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Full Length Research Paper

Hepatoprotective and antioxidant properties of extract of *Carmellia sinensis* (black tea) in rats

Ojo Opeolu Oyejide[1] and Ladeji Olushola[2]

[1]Department of Integrated Science, Federal College of Education, P.M.B. 2042, Yola, Nigeria.
[2]Department of Biochemistry, University of Jos, Nigeria.

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Hepatoprotective and antioxidant effects of water extracts of black tea (*Camellia sinensis*) were studied in sodium oxalate treated rats. Lipid peroxidation was induced in rats by administration of 100 mg/kg body weight sodium oxalate. The protective effect of black tea was assessed by monitoring the serum and tissue levels of malondialdehyde, catalase activity, aspartate transaminase (AST) and alanine transaminase (ALT) as well as serum vitamin C content in the normal, control and experimental rats after 10 and 20 days of tea administration. It was observed that tea administration lowers significantly ($p<0.05$) the serum and tissue levels of malondialdehyde, as well as AST and ALT activities in a dose dependent manner. The serum level of vitamin C and activity of catalase in the serum and tissues were however shown to be significantly elevated ($p<0.05$). After 10 days of administration of 200 mg/kg body weight of tea extract, serum level of malondialdehyde was reduced from 47.855±1.050 to 32.186±0.882 nm/h, AST activity from 59±2.95 to 31±1.40 IU and ALT activity from 39±2.51 to 25±1.25 IU. Moreover, administration of 200 mg/Kg body weight of tea for 10 days caused an increase in serum catalase activity from 7 to 10% and serum vitamin C level was increased from 45.39±9.75 to 79.11±5.13 mg/100 ml. In the tissues, the same trend was observed. The result also indicated that prolonged tea administration (for 20 days) significantly increased serum vitamin C level and the activity of catalase in both the serum, liver and the kidney ($p<0.05$). Also, the serum and tissue levels of malondialdehyde and transaminase activities (AST and ALT) were significantly reduced ($p<0.05$).

Key words: Hepatoprotective effects, lipid peroxidation, antioxidant effects, *Carmellia sinensis*, transaminases, sodium oxalate.

INTRODUCTION

The term tea (*Camellia sinensis*) is used for a family of mostly woody flowering plants belonging to the family Theaceae. The family contains about 520 species and are placed in 28 genera. The family is distributed through tropical and sub-tropical areas, but most species occur in eastern Asia and South America. Tea generally is an aromatic stimulant containing in addition to polyphenols and caffeine, essential oils, alkaloids and theobromine.

Antioxidants are compounds that help prevent free-radical damage. These protective compounds are common in foods and they include vitamins A, C, E and Coenzyme Q10. Also carotenes such as beta-carotene, which is high in carrots, and the trace mineral, selenium, are well known antioxidants. Extracts of *C. sinensis* have been implicated to have potent antioxidant properties and have become a popular natural medicine. Moreover, tea drinking is a popular act throughout the world. As an herbal remedy, green tea is often recommended to ease stomach discomfort, vomiting and to stop diarrhoea. The antibacterial action of tea is useful in treating infections and wounds.

Recent clinical studies have also indicated that regular use of green tea may reduce the risk of certain types of

*Corresponding author. E-mail: cecejyde@yahoo.com.

cancer, including oral, skin, prostate, colon, stomach, and rectal (Maity et al., 1995). Patients with pre-cancerous mouth lesions who were treated with green and black tea extracts achieved a 38% decrease in the number of pre-cancerous cells.

To further investigate the antioxidant effects of tea consumption, this paper reports the result of tea consumption in rats challenged with sodium oxalate. Sodium oxalate is well known to induce lipid peroxidation *in vivo* (Selvam and Kurien, 1987). We also report the result of preliminary investigations of the hepatoprotective effects of tea consumption in rats.

MATERIALS AND METHODS

Preparation of tea sample

Processed black tea was purchased from Jimeta market. A stock tea solution was prepared by extracting 50 g of black tea in 1000 ml of distilled water. The stock tea extract was diluted to give the desired concentration to be fed into different groups of rats. Administration of tea was through the oral route.

Experimental animals

Male albino rats of Winstar strain of average weight of 130g were used for this study. They were purchased from the animal house of the University of Jos. Experimental rats were allowed to stabilize for one week before the commencement of the experiment. They were kept in cages in a room maintained at room temperature and were allowed feed and water *ad libitum*.

Chemicals

Assay kits for the estimation of serum alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP) were purchased from Randox, UK. Sodium oxalate was purchased from Sigma Chemical Co., USA. All other chemicals were of analytical grade.

Treatment of animals

Rats were divided randomly into five groups with 10 rats in each group. Rats in Group A served as control normal and were given distilled water orally. Rats in Group B, the control experimental group, were given 100 mg/kg body weight of sodium oxalate intraperitoneally and sacrificed 12 h later. Rats in Group C were fed with 50 mg/kg body weight of tea extract daily. After the first 10 days, half of the rats in the group were given 100 mg/kg body weight of sodium oxalate and sacrificed 12 h later. The remaining half was fed with the same concentration of tea for a total period of 20 days before intraperitoneal administration of sodium oxalate and sacrificed 12 h later. Rats in Group D were fed with 100 mg/kg body weight of tea extract daily. After the first 10 days, half of the rats in the group were given 100 mg/kg body weight of sodium oxalate and sacrificed 12 h later. The remaining half was fed with the same concentration of tea for a total period of 20 days before intraperitoneal administration of sodium oxalate and sacrificed 12 h later. Rats in Group E were fed with 200 mg/kg body weight of tea extract daily. After the first 10 days, half of the rats in the group were given 100 mg/kg body weight of sodium oxalate and sacrificed 12 h later. The remaining half was fed with the same concentration of tea for a total period of 20 days before intraperitoneal administration of sodium oxalate and sacrifice 12 h later.

Blood samples were drawn from ether anaesthetized mice by cardiac puncture. The liver and kidney of rats were removed immediately after rats were killed. 0.5 g of each tissue was homogenized in 10-fold weight of ice cold 0.15 M KCl solution. The preparation was centrifuged at 1500 rpm for 15 min. The supernatant was decanted and stored frozen until required.

Measurement of biochemical parameters

Assay for malondyaldehyde was done through the method of Janeiro (1998) by measuring thiobarbituric acid reactive substances produced during lipid peroxidation. Determination of transaminases (SGOT, SGPT) was done using the method of Reitman and Frankel (1957). Catalase activity was estimated by the procedure of Sinha (1972). Determination of serum reduced ascorbic acid level was by the method of Urbach et al. (1951).

Statistical analysis

Values are expressed as Mean ± S.E.M. Randomized Complete Block Design Analysis of Variance was used for statistical analysis. *P* Values less than 0.05 were considered significant.

RESULT AND DISCUSSION

Effect of tea consumption on sodium oxalate-induced malondialdehyde formation in rats

The effect of 10 and 20 days consumption of tea extract on the level of malondialdehyde in sodium oxalate treated rats is presented in Tables 1a and b. After 10 days consumption of tea, control rats had a serum malondi-aldehyde level of 29.30±0.37 nM/h. Administration of sodium oxalate (100 mg/kg body weight) produced a statistically significant increase in serum level of malondi-aldehyde ($p<0.05$). Consumption of low concentration of tea (50 mg/kg body weight) did not seem to protect the rats. However, increasing the concentration of tea con-sumed to 200 mg/Kg body weight produced a statis-tically significant decrease in the level of malondi-aldehyde. A similar trend was observed in the liver and kidney.

In the liver, the initial level of malondialdehyde was 5.92 ± 0.47 nM/h/g of tissue. Administration of sodium oxalate (100 mg/kg body weight) produced a level of 9.12 ± 0.85 nM/h/g, the difference between the control and experimental rats was significant ($p<0.05$). The level of malondialdehyde in the kidney of untreated rat was 4.06 ± 0.30 nM/h/g. Treatment of rats with sodium oxalate (100 mg/kg body weight) increased this level to 7.63 ± 0.78 nM/h/g, while administration of 200 mg tea/kg body weight offered about 60% protection.

After 20 days consumption of tea, the level of malondialdehyde in untreated rats was significantly lower ($p<0.05$) compared to rats treated with sodium oxalate.

Table 1a. Effect of 10 days consumption of tea extract on the level of malondialdehyde in sodium oxalate treated rats.

Groups	Serum (nM/h)	Liver (nM/h/g)	Kidney (nM/h/g)
Normal (No treatment)	29.291± 0.365	5.923± 0.465	4.062± 0.298
Control (normal+10 mg sodium oxalate)	47.855± 1.050^{a}	9.120± 0.850^{a}	7.634± 1.068^{a}
Control + 5 mg extract	47.427± 3.082^{a}	8.323± 1.136^{ab}	6.091± 0.368^{ab}
Control + 10 mg extract	37.284± 2.068^{abc}	7.142± 1.150^{abc}	5.788± 0.350^{abc}
Control + 20 mg extract	32.186± 0.882^{abcd}	6.193± 0.750^{abcd}	4.523± 0.950^{bcd}

Values are mean ± S.E.M. for five determinations. A = significantly different from normal at P<0.05; b =significantly different from control at P<0.05; c = significantly different from group fed with 5 mg tea extract at P<0.05; d = significantly different from group fed with 10 mg tea extract at P<0.05.

Table 1b. Effect of 20 days consumption of tea extract on the level of malondialdehyde in sodium oxalate treated rats.

Groups	Serum (nM/h)	Liver (nM/h/g)	Kidney (nM/h/g)
Normal (No treatment)	27.528± 2.065	5.221± 0.365	3.090± 0.598
Control (normal+10 mg sodium oxalate)	42.063± 3.099^{a}	7.559± 0.850^{a}	6.560 ± 1.068^{a}
Control + 5 mg extract	39.754± 0.965^{ab}	7.023± 0.536^{a}	5.618 ± 1.268^{ab}
Control + 10 mg extract	34.043± 2.050^{abd}	6.036 ± 0.652^{abc}	5.121 ± 0.958^{ab}
Control + 20 mg extract	31.422± 1.099^{abcd}	5.858 ± 1.924^{abcd}	3.902 ± 0.557^{abcd}

Values are mean± S.E.M for five determinations. a=significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from group fed with 5mg tea extract at P<0.05. , d =significantly different from group fed with 10mg tea extract at P<0.05.

Consumption of low concentration of tea (50 mg/Kg body weight) for 20 days did not seem to protect the rats but consumption at higher concentration of tea produced a statistically significant lowering of malondialdehyde level ($p<0.05$). Moderate increase in malondialdehyde level was observed in rats treated in the liver and kidney. Prolonged tea consumption at higher concentration produced about 20% protection from sodium oxalate effect in either the liver or the kidney. There was a significantly pronounced increase in the rate of production of malondialdehyde in both the serum and liver and kidney homogenates of the rats treated with sodium oxalate.

The induction of lipid peroxidation in the test animals may be a consequence of the direct influence of oxalate. This observation is quite interesting because of the fact that calcium oxalate is the major constituent of most renal stones implicated to cause the release of higher peroxides. Since calcium chloride has no effect on lipid peroxidation (Selvam and Kurien, 1987); it is oxalate that induces this action.

Consumption of tea produced a dose dependent decrease in the rate of formation of malondialdehyde in the serum, liver and the kidney. Prolonged tea consumption has been shown to have a significant inhibitory effect on sodium oxalate-induced lipid peroxidation in rats. This is evident in the decrease in the resultant rate of formation of Thiobarbituric Reactive Substances (TBA-RS) observed in rats fed with tea extracts for 10 days and 20 days accordingly. This corroborates the findings of Sano et al. (1995) that tea has an antioxidant property against lipid peroxidation in rats.

Effect of tea consumption on catalase activity of sodium oxalate treated rats

The catalase activity in the serum, liver and kidney of rats consuming tea after sodium oxalate treatment is presented in Tables 2a and b. The initial serum catalase activity for control rats at 10 and 20 days was identical (8.6%). Rats which received sodium oxalate had a slightly lower catalase activity. Consumption of tea produced a higher level of catalase at 10 and 20 days of treatment; the difference between the experimental control and treated rats was significant ($p<0.05$). However, in the liver and kidney, catalase activity was significantly higher in rats treated with tea ($p<0.05$). The difference was quite significant at 20 days.

There was a decreased level in catalase activity in the serum as well as in the tissues. Catalase is one of the most potent catalysts known. The reactions it catalyses are crucial to life. Catalase catalyses the conversion of hydrogen peroxide, a powerful and potentially harmful oxidizing agent, to water and molecular oxygen. This

Table 2a. Effect of 10 days consumption of tea extract on catalase activity in sodium oxalate treated rats.

GROUP	Serum		Liver		Kidney	
	VOl. of H_2O_2 (Vf) ml	%A	VOl. of H_2O_2 (Vf) ml	%A	VOl. of H_2O_2 (Vf) ml	%A
Normal(No treatment)	0.285±0.005	8.60	0.233±0.004	10.28	0.221±0.012	11.16
Control (Normal +10mg Sodium Oxalate)	0.325±0.004	7.00^{a}	0.359±0.003	5.64^{a}	0.324±0.011	7.04^{a}
Control + 5mg extract	0.326±0.024	6.96^{a}	0.259±0.013	9.64^{ab}	0.221±0.020	11.16^{b}
Control+10mg extract	0.225±0.005	11.00^{abc}	0.189±0.017	10.12^{ab}	0.237±0.024	10.52^{b}
Control +20mg extract	0.250±0.025	10.00^{abc}	0.337±0.024	6.52^{abcd}	0.286±0.026	8.56^{abcd}

Values are mean± S.E.M for five determinations. a=significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from group fed with 5mg tea extract at P<0.05. , d =significantly different from group fed with 10mg tea extract at P<0.05. Note: %A = Percentage Activity. Values are mean± S.E.M for five determinations. a=significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from group fed with 5mg tea extract at P<0.05. , d =significantly different from group fed with 10mg tea extract at P<0.05.

Table 2b. Effect of 20 days consumption of tea extract on catalase activity in sodium oxalate treated rats

GROUP	Serum		Liver		Kidney	
	VOl. of H_2O_2 (Vf) ml	%A	VOl. of H_2O_2 (Vf) ml	%A	VOl. of H_2O_2 (Vf) ml	%A
Normal(No treatment)	0.288± 0.029	8.48	0.134± 0.024	14.64	0.179± 0.032	12.84
Control (Normal +10mg Sodium Oxalate)	0.324± 0.034	7.04^{a}	0.318± 0.083	7.28^{a}	0.285± 0.019	8.60^{a}
Control +5mg extract	0.248± 0.034	10.08^{ab}	0.294± 0.053	8.24^{ab}	0.116± 0.027	15.36^{ab}
Control+10mg extract	0.286± 0.025	8.56^{abc}	0.066± 0.007	17.36^{abc}	0.108± 0.026	15.68^{ab}
Control +20mg extract	0.196± 0.027	12.18^{abcd}	0.076± 0.025	18.96^{abcd}	0.097± 0.014	16.12^{ab}

Note: %A = Percentage Activity. Values are mean± S.E.M for three determinations. a=significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from rats fed with 5mg tea extract at P<0.05. , d =significantly different from rats fed with 10mg tea extract at P<0.05.

decrease is thought to occur as a result of accumulation of hydrogen peroxide, which is a product of peroxidation in tissues. As hydrogen peroxide concentration increases, more and more of the enzyme will be used up in an attempt to clear off the accumulating hydrogen peroxide. This result agrees with the findings of Selvam and Kurien (1987) who reported a significant decrease in catalase activities in both the liver and kidney 12 h after the administration of sodium oxalate in rats.

The mechanism of induction of lipid peroxidation by sodium oxalate may involve inhibition of catalase activity *in vivo* since *in vitro* studies have revealed progressive inhibition of catalase activity and increase in lipid peroxidation with increasing oxalate concentration (Selvam and Kurien, 1987). However, the control groups showed a significant increase in catalase activity as a result of tea consumption. The increase observed also followed a dose-dependent pattern with the most pronounced increase found when rats were fed for a period of 20 days. This result also presupposes that prolonged tea consumption could be chemo-preventive by gradually causing an increase in the activity of catalase.

This result, coupled with the finding that tea consumption of tea lowers the rate of production of TBA-RS in the serum and the tissues, could also suggest that the increase in catalase activity observed may be as a result of reduced extent of lipid peroxidation which in turn reduces the concentration of hydrogen peroxide that accumulates in the tissues and in the serum. This therefore means lesser amount of catalase enzyme will be used up at any given time.

Aspartate and Alanine transaminases activity in the serum, liver and kidney of sodium oxalate treated rats

AST activity in the serum, liver and kidney of rats consuming tea after sodium oxalate treatment is presented in Tables 3a and b. Sodium oxalate treatment produced a statistically significant increase in the AST level in the serum, liver and kidney. Consumption of tea within 10 days produced a significant decrease in AST activity in the serum, kidney and liver. The kidney has almost 60% protection. After 20 days of consuming the

Table 3a. Effect of 10 days consumption of tea extract on AST activity in sodium oxalate treated rats in IU.

GROUP	Serum	Liver	Kidney
Normal(No treatment)	13 ± 0.65	19± 0.68	13± 0.64
Control (Normal +10mg Sodium Oxalate)	59 ± 2.95^{a}	76± 4.56^{a}	52± 2.53^{a}
Control + 5mg extract	41± 2.46^{ab}	47± 2.35^{ab}	31± 1.58^{ab}
Control +10mg extract	36 ± 1.80^{abc}	41± 2.67^{abc}	23± 1.21^{abc}
Control + 20mg extract	31± 1.40^{abcd}	36± 1.62^{abcd}	19± 1.20^{abcd}

Values are mean+ S.E.M for three determinations. a=significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from group fed with 5mg tea extract at P<0.05. , d =significantly different from group fed with 10mg tea extract at P<0.05.

Table 3b. Effect of 20 days consumption of tea extract on AST activity in sodium oxalate treated rats in IU

GROUP	Serum	Liver	Kidney
Normal(No treatment)	10+0.56	7+0.12	7+0.11
Control (Normal +10mg Sodium Oxalate)	41+ 3.12^{a}	52+3.21^{a}	41+3.24^{a}
Control + 5mg extract	31+ 1.28^{ab}	41+ 1.56^{ab}	31+ 2.11^{ab}
Control +10mg extract	27+ 1.20^{abc}	31+ 1.22^{abc}	27+ 1.24^{abc}
Control + 20mg extract	36+ 2.01^{abcd}	41 + 2.51^{abd}	36+ 2.56^{abcd}

Values are mean+ S.E.M for three determinations. a=significantly different from normal at P<0.05 b=significantly different from control at *P<0.05, c* =significantly different from group fed with 5mg tea extract at P<0.05. *, d* =significantly different from group fed with 10mg tea extract *at* P<0.05.

Table 4a. Effect of 10 days consumption of tea extract on ALT activity in sodium oxalate treated rats in IU.

GROUP	Serum	Liver	Kidney
Normal(No treatment)	19± 0.78	17± 0.69	17± 0.87
Control (Normal +10mg Sodium Oxalate)	39± 2.51^{a}	29± 2.01^{a}	25± 1.29^{a}
Control + 5mg extract	34+2.17^{ab}	25± 2.05^{ab}	21± 1.38^{ab}
Control +10mg extract	29± 3.02^{abc}	21± 1.68^{abc}	17± 0.93^{bc}
Control + 20mg extract	25± 1.25^{abcd}	17± 0.87^{bcd}	17± 0.82^{bc}

Values are mean+ S.E.M for three determinations. a=significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from group fed with 5mg tea extract at P<0.05. , d =significantly different from group fed with 10mg tea extract at P<0.05.

Table 4b. Effect of 20 days consumption of tea extract on ALT activity in sodium oxalate treated rats in IU.

GROUP	Serum	Liver	Kidney
Normal(No treatment)	21± 1.62	17± 0.67	19± 1.05
Control (Normal +10mg Sodium Oxalate)	36± 2.35^{a}	31± 1.28^{a}	31± 2.31^{a}
Control + 5mg extract	34± 2.47^{a}	25± 2.05^{ab}	21± 1.27^{ab}
Control +10mg extract	29± 1.68^{ab}	25± 1.89^{ab}	17± 0.98^{bc}
Control + 20mg extract	43± 2.95^{ab}	34± 2.54^{abcd}	34± 2.36^{abcd}

Values are mean+ S.E.M for three determinations. a=significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from group fed with 5mg tea extract at P<0.05. , d =significantly different from group fed with 10mg tea extract at P<0.05.

tea extracts, though there was a decrease in AST level in the serum, liver and kidney, the difference was not statistically significant compared to the experimental control group.

Effects of 10 and 20 days consumption of tea extract, the serum and tissue levels of ALT activity in sodium oxalate treated rats is presented in Tables 4a and b. Administration of 100 mg/kg body weight of sodium oxalate to experimental rats produced a statistically significant rise in the level of ALT as compared with untreated rats (p<0.05). Consumption of tea produced a concentration dependent decrease in the activity of ALT.

Table 5. Effect of 10 and 20 days consumption of tea extract on serum vitamin C content of sodium oxalate treated rats in mg/100ml.

GROUP	After 10 days	After 20 days
Normal(No treatment)	92.08± 12.36	88.18± 4.87
Control (Normal +10mg Sodium Oxalate)	45.39± 9.75[a]	55.76±.20[a]
Control + 5mg extract	53.17± 5.86[ab]	67.44± 3.25[ab]
Control +10mg extract	72.62± 2.10[abc]	83.00± 2.68[abc]
Control + 20mg extract	79.11± 5.13[abcd]	101.15± 7.89[abcd]

Values are mean± S.E.M for three determinations. a =significantly different from normal at P<0.05 b=significantly different from control at P<0.05, c =significantly different from group fed with 5mg tea extract at P<0.05. , d =significantly different from group fed with 10mg tea extract at P<0.05.

Prolonged consumption of tea (for 20 days) slightly decreased the level of ALT. The difference between the untreated and experimental rats was not significant.

One of the mechanisms in this model is considered to be initiated by the accumulation of oxalate which causes consecutive lipid peroxidation of the cell membranes and endoplasmic reticulum. The peroxidative products caused the cell membrane to become leaky with the consequent release of these enzymes into the blood. This agrees with the work of Masayuki et al. (2002) who reported that lipid peroxidation is recognized to be a major factor in the liver injury model.

The decreased level of AST and ALT observed after consumption of the black tea extract would suggest that the release of these enzymes had been inhibited. Probably, a chemical component in the tea is stabilizing the integrity of the cell membrane, keeping the membrane intact and the enzymes enclosed.

Effect of tea consumption on Vitamin C depletion by sodium oxalate

The concentration of vitamin C in the serum on control rats and rats consuming tea after sodium oxalate treatment is presented in Table 5. Concentration of Vitamin C in the control rats was 92.1 mg/100 ml; administration of sodium oxalate decreased this to about 50%. Consumption of tea for either 10 or 20 days produced an increase in the serum level of vitamin C. The difference in the vitamin C content between 10 and 20 days and between normal and control rats were statistically significant ($p<0.05$).

There was a significant decrease in the serum concentration of reduced ascorbic acid in rats treated with sodium oxalate. This depletion may be a direct consequence of the accumulation of oxalate in the system of those experimental animals. The antioxidant effect of Vitamin C is well established and it is also known to have protective functions against oxidative damage to lipid membranes. This protective ability may involve degradation of some of the vitamin C molecules present in the blood.

Animals fed with tea were able to conserve the amount of vitamin C in the serum. There is also a marked difference between the serum concentrations of reduced ascorbic acid in the control and experimental groups. This conservation could be explained to be as a result of the fact that certain constituents of black tea extract are capable of free radical quenching just as vitamin C. As these constituents of black tea extract accumulates in the blood, more vitamin C molecules are freed from the free radical quenching responsibility, hence leading to the conservation of ascorbic acid in the serum.

This ascorbic acid conservation characteristic of tea extract, coupled with its ability to modulate catalase activity positively and impede lipid peroxidation, is an indication of the fact that tea extract has the wherewithal to boost body's antioxidant capacity. The observation that prolonged consumption of tea enhances vitamin C conservation and prevented vitamin C depletion by sodium oxalate (100 mg/kg body weight) would suggest the antioxidant properties of vitamin C in protecting the cell against oxidative stress.

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