Indian Journal of Biochemistry & Biophysics
Vol. 36, February 1999, pp. 59-61

Coriandrum sativum changes the levels of lipid peroxides and activity of antioxidant enzymes in experimental animals

V Chithra and S Leelamma*

Department of Biochemistry, University of Kerala, Kariavattom, Thiruvananthapuram 695 581

Received 17 February 1998; revised 21 July 1998

The antiperoxidative effect of coriander seeds (*Coriandrum sativum*) was studied in rats administered high fat diet. Significant decrease in the levels of lipid peroxides, free fatty acids and glutathione was observed when compared to control group whereas the activity of antioxidant enzymes showed increase.

Lipid peroxidation is a complex process which is initiated by abstraction of hydrogen atoms from unsaturated fatty acids of phospholipid and lipoprotein complexes yielding conjugated dienes. The conjugated dienes then react with molecular oxygen to produce peroxy radicals which propagate the chain reaction by abstracting hydrogen from other unsaturated lipids[1,2]. Some antioxidant defenses are located both in extracellular and intracellular compartments. Because our endogenous antioxidant defenses are not completely effective, dietary antioxidants are of particular importance in diminishing the cumulative effects of oxidative damage[3]. Spices have a key role as chemopreventers in human diet[4]. Effect of spice principles on scavenging of superoxide anion had been earlier investigated[5]. Turmerin, a water soluble antioxidant peptide from turmeric was studied for its protective effect against reactive oxygen species induced lipid peroxide-mediated membrane and DNA damage[6]. Breakdown of lipid hydroperoxides and endoperoxides lead to the formation of more than twenty known products of lipid peroxidation. The estimation of the end product assayed as thiobarbituric acid reactive substances (TBARS) is a measure of lipid peroxidation[7]. Free radical intermediates have been linked to clinically important disturbances like ischemic heart diseases and atherogenesis[8,9]. Aruna *et al.*[10] have reported the effect of cumin, turmeric, ajwain, pepper, aniseed, curry leaves, coriander seeds, fenugreek seeds and ginger as cancer protective agents. But no detailed investigations were carried out on the antioxidant effect of the spices. Since coriander forms one of the commonly used spices, we have studied the role of coriander seeds on lipid peroxidation and antioxidant defense mechanisms in rats given high fat cholesterol diet.

Female albino rats of Sprague-Dawley strain of age one month and weighing 70-80 g were divided into two groups of 6 rats each. The animals were procured from NIN, Hyderabad and they were maintained under good laboratory conditions. Group I, the controls, received the following diet: coconut oil (15%) and cholesterol (2%) were mixed with powdered normal laboratory feed (supplied by M/s Hindustan Lever Ltd.). Group II, the experimental group received the above diet mixed with powdered coriander seeds (10%). Water was supplied *ad libitum*. The rats were given the diet 12g /100g body wt and maintained on the respective diet for a period of 90 days. At the end of this period the rats were starved overnight and killed by decapitation. The tissues were removed to ice cold containers for various estimations.

The following procedures were used for the estimation of superoxide dismutase (SOD, EC 1.15.1.1)[11], catalase (EC 1.11.1.6)[12], glutathione reductase (GR, EC 1.6.4.2)[13], glutathione peroxidase (GPX, EC 1.11.1.9)[14], glutathione-S-transferase (GST, EC 2.5.1.18)[15], glucose-6-phosphate dehydrogenase (G-6-PDH, EC 1.1.1.49)[16], free fatty acids (FFA)[17], malondialdehyde (MDA)[18], hydroperoxides (HP)[19], conjugated dienes (CD)[20], glutathione (GSH)[21]. Protein was estimated in enzyme extract as described[22]. Statistical analysis was done using student's 't' test[23].

The animals in the two groups showed almost similar weight gain ie. 85±2.6 g. The diet consumption was also similar in both groups ie, 11.5±1.5 g/100g body wt. The results show that feeding of coriander seeds brings about alterations in the level of lipid peroxides in different tissues. Concentration of MDA decreased significantly in the liver and heart of coriander administered group. The concentration of hydroperoxides and conjugated dienes showed significant decrease in the experi-

*Author for correspondence

Table 1 — Effect of coriander seeds on the levels of peroxidation products, glutathione and the activities of antioxidant enzymes

[Values are mean ± SE of 6 rats. Group I has been compared with group II]

Parameters	Liver		Heart	
	Control	Experimental diet	Control	Experimental diet
MDA (m*M*/100g)	0.89 ± 0.02	0.42 ± 0.01[a]	0.68 ± 0.02	0.46 ± 0.01[a]
Hydroperoxides (m*M*/100g)	18.30 ± 0.49	10.50 ± 0.28[a]	49.40 ± 1.48	18.70 ± 0.56[a]
Conjugated dienes (m*M*/100g)	83.50 ± 2.34	67.20 ± 1.88[a]	18.40 ± 0.42	14.70 ± 0.34[a]
Glutathione (m*M*/100g)	485.30 ± 13.10	394.8 ± 10.65	469 ± 12.66	386.87 ± 10.45[a]
Superoxide dismutase (units*/mg protein)	11.38 ± 0.29	20.41 ± 0.53[a]	17.46 ± 0.51	32.61 ± 0.95[a]
Catalase ($\times 10^{-3}$ units^{+}/mg protein)	78.56 ± 2.2	163.35 ± 4.57[a]	22.83 ± 0.64	113.23 ± 3.17[a]
Glutathione peroxidase (g to reduced glutathione utilized/min/mg protein)	6.79 ± 0.18	16.57 ± 0.45[a]	11.30 ± 0.32	20.78 ± 0.59[a]
Glutathione-S-transferase (mmole of thioester/ min/ mg protein)	3.30 ± 0.09	5.80± 0.16[a]	4.10 ± 0.11	6.3 ± 0.17[a]
Glucose-6-phosphate dehydrogenase (mmole of $NADP^{+}$ reduced min/mg protein)	72.50 ± 2.03	110.60±3.1[a]	—	—
Glutathione reductase (mmole of thioester/min/ mg protein)	15.50 ± 0.37	22.52 ± 0.68[a]	14.80 ± 0.43	18.3 ± 0.45[a]

Unit*, Enzyme concentration required to inhibit OD at 560 nm of chromogen production by 50% in 1 min.
Unit^{+}, Velocity constant/sec.
[a] $p < 0.01$.

Table 2 — Concentration of free fatty acids in serum (mg/100 ml) liver and heart (mg/100 g).

[Values are mean ± SE of 6 rats. Group I has been compared with group II]

	Serum	Liver	Heart
Control	120.50 ± 3.37	799.30 ± 23.97	592 ± 17.16
Experimental diet	79.80 ± 2.23[a]	488 ± 14.64[a]	320 ± 9.28[a]

[a] $p < 0.01$.

mental group (Table 1). The level of free fatty acids, possibly a substrate for microsomal lipid peroxidation was significantly decreased in the experimental group (Table 2). The observed decrease in lipid peroxides could be due to the reduction of free fatty acids and increased levels of free radical scavenging enzymes, viz., superoxide dismutase (SOD) and catalase in different tissues in animals fed coriander seeds (Table 1).

Higher levels of the antioxidant enzymes have been correlated with decreased susceptibility to cell damage[24]. The decreased level of glutathione in coriander administered group suggests the optimum activity of glutathione peroxidase in liver and heart (Table 1). Activities of glutathione reductase, glutathione-S-transferase and glucose-6-phosphate dehydrogenase also maintain the glutathione level (Table 1). The increase in glucose-6-phosphate dehydrogenase activity leads to an increase in HMP (hexose monophosphate) shunt pathway and thereby increase the level of NADPH. Such an increase in NADPH level could result in the increase of glutathione reductase activity which in turn helps to keep a raised level of glutathione. Activity of glutathione-S-transferase (GST), involved in the detoxification of an extensive array of compounds is significantly increased in liver and heart of animals fed coriander seeds. GST has been reported to possess peroxidase activity and participates in the reduction of fatty acid hydroperoxides to non-toxic alcohols[10]. Thus the supplementation of 10% coriander seeds with high fat cholesterol containing diet protect the

various tissues by preventing the formation of unwanted free radicals.

References

1 Esterbauer H (1982) in *Free radicals lipid peroxidation and cancer* (Slater T F, McBrain D ed), 101-128, Academic Press, New York

2 Mccord J & Fridovich I (1978) *Ann Intern Med* 89, 122-127

3 Barry Halliwell & Gutteridge J M C (1984) *J Biochem* 219, 1-14

4 Starvic B (1997) *Clin Biochem* 27 (5), 319-323

5 Krishnakantha T P & Behur R Lokesh (1993) *Indian J Biochem Biophys* 30, 133-138

6 Srinivas L, Shalini V K & Shylaja M (1992) *Arch Biochem Biophys* 292(2), 23-28

7 Beruheim & Wilbert K M (1984) *J Biol Chem* 248, 7134-7141

8 Fgey (1986) *Bibilotheca Nutrition Dieta* 37, 53-91

9 Boccio G D, Lepenna D, Porecca E, Pinilli A, Savini F, Feliciani P, Ricci G, Cuccurullo F (1990) *Atherosclerosis* 81, 127-135

10 Aruna K & Sivaramakrishnan V M (1990) *Indian J Exp Biol* 28, 1008-1011

11 Kakkar P, Das B & Viswanath P N (1984) *Indian J Biochem Biophys* 21, 130-132

12 Machly A C & Chance B (1954) in *Methods in Biochemical Analysis* 1, 357-424

13 David M & Richard J S (1983) *Methods Enzymat Anal* 3, 258-265

14 Lawrence R A & Burck R E (1976) *Biochem Biophys Res Commun* 17, 952-958

15 Habig W H, Pubst M J & Jakaby W B (1977) *J Biol Chem* 249, 7130-7135

16 Kornberg A & Horecker B L (1955) *Methods Enzymol* 1, 323-328

17 Falhott K, Falhott W & Lund B (1973) *Clin Chem Acta* 46, 105-111

18 Nichans W G Jr & Samuelson B (1968) *Eur J Biochem* 6, 126-130

19 John A B & Steven D A (1978) *Methods Enzymol* 52, 302-305

20 Benge J A & Aust S D (1978) *Methods Enzymol* 52,306-310

21 Patterson J W & Lazarow A (1955) in *Methods in Biochemical Analysis*, (Glick D ed) Vol. 2, pp 259-278, Interscience, New York

22 Lowry O H, Rosebrough N J, Farr A L & Randall R J (1951) *J Biol Chem*, 193, 265-275

23 Bennet C A, Franklin N L (1967) in *Statistical analysis in chemistry and chemical industry*,140-148 (John Willey & Sons)

24 Wertz E & Gould M (1986) *Carcinogenesis* 7, 1197-1200