Indian Journal of Experimental Biology
Vol. 38, June 2000, pp. 604-606

Influence of dietary ginger (*Zingiber officinales* Rosc) on antioxidant defense system in rat: Comparison with ascorbic acid

Rafat S. Ahmed, Vandana Seth & B D Banerjee*

Department of Biochemistry, University College of Medical Sciences and Guru Teg Bahadur Hospital, University of Delhi, Shahdara, Delhi 110 095, India

Received 6 July 1999; revised 1 March 2000

Ginger (*Z. officinale*; 1% w/w) significantly lowered lipid peroxidation by maintaining the activities of the antioxidant enzymes—superoxide dismutase, catalase and glutathione peroxidase in rats. The blood glutathione content was significantly increased in ginger fed rats. Similar effects were also observed after natural antioxidant ascorbic acid (100 mg/kg, body wt) treatment. The results indicate that ginger is comparatively as effective as ascorbic acid as an antioxidant.

Detrimental effects caused by free radicals occur as a consequence of an imbalance between formation and inactivation of these species. Oxidative damage may be involved in the pathogenesis of major diseases such as cancer, atherosclerosis[1] and certain neurological disorders[2]. The relevance of free radicals/reactive oxygen species (ROS) in tissue damage and carcinogenesis has been reported[3]. Inactivation and removal of ROS depend on reactions involving the antioxidative defence system. Hence, it is important to identify natural antioxidative agents present in diets consumed by the human population.

Ginger rhizome (*Zingiber officinale* Rosc) commonly known as ginger, is consumed world wide as a spice and flavoring agent. In India, ginger is used extensively in both vegetarian and non-vegetarian diets. Limited *in vitro* studies have shown that water and organic solvent extracts of ginger possess antioxidant properties[4-8]. Oral consumption of ginger (dried, powdered) has been shown to result in relief of pain and swelling in patients with rheumatoid arthritis, osteoarthritis or muscular discomfort[8,9]. Ginger oil has been found to be an inhibitor of both cyclooxygenase and lipoxygenase activities[10,11]. Recently, Sharma *et. al.*[12] have suggested that ginger oil possesses antiinflammatory properties. A combination of ginger and garlic has been reported to produce hypoglycemic and hypolipidemic effects[13]. In a recent study dietary ginger protected the tissues from oxidative stress induced by organophosphate pesticide (malathion) in rats[14,15]. In view of the above, it is of much interest and practical importance to study influence of long term dietary ginger on antioxidative potential. Hence, effects of dietary ginger have been assessed on (a) lipid peroxidation; (b) antioxidant scavengers-superoxide dismutase (SOD) and catalase (CAT); and (c) glutathione linked enzyme system in blood. Simultaneously, antioxidative properties of ginger in rats were compared with ascorbic acid-a natural antioxidant.

Animals and diet—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 hr) and temperature (25°±2°C). They were provided with a nutritionally adequate standard laboratory diet. (Hindustan Lever Ltd., Mumbai, India) and tap water *ad libitum*. 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 get a diet containing 1% ginger. The rats were randomly divided into 3 groups of 10 animals each and treated for 4 weeks as follows:

Group I, Control: rats fed on normal diet

Group II, Rats fed on 1% ginger diet

Group III, Rats fed on normal diet and treated with ascorbic acid (100 mg/kg/d) orally by gastric tube at 0900 hrs throughout the experimental period.

*Correspondent author
Fax: +91-11-2290495
e.mail: dbmi@ucms.ernet.in

Samples—After overnight fasting, animals were sacrificed 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 haemolyzed. Protein content of haemolysate was estimated as per Lowry *et al*[16].

Biochemical estimations—Lipid peroxidation level in serum was measured as thiobarbituric acid reactive substances (TBARS) according to the method described by Satoh[17]. Superoxide dismutase (SOD, E.C. 1.15.1.1) activity in erythrocytes was determined[18]. Catalase (CAT, E.C. 1.11.1.6) activity was measured in Tsuchihasi extract of red cell haemolysate[19]. Total glutathione (GSH) content in blood was measured by the method of Tietze[20]. Glutathione reductase (GR, E.C. 1.6.4.2) activity in plasma[21] and total activity of glutathione peroxidase (GPx, E.C. 1.11.1.9) in red cell haemolysate were determined[22]. Serum glutathione-S-transferase (GST, E.C. 2.5.1.18) activity was measured spectrophotometrically by the method of Habig *et al*[23] using 1, chloro, 2,4 dinitrobenzene as substrate. The values are expressed as mean±SD. The data were analysed using one way ANOVA followed by Tukey's test for intergroup comparison.

Ascorbic acid is a potent antioxidant which acts directly on oxygen free radicals (OFR) as well as through interactions with vitamin E[24,25]. However, ascorbic acid *per se*, did not have any effect on SOD activity in the present study (Table 1) which is in agreement with earlier reports[26,27]. Similar effects for dietary ginger on SOD were observed (Table 1). Both CAT and GPx activities (Table 1) remained unaltered in ginger fed and ascorbic acid treated animals indicating that production of H_2O_2 is not high enough to warrant any increase in these enzymes and they are maintained at normal levels. The activities of GST and GR did not show any alteration in ginger and ascorbic acid treated groups (Table 1).

Lipid peroxidation plays a crucial role in inflammation, cancer and cardiac diseases[28]. The present results showed that dietary feeding of ginger significantly lowers lipid peroxidation, similar to ascorbic acid. Curcumin, a major component of turmeric can lower lipid peroxidation[29]. Curcumin and zingerone, compounds abundant in ginger rhizomes are known to have antioxidant activity[30,32]. Hence, ginger may lower lipid peroxidation by maintaining the activities of antioxidant enzymes and these effects may be attributed to the curcumin and zingerone components present in ginger.

Glutathione is the first line of defense against prooxidant stress and GSH was elevated after ginger feeding. GSH system may have the ability to manage oxidative stress with adaptational changes in enzymes regulating GSH metabolism. However, the mechanism by which ginger provides an environment for the enhanced level of GSH is yet to be ascertained. The results of the present study suggest that ginger exerts antioxidative effect by decreasing lipid peroxidation, increasing GSH content and maintaining normal levels of antioxidant enzymes (SOD, CAT, GPx and GR). Scavenging of superoxide radicals by some spice principles have been documented[5,33].

Present study in rats given normal diet along with ginger prevented the formation of unwanted free radicals, maintained the integrity of erythrocytes and

Table 1—Effect of ginger or ascorbic acid on various biochemical parameters.

[Values are mean±SD of 10 animals/group]

Parameters	Group I	Group II	Group III
Serum TBARS (n.mol/ml)	3.11±0.77	2.20±0.50*	1.97±0.33*
SOD (U/gm Hb)	620.00±22.56	640.60±28.90	643.0±37.00
CAT (U/gm Hb)	2.51±0.49	2.40±0.37	2.38±0.17
Blood GSH (μ mol/ml)	218.30±46.2	250.50±21.40*	252.3±17.00*
Serum GR (U/ml)	1.20±0.26	1.01±0.09	1.12±0.17
Serum GST (n.mol/mg protein)	0.98±0.37	0.85±0.22	0.86±0.17
RBC GPx (U/gm Hb)	5.94±0.45	5.40±0.34	5.57±0.24

*$P < 0.001$

the effects were similar to that of ascorbic acid, a known natural antioxidant. Hence ginger may be taken regularly in the normal diet for therapeutic purposes. Furthermore, similar studies on antioxidant status during a free radical challenge can be used as an index of protection against the development of lipid peroxidation in experimental animals[34,35].

References

1 Knight J A, Diseases related to oxygen-derived free radicals, *Ann Clin Lab Sci*, 25 (1995) 111.

2 Jenner P, Oxidative damage in neurodegenerative disease, *Lancet*, 344 (1994) 796.

3 Oberly L W & Beuttner G R, Role of superoxide dismutase in cancer, *Cancer research*, 39 (1979) 1141.

4 Kiuchi F, Iwakami S, Shibuye M, Hanaoka F & Sankawa U, Inhibition of prostaglandin and leukotriene biosynthesis by gingerols and diarylpeptanoids, *Chem & Pharm Bull* (Tokyo), 40 (1992) 387.

5 Krishnakantha T P & Lokesh B R, Scavenging of superoxide anions by spice principles, *Indian J Biochem Biophys*, 30 (1990) 133.

6 Reddy A C & Lokesh B R, Studies on spice principles as antioxidants in the inhibition of lipid peroxidation in rat liver microsomes, *Mol Cell Biochem*, 111 (1992) 117.

7 Jitoe A, Masuda T, Tengah I G P, Suprapta D N, Gara I W & Nakatani N, Antioxidant activity of tropical ginger extracts and analysis of the contained curcuminoids, *J Agric Food Chem*, 40 (1992) 1337.

8 Srivastava K C & Mustafa T, Ginger (*Zingiber officinale*) and rheumatic disorders, *Med Hypotheses*, 29 (1989) 25.

9 Srivastava K C & Mustafa T, Ginger (*Zingiber officinale*) in rheumatism and musculoskeletal disorders, *Med Hypotheses*, 39 (1992) 342.

10 Kiuchi F, Shibuya M & Sankawa U, Inhibition of prostaglandin biosynthesis from ginger, *Chem & Pharm Bull* (Tokyo), 30 (1982) 747.

11 Flynn D L, Rafferty M F & Boctor A M, Inhibition of human neutrophil 5-lipoxygenasse activity by gingerdione, shogao, capsaicin and related pungent compounds, *Prostaglandins Leukot Med*, 24 (1986) 195.

12 Sharma J N, Srivastava K C & Gan E K, Suppressive effects of eugenol and ginger oil on arthritic rats, *Pharmacology* (Basel), 49 (1994) 314.

13 Ahmed R S & Sharma S B, Biochemical studies on combined effects on garlic (*Allium sativum* Linn) and ginger (*Zingiber officinales* Rosc) in albino rats, *Indian J Exp Biol*, 35 (1997) 841.

14 Banerjee B D, The influence of various factors on immune toxicity assessment of pesticide chemicals, *Toxicol Letters* 107 (1999) 21.

15 Seth V, Ahmed R S, Pasha S T & Banerjee B D, Malathion induced oxidative stress in rats: modulation by *Zingiber officinales* Rosc (ginger), Proceedings 67th Annual Meeting. Society of Biological Chemists, N. Delhi, India (1999).

16 Lowry O H, Rosebrough N J, Farr A L & Randall R J, Protein measurement with the Folin phenol reagent, *J Biol Chem*, 193 (1951) 265.

17 Satoh K, Serum lipid peroxide in cerebrospinal disorder determined by a new colorimetric method, *Clinical Chemica Acta*, 90 (1978) 37.

18 Nandi A & Chatterjee J B, Assay of superoxide dismutase activity in animal tissue, *J Biosciences*, 13 (1988) 305.

19 Sinha A K, Colorimetric assay of catalase, *Anal Biochem*, 47 (1972) 389.

20 Tietze F, Enzymatic method for quantitative determination of nanogram amounts of total and oxidized glutathione: application to mammalian blood and other tissues, *Anal Biochem*, 27 (1969) 502.

21 Goldberg D M & Spooner R J, Glutathione reductase, in *Methods in Enzymology*, edited by H U Bergmeyer (Verlag Chemie, Basel) 1983, 258

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

23 Habig W H, Pabst M J & Jacoby W B, Glutathione-S-transferase: the first step in mercapturic acid formation, *J Biol Chem*, 249 (1974) 7130.

24 Sharma M K & Buettner G R, Interaction of Vit C and Vit E during free radical stress in plasma: an ESR study, *Biochemistry*, 32 (1993) 10692.

25 Johnston C S, Luo B & Monte W C, Vitamin C elevates red blood cell glutathione in healthy adults, *Am J Clin Nutr* 586 (1993) 103.

26 Koner B C, Banerjee B D & Ray A, Organochlorine pesticide-induced oxidative stress and immune suppression in rats, *Indian J Exp Biol*, 36 (1998) 395.

27 Beisel W R, Single nutrients and immunity, *Am J Clin Nutr*, 35 (1982) 417.

28 Freeman B A & Crapo J D, Biology of disease: Free radicals and tissue injury, *Lab Investigation*, 17 (1982) 112.

29 Reddy AC & Lokesh B R, Effect of dietary turmeric (*Curcuma longa*) on iron-induced lipid peroxidation in the rat liver, *Food Chem Toxicol*, 32 (1994) 279.

30 Sarma O P, Antioxidant activity of curcumin and related compounds, *Biochem Pharmacol*, 25 (1976) 1811.

31 Toda S, Miyake T, Arichi H, Tanizawa H & Takino Y, Natural antioxidants III, antioxidative components isolated from rhizomes of *Curcuma longa* L., *Chem Pharma Bull* (Tokyo), 33 (1985) 1725.

32 Aeschbach R, Loliger J, Scott B C, Murcia A, Butler J, Halliwell B & Aruoma OI, antioxidant actions of thymol, carvacrol, 6-gingerol, zingerone and hydroxytyrosol, *Food Chem Toxicol*, 32(1994) 31.

33 Kunchandy E & Rao M N A, Oxygen radical scavenging activity of curcumin, *Int J Pharm*, 58 (1990) 237.

34 Banerjee B D, Seth V, Bhattacharya A & Chakraborty A K, Oxidative stress in human poisoning cases following malathion and propoxur ingestion, *Toxicol Letters* 95 (Suppl. 1) (1998) 58.

35 Banerjee B D, Seth V, Bhatacharya A, Pasha S T & Chakraborty A K, Biochemical effects of some pesticides on the lipid peroxidation and free radical scavengers, *Toxicol Letters*, 107 (1999) 33.