($p < 0.05$ vs. irradiated rats).

Table 1: Effect of C.E. administration to irradiated rats on serum liver markers

Group	AST (U ml ⁻¹)	ALT (U ml ⁻¹)	ALP (U l ⁻¹)	LDH (U l ⁻¹)
Control	28.54±1.61 ^b	14.84±0.94 ^c	79.68±1.27 ^b	187.10±5.56 ^b
C.E.	26.08±1.23 ^b	14.26±0.86 ^c	80.61±2.07 ^b	176.13±4.27 ^b
Irrad.	50.27±2.46 ^a	46.61±1.89 ^a	140.84±1.06 ^a	292.19±5.60 ^a
C.E. + Irrad.	33.19±2.27 ^b	28.29±0.83 ^b	92.86±0.93 ^b	199.17±4.79 ^b

Data = means ± SE; n = 8 rats. Values not sharing a common superscript letter differ significantly at P=0.05.

Effect of gamma rays alone and ameliorating effect of CS extract on some renal biochemical variables of various groups were assessed and are presented in Table (2). In comparison to normal control rats, a significant (p< 0.05) increase in the U and Cr level was recorded in radiation exposed rats. While, a significant (p< 0.05) decrease in T.P and Alb content followed by radiation exposure was also noticed in irradiated group, as compared to control animals. Rats treated with C.E. significantly (p< 0.05) reduced gamma exposure induced increase in the levels of U and Cr, as compared to radiation treated animals. On the other hand, radiation exposure -induced depletion in serum T.P and Alb content was insignificantly ameliorated by treatment of rats with C.E. when compared with irradiated animals.

Table 2: Effect of C.E. administration to irradiated rats on serum kidney indexes

Group	U (mg dl ⁻¹)	Cr. (mg dl ⁻¹)	T. p. (g dl ⁻¹)	Alb. (g dl ⁻¹)
Control	31.09± 2.16 ^b	0.71 ± 0.004 ^{bc}	6.51±0.42 ^a	4.13± 0.29 ^a
C.E.	32.97± 2.11 ^b	0.67 ± 0.003 ^c	6.23±0.37 ^a	3.94 ± 0.33 ^a
Irrad.	54.45 ± 2.70 ^a	0.89 ± 0.004 ^a	4.89±0.22 ^b	3.08 ± 0.24 ^b
C.E. + Irrad.	36.18 ± 1.82 ^b	0.76 ± 0.002 ^b	5.96±0.29 ^a	3.83 ± 0.19 ^a

Legends as table 1

As shown in Table (3) ?- irradiation of rats significantly (P<0.05) altered serum lipid profile. The level of TC, TG and LDL-C was significantly (P<0.05) increased and HDL-C was significantly decreased in the irradiated rats group when compared to control group. The administration of C.E. significantly (P<0.05) lower the alteration in the serum lipid profile levels when compared to the irradiated rats.

Table 3: Effect of C. E. administration to irradiated rats on serum lipid profile

Groups	TC (mg dl ⁻¹)	TG (mg dl ⁻¹)	HDL-C (mg dl ⁻¹)	LDL-C (mg dl ⁻¹)
Control	151.71±4.27 ^c	113.38±2.87 ^c	44.75±1.27 ^a	84.28±3.42 ^c
C. E.	148.27±3.81 ^c	109.07±3.27 ^c	46.22±2.41 ^a	80.24±3.25 ^c
Irrad.	207.22±5.62 ^a	182.03±3.58 ^a	36.84±1.96 ^b	134.01±4.62 ^a
C. E. +Irrad.	185.60±4.35 ^b	157.22±4.16 ^b	40.88±1.76 ^{ab}	113.28±2.68 ^b

Legends as table 1

Effect of gamma rays alone and ameliorating effect of C.E. on LPO and antioxidant related parameters in liver and kidney of various groups were assessed and are presented in Table (4 & 5). Whole body gamma irradiation of rats caused a significant (p< 0.05) increase in the level of TBARS in liver and kidney. Whereas, significant decrease (p< 0.05) in SOD and CAT activities, and GSH content of rats were observed in irradiated animals, in comparison with those of control group. However, treatment with C.E. caused a significant reduction (p<0.05) in the hepatic and renal LPO

level when compared with gamma irradiated group. A significant increase ($p < 0.05$) in SOD, CAT activities and GSH content were observed in rats treated with C.E., compared to gamma rays exposed group.

Table 4: Effect of C.E. and/ or Irrad. on the level of tissue TBARS and GSH content of rats

Group	TBARS (n mol/g tissue)		GSH (mg/g tissue)	
	Liver	Kidney	Liver	Kidney
Control	161.88 ± 1.91 ^c	102.78 ± 2.42 ^c	51.38 ± 1.52 ^a	34.29 ± 2.46 ^a
C.E.	155.91 ± 4.20 ^c	100.94 ± 2.49 ^c	52.40 ± 2.67 ^a	34.77 ± 1.58 ^a
Irrad.	220.85 ± 5.36 ^a	126.89 ± 4.81 ^a	26.52 ± 2.72 ^c	19.28 ± 1.51 ^b
C.E. + Irrad.	178.68 ± 5.12 ^b	109.93 ± 1.34 ^b	42.25 ± 2.12 ^b	31.98 ± 2.69 ^a

Legends as table 1

Table 5: Changes in the activity of hepatic and renal SOD and CAT of experimental rats

Group	SOD (U/mg protein)		CAT(U/mg protein)	
	Liver	Kidney	Liver	Kidney
Control	51.61 ± 3.19 ^a	42.36 ± 1.76 ^a	15.94 ± 0.27 ^a	13.68 ± 0.18 ^a
C.E.	52.54 ± 1.93 ^a	44.59 ± 2.48 ^a	16.71 ± 0.13 ^a	12.98 ± 0.21 ^a
Irrad.	29.47 ± 1.78 ^b	26.44 ± 1.85 ^b	10.31 ± 0.12 ^c	8.53 ± 0.16 ^b
Irrad. + C.E.	48.40 ± 4.04 ^a	39.61 ± 1.71 ^a	13.27 ± 0.31 ^b	12.47 ± 0.13 ^a

Legends as table 1

In the present study, rats treated with C.E. without exposure to radiation revealed non significant changes in the investigated biochemical parameters indicating its safe use.

DISCUSSION

Ionizing radiation produces harmful effects on the organisms and due to the wide spread use of radiation in diagnosis therapy, industry, therefore, pharmacological intervention could be most potent strategy to protect human or ameliorates the deleterious effect of ionizing radiation ⁽⁴⁾. Ionizing radiations induce significant elevations in the physiological and metabolic processes, as well as, disorders in blood biochemical parameters, ⁽²⁹⁾ and causing chain reaction of oxidation ⁽³⁰⁾.

The present study has investigated the efficacy of *Coriandrum sativum*, which is considered a traditional natural medicine and an edible vegetable, in minimizing the hazardous effects of ionizing radiation using a rat model. It is evident from the results of the current investigation that supplementation of C.E. protected animals from harmful effects of irradiation in general and oxidative stress in particular. Rats administered C.E. restored the altered levels to some extent suggesting that the active ingredients in the coriander possess antioxidant properties and protects against radiation induced oxidative stress.

In current findings, rats whole body γ - irradiated showed elevation in serum AST, ALT, ALP and LDH activities. Liver enzymes such as ALT, AST, ALP and LDH are marker enzymes for liver function and integrity ⁽³¹⁾. These enzymes are usually raised in acute hepatotoxicity or mild hepatic cellular injury. The C.E. mediated suppression of the increased AST, ALT, ALP and LDH activities proposed the possibility of the extract to give protection against hepatic injury upon gamma radiation of rats. The efficiency of C.E. was due to presence of several pharmacological effects such as hypotensive ⁽³²⁾, Hypocholesterolemic ⁽³³⁾, hypoglycaemic ⁽³⁴⁾ and hepatoprotective ⁽³⁵⁾ properties.

Thus, the present study using coriander recommended that this plant is capable of scavenging radiation induced free radical generation. Kunwar et al. ⁽³⁶⁾ reported that ionizing radiation induce augmentation in the levels of serum AST and ALT that were significantly ameliorated by treatment with natural radio-protector which accord with the present findings.

The exposure of rats to a single dose of γ - radiation led to a significant increase in serum U and Cr levels (Table 2). These results are in agreement with those of Mohamed ⁽³⁷⁾ who attributed these increments in U and Cr level to the increased protein breakdown as the U is the end product of protein catabolism. Previously, Moulder *et al.* ⁽³⁸⁾ sustained that rats exposure to ionizing radiation is accompanied by an increase in the serum U and different tissues free ammonia. Also, ionizing radiation induce extensive retention in daily excreted urine that lead to increased Cr and U levels in the blood ⁽³⁹⁾. Increase in serum U was due to increase in glutamate de-hydrogenase enzyme as a result of irradiation and this may increase carbamoyl phosphate synthetase activity leading to increase in U concentration ⁽⁴⁰⁾. Feeding rats with *Coriandrum sativum* powder induced a significant decrease in the levels of serum U and Cr compared to rats left without any *Coriandrum sativum* administration ⁽⁴¹⁾.

Level of total protein is a rough measure of protein status but reflects major functional changes in kidney and liver functions. Table (2) shows a significant decrease ($P < 0.05$) in serum T.P and Alb levels in γ -irradiated rats as compared with the corresponding control rats. Moulder *et al.* ⁽³⁸⁾ declared degradation of protein by exposure of rats to ionizing radiation. This marked decrease in T.P may indicate protein catabolism dysfunction ⁽⁴²⁾. As well as, may be attributed to defects in protein biosynthesis and damage of vital biological processes or due to change in the permeability of liver, kidney and other tissues resulting in leakage of protein via the kidney ⁽⁴³⁾. The decline in the level of Alb concentration could be due to enhanced degradation as well as enhanced loss of Alb through the gastrointestinal tract ⁽⁴⁴⁾. Administration of C.E. significantly increased T.P and Alb contents. The improvements in the level of T.P after administrated rats with C.E. are in agreement with that found by Haggag et al. ⁽⁴¹⁾. The ability of *Coriandrum sativum* to maintain the T.P may be due to the nontoxic antioxidant constituents present in the powder ⁽⁴⁵⁾. Gamma irradiation caused significant increase in the levels of kidney and liver peroxidation but treatment of rats with natural radio-protector significantly decreased the level of kidney and liver function when compared to the irradiated group ⁽³¹⁾. These conclusions agree with the present results.

Gamma irradiation of rats induced a significant elevation in the level of serum cholesterol on the first day post-irradiation but administration of rats with natural radio-protector before irradiation exposure restored the normal level⁽⁴⁶⁾. These results agree with the present data. The hyperlipidaemic state observed after irradiation could be attributed to the mobilization of fats from the adipose tissue to the blood stream ⁽⁴⁷⁾, in addition to mitochondrial dysfunction ⁽⁴⁸⁾. Irradiation induces hyperlipidemia through cell membrane destruction, enhancement of lipid metabolism, cholesterol release and triglycerides synthesis ⁽⁴⁹⁾. Free radicals destruct cell membranes and enhance cholesterol release and increase lipid peroxidation ⁽⁵⁰⁾. Increased triglycerides after irradiation might result from inhibition of lipoprotein lipase activity leading to reduction in uptake of triglycerides by adipose cells ⁽⁵¹⁾. The hypercholesterolemia and hypertriglyceridemia might be attributed to an increase in the activity of 3-hydroxyl methyl glutaryl COA, as an early reaction necessary for the restoration of biomembranes ⁽⁵²⁾. In addition to decreased fatty acid oxidation ⁽⁵³⁾. Moreover, radiation enhanced the process of lipid peroxidation which results in cell membrane damage and the release of fats from peripheral and adipose tissues to the blood ⁽⁵¹⁾. This increase may be due to increased lipolysis of triglycerides in

adipose tissues which liberates free fatty acids which are taken up by the liver tissue and resynthesized, leading to the hypertriglyceridemic condition ⁽⁵¹⁾.

Kousar et al. ⁽⁵⁴⁾ recorded that treatment of rabbits with *coriandrum sativum* extract (100 mg/kg) for three weeks exerted a significant antilipidemic effect against salbutamol-induced myocardial infraction by lowering the level of serum LDL-C and TG and increasing the level of HDL-C in serum of rabbits. Coriander contain significant amount of polyphenols. Polyphenols are potent antioxidants and reduces the oxidative stress. Polyphenols have great importance in the prevention of free radicals associated diseases.

Lipid peroxidation, a process induced by free radicals leads to oxidative deterioration of polyunsaturated lipids ⁽⁵⁵⁾. Under normal physiological conditions, only low levels of lipid peroxides occur in body tissues. The excessive generation of free radicals leads to peroxidative changes that ultimately result in enhanced lipid peroxidation ⁽⁵⁶⁾. Radiation exposure has been reported to be associated with increased disruption of membrane lipids leading to subsequent formation of peroxide radicals ⁽⁵⁷⁾.

In the present investigation, such a disruption of membrane lipids led to the possibly accumulated and increased level of TBARS in the hepatic and renal tissues of irradiated rats. In addition, insufficient levels of antioxidants to scavenge peroxy-radicals during radiation could also have contributed to the elevated level of TBARS in irradiated rats ⁽⁵⁸⁾. Antioxidants are necessary for preventing the formation of free radicals and they inhibit some of the deleterious actions of reactive oxygen species on lipids, DNA and proteins ⁽⁵⁹⁾.

Glutathione is the most abundant nonprotein sulfhydryl containing compound and constitutes the largest component of the endogenous thiol buffer ⁽⁶⁰⁾. Assessment of GSH in biological samples is essential for evaluation the redox homeostasis and detoxification status of cells in relation to its protective role against oxidative and free radical mediated cell injury ⁽⁶¹⁾. The present study recorded a significant depletion of GSH content in hepatic and renal tissues of irradiated animals as compared to control group due to oxidative stress. The depletion of GSH content in irradiated rats might be due to enhanced utilization during detoxification process. The resultant reduction in GSH level may thus increase susceptibility of the tissue to oxidative damage including lipid peroxidation. However, Glutathione has diverse cellular functions in addition to its antioxidant properties including enzymatic conjugation through the glutathione- S transferase family of proteins and non enzymatic conjugation to cytotoxic compounds. Glutathione may react with H₂O₂ and lipid peroxides by action of GSH-PX to reduce their toxicity ⁽⁶²⁾.

Depletion in GSH level after radiation exposure might be resulted from diffusion through impaired cellular membranes and / or inhibition of GSH synthetase. Also, the decrease in the content of organs GSH might result from a diminished activity of glutathione reductase and a deficiency of NADPH which is necessary to change the oxidized glutathione to its reduced form ⁽⁶³⁾. GSH can function as an antioxidant in many ways. It can react chemically with singlet oxygen, superoxide and hydroxyl radicals and therefore function directly as a free radical scavenger. GSH may stabilize membrane structure by removing acyl peroxides formed by lipid peroxidation reactions ⁽⁶⁴⁾.

The decrease in the activity of CAT could be due to a feed back inhibition or oxidative inactivation of enzyme protein caused by ROS generation ⁽⁶⁵⁾. The current study recorded a significant

depletion of CAT activity in irradiated rats. However, CAT is one of three families of primary antioxidant enzymes in mammalian cells which are critical to peroxide removal. So, the recorded depletion of CAT activity may be due to the increased utilization of this antioxidant to counteract lipid peroxidation production⁽⁶⁶⁾. Also, the decrease in the activity of CAT might be attributed to excess of ·OH resulting from water radiolysis after exposure to ionizing radiation which causes oxidative damage to enzymes that lead to the modification of the activity of CAT⁽⁶⁷⁾.

In the present study, it is reported that the administration of C.E. caused an increase in SOD, CAT activity and GSH content and decrease in LPO level in irradiated rat tissues (liver and kidney), supporting the antioxidant effect of aqueous plant extract. These results are in accordance with those of Chithra and Leelamma⁽⁶⁸⁾ who reported that *Coriandrum sativum* administration to rats significantly declined formation of lipid peroxides and increased antioxidant enzyme activities. Thus, it appears that the orally administered C.E. protects against gamma rays induced toxicity possibly through the inhibition of increased LPO level in tissues. Plant extract treatment was found to be beneficial in improving SOD enzyme activity which could explain the decrease in LPO levels. Increase in SOD activity should accelerate the removal of the ROS.

Previous studies demonstrated that *C.sativum* had strong antioxidant activity which was even superior to known antioxidants like ascorbic acid^(69, 35 and 70). The antioxidative property of coriander seed is related to the large amounts of tocopherols, carotenoids and phospholipids⁽⁷¹⁾, which act through different mechanisms. Carotenoids act as primary antioxidants by trapping free radicals and as secondary antioxidants by quenching singlet oxygen⁽⁷²⁾. Tocopherols and sterols interact with oil surfaces and release hydrogen, inhibiting the propagation step of radical reactions⁽⁷²⁾. Synergetic effects were evidenced with combinations of carotenoids and tocopherols⁽⁷²⁾. Although the exact mechanism of antioxidative action of phospholipids is still not fully established, these substances would synergistically act with tocopherols, would form barrier for O₂ between air/oil interfaces, would favor formation of Mallard like compounds with oxidation products or would chelate pro-oxidant metals with phosphate groups⁽⁷³⁾. There is another class of bioactive substances called phthalides, which have anticarcinogenic potential. They are found in umbelliferous plants like celery, parsley, cumin, dill, fennel and coriander. The phthalides are known to increase the glutathione-S-transferase level⁽⁷⁴⁾. This could thus be attributed to the possibility that coriander might provide some recovery in GSH level.

In conclusion, the results of the present study indicate that the administration of coriander to rats may prevent the deleterious effects of irradiation. The overall renal and hepatoprotective effect of coriander is probably due to a counteraction of free radicals by its antioxidant nature which decrease TBARS production and increase antioxidant GSH content, enzyme SOD and CAT activities and /or to its ability to inhibit lipid accumulation by its antilipidemic property. Therefore, it is greatly recommended to incorporate coriander as a nutritional supplement before and during radiotherapy to prevent the oxidative daily damage induced by radiotherapy.

REFERENCES

- (1) M.I. Michael and M.M. Amer; J. Rad. Res. Appl. Sci.; 3, 855 (2010).
- (2) T.K. Lee, K.F. O'Brien, W. Wang, R.M. Johnke, C. Sheng, S.M. Benhabib, T. Wang and R.R. Allison; J. Altern. Complement Med.; 16, 561 (2010).

- (3) R. Arora, D. Gupta, R. Chawla, R. Sagar, A. Sharma, R. Kumar, J. Prasad, S. Singh, N. Samanta and R. K. Sharma; *Phytotherapy Res.*; 19, 1 (2005). ?
- (4) G.C. Jagetia; *J. Clin. Biochem. Nutr.*; 40, 74 (2007).
- (5) M. Bala and H.C. Goel; *J. Environ. Pathol. Toxicol. Oncol.*; 23, 139 (2004).
- (6) R.R. Arora, S. Chawla, R.K. Singh, A.K. Kumar, S.C. Sharma, A.K. Puri, R.P. Tripathi and R.K. Sharma; Radioprotection by Himalayan High-Altitude Region Plants. In: *Herbal Drugs: A Twenty-first Century Perspective*, R.K. Sharma and R. Arora (Eds.) Jaypee Brothers Medical Publishing Limited, New Delhi; 301-325 (2006).
- (7) B. Patwardhan, A.D. Vaidya and M. Chorghade; *Current Sci.*; 86, 789 (2004).
- (8) B. Nickavar, M. Kamalinejad, M. Haj-Yahya, B. Shafaghi; *Pharm. Biol.*; 44, 208 (2006).
- (9) M.M. Sharma and R.K. Sharma; *Biochem. Soc. Trans.*; 33, 1375 (1999).
- (10) E.A. Melo, F.M. Bion, J.M. Filho and N.B. Guerra; *Eur. J. Lipid Sci. Tech.*; 105, 483 (2003).
- (11) L. Kansal, V. Sharma, A. Sharma, S. Lodi and S.H. Sharma; *International J. Appl. Biol. Pharm. Technol.*; 2, 65 (2011).
- (12) S. Reitman and S. Frankel; *Am. J. Clin. Pathol.*; 28, 56 (1957).
- (13) P. Kind and E. King; *J. Clin. Pathol.*; 7, 322 (1954).
- (14) S.B. Rosalk; In *advanced clin. chem.*, Academic Press. Chap.; 17, 53 (1957).
- (15) C.C. Allain, L.S. Poon, C.S. Chan, W. Richmond and P.C. Fu; *Clin. Chem.*; 20, 470 (1974).
- (16) P. Fossati and L. Prencipe; *Clin. Chem.*; 28, 2077 (1982).
- (17) P.N. Demacker, H.E. Vos-Janssen, A.G.M. Hifmans, A. Van't Laar and A.P. Jansen; *Clin. Chem.*; 26, 1780 (1980).
- (18) W.T. Friedewald, R.I. Levy and D.S. Fredrickson; *Clin. Chem.*; 18, 499 (1972).
- (19) C. Patton and S. Crouch; *Anal. Chem.*; 49, 464 (1977).
- (20) R.J. Henery; *Clin. Chem. Principle and Technics.*, 2nd Ed. Harper and Row.; 521 (1974) .
- (21) O.H. Lowry, N.I. Rosebrough, A.I. Farr and R.J. Randall; *Biol. Chem.*; 193, 265 (1951).
- (22) B. Doumas, W. Waston and H. Biggs; *Clin. Chem. Acta.*; 31, 87 (1971).
- (23) T. Yoshioka, K. Kawada, T. Shimada and M. Mori; *Am. J. Obstet. Gynecol.*; 135, 372 (1979).
- (24) M. Minami and H. Yoshikawa; *Clin. Chem. Acta.*; 92, 337 (1979).
- (25) H. Aebi; *Methods Enzymol.*; 105, 121 (1984).
- (26) E. Beutler, O. Duron and B.M. Kelly; *Lab. Clin. Med.*; 61, 882 (1963).
- (27) SAS 'Statistical Analysis System', SAS / STAT User's guide. SAS Institute Inc Cary NC, USA (2004).
- (28) D.B. Duncan; Multiple range and multiple F tests. *Biomet.*; 11, 1 (1955).
- (29) F.S El-Masry and T.M. Saad; *Isotope and Rad. Res.*; 37, 1261 (2005).
- (30) A.A. Ammar; *Isotope and Rad. Res.*; 41, 911 (2009).
- (31) O.A. Adaramoye, O.O. Okiti and E.O. Farombi; *Exp. Toxicol. Pathol.*; 63, 635 (2011).
- (32) G.A. Burdock and I.G. Carabin; *Food Chem. Toxicol.*; 47, 22 (2008).
- (33) P. Dhanapakiam, J.M. Joseph, V.K. Ramaswamy, M. Moorthi, A.S. Kumar; *J. Environ. Biol.*; 29, 53 (2008).
- (34) M. Eidi, A. Eidi, A. Saeidi, S. Molanaei, A. Sadeghipour, M. Bahar and K. Bahar; *Phytother. Res.*; 23, 404 (2009).
- (35) I. Samojlik, N. Lakic, N. Mimica-Dukic, K. Dakovic-Svajcer and B. Bozin; *J. Agric. Food Chem.*; 56, 139 (2010).
- (36) A. Kunwar, P. Bansal, S.J. Kumar, P.P. Bag, P. Paul, N.D. Reddy, L.B. Kumbhare and V.K. Jain; *Isotope and Rad. Res.*; 41, 911 (2010).
- (37) N. E. Mohamed; *J. Am. Sci.*; 7, 406 (2011).
- (38) J.E. Moulder, B.L. Fish, K.R. Regner, E.P. Cohen and T.J. Raife; *Rad. Res.*; 157, 199 (2002).
- (39) M. Robbins, O. Malley, Y. Davis and C. Bonsib; *Rad. Res.*; 155, 481 (2001).
- (40) L.A. Ramadan, S.A. Shouman, A.M. Sayed and O.H. El-Habit; *Pharmacol. Res.*; 43, 185 (2001).

- (41) M.H. Haggag; The 6th Arab and 3rd International Annual Scientific Conference on: Development of Higher Specific Education Programs in Egypt and the Arab World in the Light of Knowledge Era Requirements. Faculty of Specific Education Mansoura University - Egypt April; 13-14, (2011).
- (42) M.A. Abdel-Wahhab, S.H. Abdel-Azim and A.A El-Nekeety; *Toxicol.*; 52 (4) (2008).
- (43) H.M. Roushdy, H.A. Ibrahim and G.M.F. Edress; *J. Agric. Food Chem.*; 14 (1984).
- (44) M.M. Abbady; Ph.D. Thesis, Fac. of Science, Ain Shams Univ (1994).
- (45) K.G. Nevan and P.L. Vijayammal; *Fitoterapia.*; 74, 578 (2005).
- (46) M.A. El-Missiry, T.A. Fayed, M.R. El-Sawy and A.A. El-Sayed; *Ecotoxicology and Environmental Safety*; 66, 278 (2007).
- (47) T. S. Chajek, G. Friedman, O. Stein, E. Shiloni, J. Etienne and Y. Stein; *Biochim. Biophys. Acta.*; 1001, 316 (1989).
- (48) R. Nageswara, A. Madamanchi and S. Marschall; *Circul. Res.*; 100, 460 (2007).
- (49) D.A. Bodwen, P. McLea and A. Steinmetz; *Lipid Res.*; 30, 1895 (1989).
- (50) M. Karbowink and R.J. Reiter; *Proc. Soc. Exp. Biol. Med.*; 225, 9 (2000).
- (51) A. Sedlakova, Y. Timoko, E. Paulikova and K.I. Dyatelink; *Radiobiologiya*; 28, 80 (1998).
- (52) I.K. Kolomijtseva; *Radiobiologiya*; 26, 3 (1986).
- (53) S.D. Clark; *J. Nutr.*; 131, 1129 (2001).
- (54) S. Kousar, N. Jahan, U.R. Khalil and S. Nosheen; *Biosci. Res.*; 8, 8 (2011).
- (55) A. Catala; *Chem. Phys. Lipids.*; 157, 1 (2009).
- (56) R. Joshi, J.P. Kamet and T. Mukherjee; *Chem. Biol. Interact.*; 167, 125 (2007).
- (57) N. Rajapakse, M. Kim, E. Mendis and S.K. Kim; *Bioorg. Med. Chem.*; 15, 997 (2007).
- (58) K. Manda, M. Ueno, T. Moritake and K. Anzai; *Am. J. Physiol Regul. Integr. Comp. Physiol.*; 292, 18 (2007).
- (59) M.M. Boojar and A. Shockravi; *Bioorg. Med.Chem.*; 15, 3437 (2007).
- (60) A. Holmgren, C. Johansson, C. Berndt, M.E. Lonn, C. Hudemann and C.H. Lillig; *Cell Biol.Toxicol.*; 23, 129 (2005).
- (61) R. Rossi, I. Dalle- Donne, A. Milzani and D. Giustarini; *Clin. Chem.*; 52, 1406 (2005).
- (62) W.J. Davis, Z. Ronai and K.D. Tew; *J. Pharmacol. Exp. Ther.*; 296, 1 (2001).
- (63) J. Pulpanova, H. Kovarova and M. Ledvina; *Radiobiol.*; 30, 413 (1982).
- (64) A. Price, P. Lucas and P. Lea; *Expl. Biol*; 41, 1309 (1990).
- (65) Y. Ohta, N. Kongo, M. Nishimura, T. Matsura, K. Yamaa, A. Kitagawa and T.J. Kishikawa; *Pineal Res.*; 36, 10 (2004).
- (66) C. Kalpana and V.P. Menon; *Pol. J. Pharmacol.*; 56, 581 (2004).
- (67) K.C. Kregal and H.J. Zhang; *J. Environ. Biol.*; 29, 53 (2007).
- (68) V. Chithra and S. Leelamma; *Ind. J. Biochem. Biophys.*; 36, 59 (1999).
- (69) T.A. Misharina and A.I. Samusenko; *Prikl. Biokhim. Mikrobiol.*; 44, 482 (2008).
- (70) S. Sultana, F.A. Ripa and K. Hamid; *Pak. J. Biol. Sci.*; 13, 340 (2010).
- (71) M.F. Ramadan and J.T. Missel; *J. Agric. Food Chem.*; 51, 969 (2004).
- (72) D.W. Reische, D.A. Lillard and R.R. Eitenmiller; second edition. Eds. Akoh CC, Min DB, Marcel Dekker NY (USA); 489- 516 (2002).
- (73) K.M. Haila, S.M. Lievonon and M.I. Heinonen; *J. Agric. Food Chem.*; 44, 20096 (1996).
- (74) R.E.C. Wildman; *Handbook of Nutraceuticals and Functional Foods*. London, New York, Washington D.C.; 16 (2000).