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# Protective Effects of Dietary Ginger (*Zingiber officinales* Rosc.) on Lindane-induced Oxidative Stress in Rats

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**The protective effect of dietary feeding of *Zingiber officinales* Rosc. (ginger) against lindane-induced oxidative stress was investigated in male albino rats. Oxidative stress was monitored by estimating the extent of lipid peroxidation, activities of the oxygen free radical (OFR) scavenging enzymes superoxide dismutase (SOD) and catalase (CAT) and the status of the glutathione redox cycle antioxidants. Lindane administration (30 mg/kg bw orally for 4 weeks) was associated with enhanced lipid peroxidation and compromised antioxidant defenses in rats fed a normal diet. Concomitant dietary feeding of ginger (1%w/w) significantly attenuated lindane-induced lipid peroxidation, accompanied by modulation of OFR scavenging enzymes as well as reduced glutathione (GSH) and the GSH dependent enzymes glutathione peroxidase (Gpx), glutathione reductase (GR) and glutathione-S-transferase (GST) in these rats. These findings suggest that a diet containing naturally occurring compounds is effective in exerting protective effects by modulating oxidative stress.**


*Keywords:* ginger; antioxidant; lipid peroxidation; lindane; protective effect.

## INTRODUCTION

Lindane, the gamma isomer of hexachlorocyclohexane (HCH) is one of the oldest synthetic pesticides still in use worldwide. HCH has been extensively used to control malaria, but is more commonly used to eradicate insects in agriculture and to treat lice infestation in humans, poultry and livestock (Videla *et al.*, 1990; Seth *et al.*, 2000; Walsh and Stocco, 2000). Because of its widespread use HCH has become widely distributed in ecosystems and is now a global pollutant. Several studies have revealed the presence of lindane above permissible limits in body fat, blood, milk and food commodities both in India and abroad (Banerjee *et al.*, 1997; Samanta *et al.*, 1999).

An increasing number of reports suggest that oxidative stress plays a crucial role in toxicity of xenobiotics like pesticides (El Sharkawy *et al.*, 1994; Banerjee *et al.*, 2001). The alterations of pro- and anti-oxidant status by lindane have been of increasing concern (Almeida *et al.*, 1997; Banerjee *et al.*, 1999). Induction of oxidative stress in rats and immunological alterations by this pesticide in human poisoning cases has been reported recently (Seth *et al.*, 2000, 2005).

Nowadays, there is considerable emphasis on identifying the potential of natural plant products as chemotherapeutic agents present in food consumed by human populations (Lampe, 2003). Spices and herbs are recognized as sources of natural antioxidants that can protect from oxidative stress and thus play an important role in chemoprevention of diseases that have their etiology and pathophysiology in reactive oxygen species (Atawodi, 2005). Ginger (rhizome of *Zingiber officinales* Rosc.) is used worldwide as a cooking spice, condiment and herbal remedy for treatment of various diseases (Capasso *et al.*, 2003). Ginger is also extensively consumed as a flavouring agent, it is estimated that in India, the average daily consumption is 8–10 g of fresh ginger root (Murray, 1995).

It was shown previously that long term dietary feeding of ginger has hypoglycemic and hypolipidemic effects in rats and the antioxidant effect of this dietary constituent is as effective as ascorbic acid (Ahmed and Sharma, 1997; Ahmed *et al.*, 2000). Hypolipidemic and antiatherosclerotic effects of ginger extract were also demonstrated in cholesterol fed rabbits (Sharma *et al.*, 1996). Plants that contain most antioxidants include members of several families such as Rosaceae (dog rose), Zingiberaceae (ginger) etc. (Halvorsen *et al.*, 2002). The superoxide scavenging and tyrosinase inhibitory activity of ginger is well documented (Khanom *et al.*, 2003; Masuda *et al.*, 2004). Due to the extensive and often indiscriminate use of pesticides in the environment and the potential hazards involved therein, studying the role of various indigenous plant products on attenuation of pesticide-induced oxidative stress is not only very interesting but also of practical importance. Given its long history of use as a food, ginger is presumed safe for supplemental use. Hence, the present study was designed to evaluate the effect of ginger on the modulation of oxidative stress induced by subchronic lindane exposure in experimental animals.

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## MATERIALS AND METHODS

**Chemicals.** NADPH, oxidized and reduced glutathione; 1,chloro,2,4,dinitrobenzene (CDNB), glutathione reductase, bovine serum albumin (Fraction V) and lindane (99% purity) were obtained from Sigma Aldrich Company (St Louis, MO, USA). Pyrogallol and 2-thiobarbituric acid (TBA) were obtained from E. Merck, Mumbai, India. All other reagents used were of analytical grade and obtained either from BDH or Sisco Chemicals, Mumbai, India.

**Animals, diet and treatment.** Male albino rats (Wistar strain) weighing 200–250 g were placed in individual raised-bottom, galvanized wire cages and kept under standard laboratory conditions of light–dark cycle (12–12 h) and temperature (25 ± 2 °C). They were provided with a nutritionally adequate standard laboratory diet (Gold Mohur rat feed containing 21% crude protein, 5% ether extract, 4% crude fiber, 8% ash, 1% calcium, 0.6% phosphorus, 53% nitrogen free extract, enriched with vitamins and minerals; total metabolizable energy 3600 kcal/kg) obtained from Hindustan Lever Ltd, Mumbai, India.

The experimental diet (1% ginger) was prepared as follows. Fresh ginger was purchased from the local market, peeled, washed, coarsely minced, air dried and pulverized with a blender to fine powder. This was added w/w to already pulverized feed and thoroughly mixed so as to give a diet containing 1% ginger. Lindane was dissolved in groundnut oil of pharmaceutical grade and administered by gavage, once daily. Control rats received the same volume of vehicle in identical manner. The animals were observed daily for symptoms of intoxication (e.g. salivation, shivering, muscle tonus, food intake etc.) Body weight was recorded weekly.

The rats were randomly divided into four groups of 10 animals each and treated for 4 weeks as follows: Group I, Control: rats fed on normal diet; Group II, Ginger; rats fed on 1% ginger diet; Group III, Lindane: rats received lindane (30 mg/kg, b.w., orally) along with normal diet. Group IV, Lindane + ginger: rats received lindane (30 mg/kg, b.w. orally) along with 1% ginger diet.

All rats were given free access to respective diets and water. Food consumption, general condition and any other symptoms were observed daily and body weight was recorded weekly.

**Samples.** After overnight fasting, animals were killed by decapitation and heparinized blood samples were collected and processed for isolation of erythrocytes. Whole blood samples were also collected and sera separated for various biochemical investigations. Hb concentration was determined spectrophotometrically at 540 nm using Drabkin's reagent. Erythrocytes were isolated and haemolysed. Protein content of haemolysate was estimated by the method of Lowry *et al.* (1951). Red cells were stored at 4 °C and all serum samples at –20 °C.

**Lipid peroxidation.** The lipid peroxidation levels in serum was measured as thiobarbituric acid reactive substances (TBARS) following the method described by Satoh (1978).

**Antioxidant enzymes.** Superoxide dismutase (SOD, E.C. 1.15.1.1) activity in erythrocytes was determined as described by Nandi and Chatterjee (1988). The unit of activity is defined as the amount of enzyme that inhibits the rate of autooxidation of pyrogallol by 50% under standard conditions and was expressed as U/g Hb. Catalase (CAT, E.C. 1.11.1.6) activity in Tsuchihasi extract of red cell hemolysate was determined according to the method of Sinha (1972).

**Glutathione and related enzymes.** Total glutathione (GSH) content in blood was measured by the method of Tietze (1969) using di-thio nitro benzene and expressed as μmol/mL. Glutathione reductase (GR, E.C. 1.6.4.2) activity in serum was determined by following the oxidation of NADPH to NADP during the reduction of oxidized glutathione (Goldberg and Spooner, 1983) and expressed as μmol of NADPH oxidized/min/mL. Total activity of glutathione peroxidase (GPx, E.C. 1.11.1.9) in red cell hemolysate was determined (Paglia and Valentine, 1967). Serum glutathione-S-transferase (GST, E.C. 2.5.1.18) was measured spectrophotometrically by the method of Habig *et al.* (1974) using 1,chloro 2,4,dinitrobenzene as substrate.

**Ethical approval.** The experimental protocols were conducted in accordance with internationally accepted principles for laboratory animal use and care. The study was ethically approved from the Institutional Ethical Committee.

**Statistical analysis.** Data were analysed by one-way ANOVA using SPSS version 5, statistical program and the individual comparisons of treatment/diet were obtained by using Tukey's multiple comparison procedure at $p < 0.05$.

## RESULTS

Exposure of rats to lindane did not produce any overt sign of toxicity/mortality. No significant difference was observed in body growth rates or food intake between control and treated rats.

Lindane treatment enhanced lipid peroxidation in rats fed normal diet. However, levels of TBARS were significantly lower in rats fed the ginger supplemented diet compared with controls or the lindane treated group (Table 1). The SOD activity remained unchanged in rats fed the ginger diet, whereas lindane caused a nearly two-fold increase in activity (Table 1). However, dietary ginger caused a significant decrease ($p < 0.001$) in SOD activity in lindane treated rats, although it was still significantly higher than the control. Similarly, lindane treatment significantly enhanced the erythrocyte CAT activity in animals fed the basal diet. The treatment with dietary ginger significantly prevented the increase in CAT activity (Table 1).

Lindane treatment considerably reduced the GSH levels in rats fed the normal diet. However dietary ginger caused a significant increase in the control as well as in the lindane treated animals (Table 2). Lindane enhanced both the GPx and GR activity in rats fed the normal diet. Concomitant feeding of ginger restored the activities to near normal. Ginger alone did not cause



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**Table 1. Effect of dietary ginger on serum TBARS levels and activities of SOD and CAT in erythrocytes of rats treated with lindane**

| Treatment group | MDA (nmol/mL) | SOD (U/g Hb) | CAT (U/g Hb) |
|---|---|---|---|
| Control | 2.20 ± 0.34 | 620.3 ± 62.9 | 2.34 ± 0.44 |
| Ginger | 1.84 ± 0.32[a] | 630.6 ± 388 | 1.93 ± 0.5[a] |
| Lindane | 3.16 ± 0.22[ab] | 1223.1 ± 64.1[ab] | 4.06 ± 0.41[ab] |
| Lindane + Ginger | 1.98 ± 0.48[c] | 873.1 ± 42.2[abc] | 2.79 ± 0.61[bc] |

Values are expressed as mean ± SD, $n = 10$ animals in each group. Significantly different from [a] Control, [b] Ginger, [c] Lindane ($p < 0.001$).

**Table 2. Effect of dietary ginger on blood glutathione (GSH) content, erythrocyte glutathione peroxidase (GPx) and serum glutathione reductase (GR) and glutathione-S-transferase (GST) activities in rats treated with lindane**

| Treatment group | GSH (μmol/mL) | GPx (U/g Hb) | GR (U/mL) | GST (nmol/mg protein) |
|---|---|---|---|---|
| Control | 212.2 ± 7.00 | 5.94 ± 0.45 | 1.07 ± 0.23 | 0.98 ± 0.37 |
| Ginger | 248.0 ± 11.47[a] | 5.80 ± 0.34 | 0.81 ± 0.09 | 0.85 ± 0.20 |
| Lindane | 126.3 ± 5.28[ab] | 13.26 ± 0.77[a] | 3.55 ± 0.49[ab] | 1.69 ± 0.14[ab] |
| Lindane+ Ginger | 232.0 ± 9.26[c] | 5.42 ± 0.84[c] | 1.13 ± 0.11[c] | 1.28 ± 0.22[bc] |

Values are expressed as mean ± SD, $n = 10$ animals in each group. Significantly different from [a] Control, [b] Ginger, [c] Lindane ($p < 0.001$).

any significant alteration in these enzymes, as well as GST (Table 2). The increase in GST activities due to lindane treatment was significantly reduced when the diet was supplemented with ginger.

## DISCUSSION

An imbalance of reactive oxygen species (ROS) and antioxidant defence system may lead to chemical modification of biologically relevant macromolecules and it provides a logical pathobiochemical mechanism for initiation and development of several disease states (Bandopadhyay *et al.*, 1999; Banerjee *et al.*, 2001). Therapy using free radical scavengers or antioxidants has the potential to prevent, delay or ameliorate many of these disorders (Halliwell, 1999). At present there is considerable interest in free radical mediated damage in biological systems due to pesticide exposure (Banerjee *et al.*, 1999, 2001) and the search for herbal drugs with antioxidant activity has gained importance as the dietary intake of antioxidants obtained from natural sources is considered to be relatively safe and without undesirable side effects (Xavier *et al.*, 2004).

The large scale application of lindane over a number of years, coupled with its continued use and slow metabolism has led to environmental contamination and potential health hazards. The effect of lindane has been related to its highly lipophilic nature, which allows the accumulation and interaction of this insecticide with membrane lipids. Lindane intoxication was found to induce oxidative stress in the liver of rodents, whose temporal development and magnitude correlated with the observed hepatotoxic lesions (Videla *et al.*, 1990). Oxidative damage and changes in glutathione redox system in erythrocytes from rats treated with lindane has also been reported (Seth *et al.*, 2000).

Over the past two decades, an expanding body of evidence from epidemiological and laboratory studies has demonstrated that some edible plants as a whole or their identified ingredients with antioxidant properties have substantial protective effects on a variety of human disease states (Halvorsen, 2002; Atawodi, 2005). Spices and herbs are recognized as sources of natural antioxidants that can protect from oxidative stress and thus play an important role in the chemoprevention of diseases (Surh, 2002). Ginger has been used extensively in folklore medicine to treat common ailments and new scientific evidence in favour of some of these beneficial properties is emerging which supports its consumption and use to ameliorate different human disorders (Vishwakarma *et al.*, 2002; Khanom *et al.*, 2003). In rats fed with high fat diet, supplementation with ginger provided significant antioxidant effects, raising tissue concentrations of superoxide dismutase, catalase and reduced glutathione (Jeyakumar *et al.*, 1999). However, studies on the protective effect of plant products on the attenuation of pesticide induced toxic effects is scarce and to our knowledge there is no information concerning the protective effect of dietary ginger on oxidative stress induced by lindane. Study of the protective effect of ginger on free radical mediated toxicity induced by organochlorine pesticides such as lindane therefore appeared to be of interest.

In the present study the elevated level of TBARS observed in lindane treated rats indicates excessive formation of free radicals and activation of lipid peroxidation system. The significant decline in the serum TBARS level of rats fed on lindane along with ginger indicates the antilipid peroxidative effect of this plant product.

SOD, CAT and GPx constitutes a mutually supportive team of defence against ROS (Bandopadhyay *et al.*, 1999). SOD is a metalloprotein and is the first enzyme involved in the antioxidant defence by lowering the steady state level of $O_2^{\cdot-}$. CAT is a hemoprotein that



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catalyses the decomposition of $H_2O_2$ to water and oxygen and thus protects the cell from oxidative damage by $H_2O_2$ and $OH^-$. GPx is a seleno enzyme that catalyses the reaction of hydroperoxides with reduced glutathione to form glutathione disulphide (GSSG) and reaction product of hydroperoxide. GST plays an essential role in eliminating toxic compounds by conjugating them with glutathione. GR is concerned with the maintenance of the cellular level of GSH in the reduced state. Administration of lindane at 30 mg/kg, b.w. for 4 weeks was found to elicit a significant alteration of some antioxidant mechanisms in erythrocytes; the activities of SOD, CAT, GPx, GR and GST were increased with lindane treatment, whereas the GSH level in blood was significantly decreased.

No report was found of ginger (or any other spice), interfering in the absorption of pesticides from the gut. The vehicle used for the treatment of lindane was groundnut oil. Absorption of lindane across the skin as well as in the gut is enhanced by the presence of fat and fat solvents (Reigart and Roberts, 1999). Moreover, the fat/oil content in ginger is 1–2%. Hence, it can be safely assumed that ginger does not interfere in the absorption of lindane from the gut. Moreover, although dietary ginger caused a significant decrease in SOD activity in lindane treated rats, it was still significantly higher than the control (Table 1). Increased SOD activity in the lindane + ginger group compared with the control/ginger alone shows the presence of oxidative stress which can be attributed to the presence of lindane in the blood, although blood lindane levels were not measured.

These findings indicate that the increase in free radical scavenging enzymes followed by a decrease in GSH by lindane could be initiated by oxidative stress and/or associated with the initial formation of specific lindane derived reactive metabolites. The reduction of GSH in blood after lindane exposure may have resulted from activity of GPx in reducing lipid hydroperoxides to stable non-radical lipid alcohols utilizing GSH as a source of reducing equivalents. Alternatively, GSH concentration may have been reduced by the direct utilization of GSH as an antioxidant in terminating free radical reaction initiated by lindane. Ginger supplementation in the diet, however, provokes a sustained higher level of GSH while nullifying the increase in the level of antioxidant enzymes.

Although a number of antioxidant compounds have been isolated from ginger (Masuda *et al.*, 2004) the exact mechanism by which ginger exerts antioxidant effect is not yet clear. Two new curcuminoids isolated from tropical ginger (casumunin A and B) have been shown to protect rat thymocytes suffering from oxidative stress in an *in vitro* study (Nagano *et al.*, 1997).

Natural antioxidants strengthen the endogenous antioxidant defence mechanism and restore the optimal balance by neutralizing the reactive species and thus the search for crude drugs of plant origin with antioxidant activity has become a central focus of study in recent years. Such studies on the oxidative/antioxidant status during a free radical challenge can be used as an index of protection against the development of lipid peroxidation in experimental animals for assessing dietary and therapeutic measures. Curcumin and zingerone – compounds abundant in ginger rhizomes are known to have antioxidant activity (Wang *et al.*, 2003). Hence, ginger may attenuate free radical mediated toxic effects by lowering lipid peroxidation and maintaining the activities of antioxidants and this may be attributed to curcumin and zingerone components present in ginger.

In conclusion, the present study demonstrates that ginger exerts significant protective effects against lindane-induced oxidative stress by augmenting host antioxidant defense mechanisms. Thus, ginger is a promising agent for the prevention of pesticide induced toxicity through induction of antioxidative and phase II drug metabolizing enzymes as well as lowering lipid peroxidation.

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