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ANTIOXIDANT AND RADICAL SCAVENGING EFFECTS OF TEA AGAINST OXIDATIVE STATUS OF LIPOPROTEIN IN MALE MICE

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ABSTRACT

To investigate the effect of tea polyphenols against oxidative status of lipoprotein, thirty six male mice were used. Six groups of mice (6 each) were assigned. Groups 1 and 2 (GI and GII) were served as negative and positive control groups respectively. Groups 3 and 4 (GIII and GIV) drank black (12.5 gm/L) and green (12.5gm/L) teas respectively, while groups 5 and 6 (GV and GVI) drank black and green teas respectively and both were injected with cyclophosphamide (20 mg/kg body weight, i.p) for three consecutive days, (used also with GII). The experimental period extends for 73 days.

Results indicated that the bioactive ingredients of tea caused inhibition of urine nitrite and hydroxylamine formation in tea drinking groups (GIII and GIV), while liver cytochrome P450 decreased in green tea group (GIII). Also both teas lowered the concenteration of plasma cholesterol, triglycerides (TG), low density lipoprotein cholesterol (LDL), and very low density lipoprotein cholesterol (VLDL) lipid fractions, while it increased the high density lipoprotein cholesterol (HDL), in group GV. Total antioxidant capacity (TAC) increased in all treated groups (GIII, GIV, GV and GVI), while it decreased in GII. Meanwhile Thiobarbituric acid reactive substansces (TBARS) showed significant reduction in both green tea treated groups (GIV and GVI). In conclusion, both black and green teas polyphenols improved blood lipid profile, strengthens blood plasma antioxidant capacity leading to decrease in oxidation product levels TBARS, which demonstrated reduced oxidation reaction in the body. This work also showed that teas supplementation were capable of inhibiting the nitrosation of secondary amine.

Keywords: Green and black tea,nitrite, Lipid fractions, TBARS.

INTRODUCTION

Tea is particularly rich in polyphenols, including catechins, theaflavins and thearubingins, which are thought to contribute to the health benefits of tea. Tea polyphenols act as antioxidants in vitro by scavenging reactive oxygen and nitrogen species and chelating redox-active transition metal ions (Balz and Jane, 2003 and Peterson et al, 2005).

Green and black tea are both high in catechins. These compounds are powerful antioxidants, capable of rapid reduction of superoxide radical and alkyl proxy radicals. Catechins may also repair vitamin E radicals. Such potent antioxidant ability may be important in inhibiting the in-vivo oxidation of LDL and VLDL and the subsequent atherogenesis (Vinson and Dabbagh, 1998).

Oxidation of cholesterol fraction, in particular of low-density lipoprotein (LDL) cholesterol, has been accepted as playing an important role in atherosclerosis (Liu *et al.*, 1992). Choleterol, Cholesterol esters and

triglyceride components of the lipoprotein fractions can be oxidized by toxic radicals and can lose their chemical structure and cellular functions (Durak *et al.*, 2004 and Gramza and korczak, 2005). Lipid peroxidation is accepted to be a free radical process implicated in the formation of athreosclerosis (Wen *et al.*, 1996), and the aldehyde products of lipid hydroperoxide breakdown to be responsible for the modification of LDL apoprotein (Estrbaure *et al.*, 1993).

Green tea extracts are described as preventive against induced cancers in animals (Wang *et al.*, 1991). Epigallocatechin gallate (EGCG), a major constituent of green tea polyphenolics, has been shown to inhibit the promotion step of induced carcinogenesis in animals. Catechins are known to be reactive towards nitrite (Bartsch *et al.*, 1993). Since a major source for the exposure of humans to carcinogenic nitrosodialkylamines is suspected to be nitroso compounds formed in the digestive tract from dialkylamines and nitrite, degradation of nitrite by dietary catechins may be regarded as a possible protective measure against human exposure to these carcinogenic compounds (Tanaka *et al.*, 1998).

The present study was designed to ascertain the beneficial role of tea polyphenols against oxidative statue of lipoprotein and nitrite-derived health hazards in male mice.

MATERIALS AND METHODS

1.Chemicals

Cyclophosphamide, thiobarbituric acid and all other chemicals used were purchased from Sigma Chemical Company (Saint Louis, USA).

2. Animals

Thirty six male mice with average body weight of 25-30 gm were obtained from Medical Research Institute, Alexandria University, Egypt, and acclimated for two weeks prior to the experiment. They were assigned to six groups and housed in Universal galvanized wire cages at room temperature (22-25 °C) and in a photoperiod of 12 hrs/day. Animals were provided with a commercial balanced diet.

3. Experimental design

Six groups of mice (6 each) were randomly assigned as: Group I (GI) served as control and injected with physiological saline, group II (GII) was injected with cyclophosphamide (20 mg/kg body weight, i.p.) for three consecutive days to induce oxidative stress, group III (GIII) drank black tea (12.5 gm/L) and injected with physiological saline, group IV (GIV) drank green tea (12.5 gm/L) and injected with physiological saline, group V (GV) was injected with cyclophosphamide (20 mg/kg body weight, i.p.) and drank black tea, group VI (GVI) was injected with cyclophosphamide (20 mg/kg body weight, i.p.) and drank green tea. Urine was collected weekly (for nine weeks) after 7 days post cyclophosphamide treatment, using metabolic cages. Animals were decapitated at the end of the experimental period (73 days).

4. Tissue preparation

4.1. Preparation of liver microsomes

At the end of the treatmental period, mice were fasted for 24 hrs prior to being sacrificed by cervical dislocation. The abdominal cavity was opened immediately and liver was removed, washed with cold 0.1 M phosphate buffer, pH 7.4, weighed and chilled on ice. All the following procedures were carried out in cold conditions. A 33% (W/V) crude homogenate was prepared in 0.1 M phosphate buffer, pH 7.4 by homogenization with a teflon pestle, using 5 strokes. The crude homogenate was then centrifuged at 11,000 xg for 20 min at 4°C to remove the intact cells, nuclei and mitochondria. The supernatant solution was subsequently centrifuged at 105,000 xg for 60 min at 4°C to sediment the microsomal pellet. The pellet was resuspended in 0.1 M phosphate buffer, pH 7.4, kept in ice bath and used as the enzyme source.

4.2. Separation of blood plasma

Blood samples were obtained by sacrificing the animals, and were placed immediately on ice. Heparin was used as anticoagulant. Plasma was obtained by centrifugation at 3,000 rpm for 20 min and then stored at –20°C until used for analysis.

5. Biochemical assays

5.1. Protein determination

Protein concentration of the hepatic microsomal fraction was determined by the method of Lowery *et al.* (1951).

5.2. Liver cytochrome P450

Liver microsomal cytochrome P450 was determined according to Omura and Sato (1964), using molar extinction coefficient 91 cm^{-1} mM^{-1}.

5.3. Blood biochemical assays

Blood plasma cholesterol was determined using commercial kits obtained from Bio ADWIC, Egypt. Plasma triglyceride (TG) was determined by triglyceride-GPO kits obtained from Pasteur Lab, Egypt, according to McGowan *et al.* (1983). Plasma low-density lipoprotein (LDL) and serum high-density lipoprotein (HDL) were assayed using Biosystems reagents Kits, Spain, according to Assman *et al.* (1984) and Biosystems reagents Kits, Spain, according to Burstein *et al.* (1980), respectively. Plasma very low density lipoprotein (VLDL) was calculated from triacylglycerols according to Friedwald *et al.* (1972) who reported that VLDL is present in a concentration equal to one fifth of triacylglycerols concentration in blood plasma of less than 400 mg/dl. Thiobarbituric acid-reactive substances (TBARS), were measured in blood plasma as described by Tapel and Zalkin (1959). The color intensity of the TBARS reactants was measured at 532 □m and a molar extinction coefficient of 156,000 cm^{-1} M^{-1} was used for calculation of the concentration. Total antioxidant capacity was measured according to the method of Koracevic *et al.* (2001) using commercial kits obtained from Biodiagnostic Co., Egypt.

5.4. Urine biochemical assays

Hydroxylamine and nitrite in urine were determined using formation of Azo-dye compound according to Feigel and Anger (1966).

6. Statistical analyses

Statistical analyses were made to obtain the standard deviation and standard errors of mean. The data for the treated animals were compared with data for the control animals by using the Student's t-test SAS (2000).

RESULTS AND DISCUSSION

Nitrite represents a potential hazard because of its involvement in the nitrosation reaction. The present study showed that tea polyphenols, in particular green tea, were markedly reduced nitrite formation specially in the last four weeks in urine samples (Figure 1).

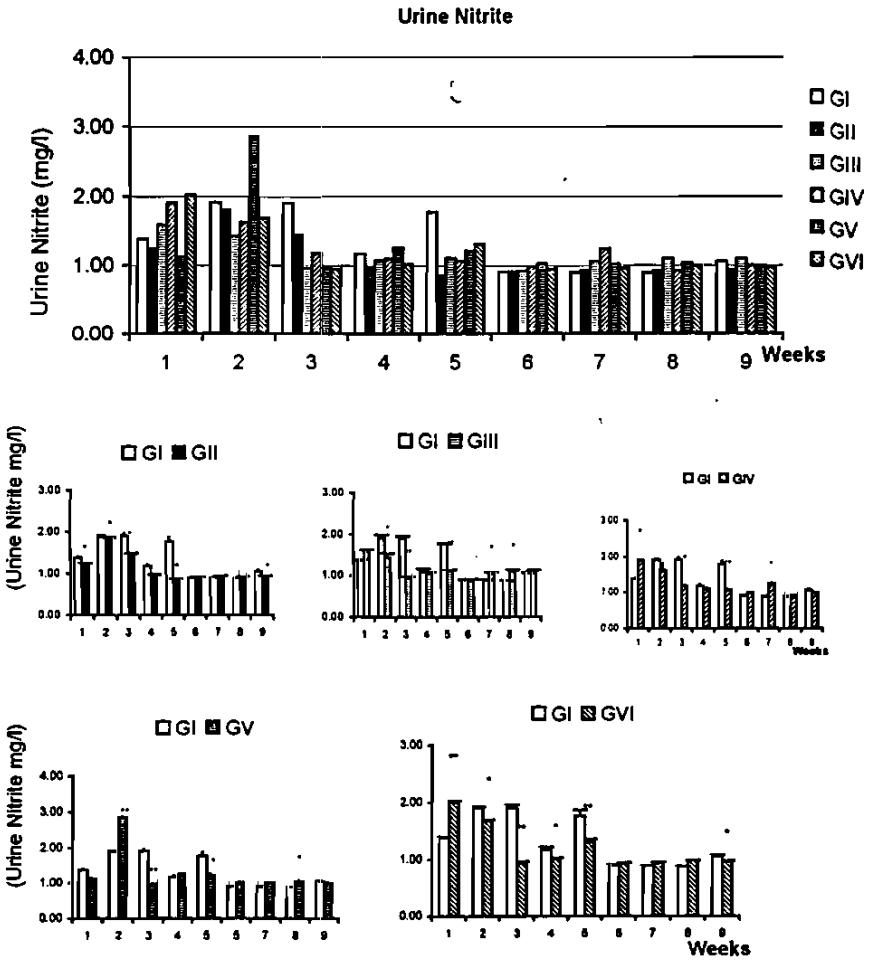

Fig 1: Effect of teas supplementation on nitrite (mg/l) formation in male mice urine
(NS = $p > 0.05$, * = $p < 0.05$, ** = $p < 0.01$, ***= $p < 0.001$)

It was noticed that cyclophosphamide (positive control) has no adverse effect in regard with nitrite. The same trend was obsearved for hydroxyl amine, where green tea polyphenols highly affected the inhibition of hydroxylamine formation, also cyclophosphamide has no adverse effect towards hydroxylamine (Figure 2). There are number of reports dealing with inhibition of nitrosodiakylamine formation by dietary components (Bartsch *et al.*, 1993).

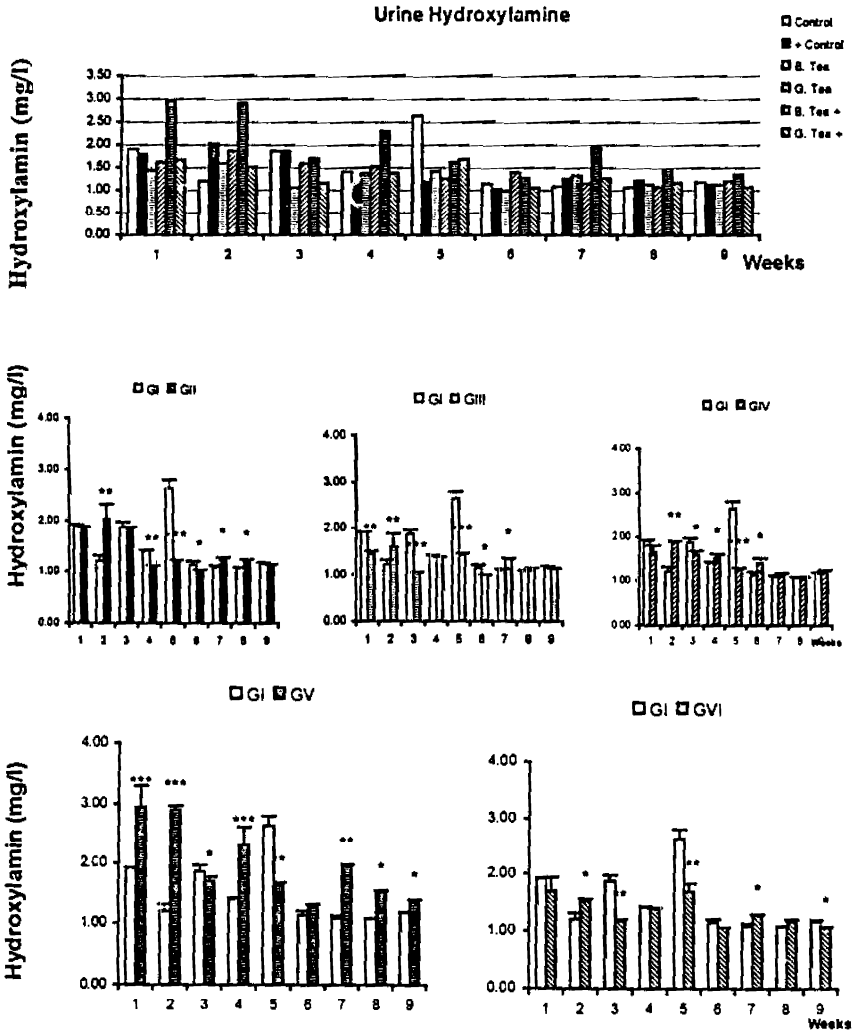

Fig 2: Effect of teas supplementation on hydroxylamine (mg/l) formation in male mice urine.
(NS = $p > 0.05$, * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$)

Regarding the tea extracts, Nakamura and Kawabata (1981) briefly described the blocking ability of the extracts against N-nitrosodimethylamine (NDMA) formation in vitro from dimethylamine and nitrite. The present study revealed that tea polyphenols, in particular green tea, is a powerful antioxidant capable of rapid inhibiting formation of nitrite, which are involved in the nitrosation reactiom with appropriate nitrosatable substrate (S) giving rise to the formation of a potent of chemical carcinogens, the N-nitroso compounds, most of which have induced tumors in many species of laboratory animals tested, and in virtually every tissue (Schlag *et al.*, 1982).

The same observation was indicated for hydroxylamine, where tea polyphenoles were capable of reducing the formation of this hazardous compound. Hydroxylamine derivatives are formed in the liver and then converted into glucuronide. The glucuronide conjugate is excreted in urine, where the acidic pH can convert it back to hydroxylamine which is rearranged to form nitrenium ion by a loss of water.

The electrophilic nitrenium ion can then react with nucleophilic targets in urinary bladder epithelium (Kaldlubar *et al.*, 1977).

The importance of nutrition in protecting the living organisms from the toxic effects of environmental carcinogens has recently been realized. Table (1) revealed that the hepatic content of microsomal cytochrome P450 was significantly ($p<0.05$) decreased by 33% in the group treated with green tea. Meanwhile, cyclophosphamide significantly ($p<0.05$) increased this content by 67% than that of the control group.

The cytochrome P450 enzymes are responsible for the oxidation of xenobiotic chemicals including drugs, pesticides and carcinogens. Inhibition of cytochrome P450 system was found to be effective in protecting the liver against the toxicity of a wide variety of toxic agents (Sheweita *et al.*, 2001 and Jorquera *et al.*, 1996). Treatment of male mice with black and green tea only was found to decrease the hepatic content of cytochrome P450. Inhibition of cytochrome P450 could protect the liver against the possible side effects of cyclophosphamide.

The effect of tea polyphenols on cholesterol, triglycerides (TG), high density lipoprotein cholesterol (HDL), low density lipoprotein cholesterol (LDL), very low density lipoprotein cholesterol (VLDL) were also investigated. Results presented in (Table 1) showed that black tea, green tea, black tea+cyclophosphamide and green tea+cyclophosphamide treated groups significantly ($p<0.001$) decreased the level of cholesterol by 29, 34, 32 and 36% respectively. Triglycerides were significantly ($p<0.001$) decreased in the same above mentioned groups by 25, 43, 28 and 35% respectively. HDL was significantly ($p<0.001$) decreased in cyclophosphamide treated group by 36%, meanwhile black tea + cyclophosphamide significantly ($p<0.001$) increased HDL by 27%. As shown in (Table 1), both teas were powerful inhibitors of LDL and VLDL.

Green tea was significantly better in vivo as antioxidant than black tea, where the total antioxidant capacity (TAC) increased significantly ($p<0.001$) in green and black tea treated groups compared with control by 78 and 41% respectively (Table 1).

Table 1: Effects of teas supplementation on liver microsomal cytochrome P450, blood plasma lipid, total antioxidant capacity and lipid peroxidation in male mice (Mean ± SEM).

Parameter	Control (GI)	Positive (GII)	Black T (GIII)	Green T (GIV)	Black (GV)	Green (GVI)
Cyt. P450 (p mol/mg protein)	2.27 ± 0.08	3.79 ± 0.18*	2.04 ± 0.09 NS	1.51 ± 0.01*	1.82 ± 0.14 NS	2.06 ±0.12 NS
Cholesterol (mg/dL)	329 ± 19.09	328 ± 49.3 NS	233 ± 12.2***	216 ± 9.2***	225 ± 11.0***	208 ±7.1***
Trigelycerids (mg/dL)	187 ± 13.7	165 ± 12.9**	140 ± 11.2***	106 ± 8.5***	134 ± 9.2***	122 ±11.0***
HDL (mg/dL)	36.8 ± 6.24	23.4 ± 3.35***	43.0 ± 6.10 NS	40.0 ± 6.42 NS	46.9 ± 5.28***	40.3 ±6.14 NS
LDL (mg/dL)	111.3 ± 12.97	56.7 ± 5.89***	57.0 ± 3.94***	39.3 ± 5.99***	54.5 ± 6.60***	33.6 ±4.39***
VLDL (mg/dL)	37.4 ± 2.73	33.0 ± 2.57**	28.0 ± 2.24***	21.3 ± 1.70***	26.7 ± 1.83***	24.4 ±2.19***
TAC (mM/L)	1.06 ± 0.10	0.86 ± 0.13 NS	1.49 ± 0.06***	1.89 ± 0.05***	1.32 ± 0.03**	1.71 ±0.03***
TBARS (n mole/g tissue)	2.97 ± 0.11	4.28 ± 0.18**	2.82 ± 0.04 NS	1.62 ± 0.07***	2.93 ± 0.03 NS	1.76 ±0.06***

NS = $p > 0.05$ * = $p < 0.05$ ** = $p < 0.01$ ***= $p < 0.001$

Thiobarbituric acid reactive substansces (TBARS), which is an important indicator of lipid peroxidation increased significantly ($p<0.01$) in cyclophosphamide treated group, meanwhile green tea decreased this level significantly ($p<0.001$) and corrected the damage caused by cyclophosphamide as seen in Table (1).

As seen from the results, tea polypenols, in particular green tea which represent the richest source of natural polyphenols. can lower blood cholesterol level and can improve blood lipid profile to a significant extent. It also increased blood plasma antioxidant potential and decreased TBARS level. All of these results showed that tea polyphenols exerted considerable antioxidant power in vivo as well, and protected cellular structures against peroxidation. This high antioxidant potential of tea may be a result of its high content of epicatechin and epigallocatechin gallate (Skrzydlewska *et al.*, 2002). Oxidation of cholesterol fractions (in particular, LDL) has been accepted as playing an important role in atherosclerotic process (Liu *et al.*, 1992), and because lipid peroxidation is a radial process implicated in this formation (Wen *et al.*, 1996). It has been proposed that extracts such as teas, that are rich in antioxidant content may confer beneficial effects in this regard. HDL has a protective function in the prevention of oxidation reaction and the consumption of antioxidant potency (Durak *et al.*, 2004). With respect to the cholesterol lowering properly of tea polyphenols, it has been suggested that some constituents (as tea epicatechins) may act as inhibitors of some enzymes such as hydroxy methyl glutaryl CoA reductase, which participates in cholesterol synthesis (Chan *et al.*, 1999).

In conclusion, the present work demonstrated that adding black and green tea in drinking water to mice for 73 days lowered the concenteration of lipid peroxidation products and increased the total antioxidant potential of the liver and blood plasma and improving lipid profile, also these results may be useful in evaluating the role of teas in the inhibition of tumor initiations.

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تأثير الشاى كمضادات للأكسدة وإمتصاص الشقائق الشاردة على حـالات أكـسدة الليبوبروتينات في الفئران

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أجريت هذه الدراسة للكشف عن مدى تأثير المواد عديدة الفينول الموجـودة فـى الـشاى بنوعيه الأسود والأخضر كمضادات للأكسدة على كل من أكسده الليبوبروتينات وبـدايات ظهـور الأورام. لذا صممت الدراسة على معاملة ٣٦ فأرا من ذكور فئـران التجـارب مقـسمة إلـى ٦ مجموعات، كل مجموعة ستة فئران. المجموعتان الأولى والثانية تمثل المجموعة الضابطة السالبة (حقن فى الغـشاء البريتـوني بمحلـول ملحـى) والموجبـة (حقـن بمـادة الـسيكلوفوسفاميد cyclophosphamide ٢٠ ملليجرام/ كجم) علي التوالى. المجموعتان الثالثة والرابعـة تمثـل المجموعات المتناولة لكل من الشاى الأسود والأخضر على التوالى بتركيز ١٢,٥ جرام/ لتر. بينما المجموعتان الخامسة والسادسة تمثل المجموعتان المعاملتان بمادة السيكلوفوسفاميد بنفس الجرعـة السابقة لمدة ثلاث أيام على التوالى مع تناول كل من الشاى الأسود والأخـضر، وقـد إسـتمرت الدراسة لمدة ٧٣ يوما.

أوضحت النتائج أن الجزء الفعال للشاى قد أدى إلى تثبيط معنوى في تكوين كـل مـن النترات والهيدروكسيل أمين فى البول لكل من المجموعتين الثالثة والرابعة كما أدت أيـضا إلـى نقص للسيتوكروم في الكبد فى المجموعة المتناولة للشاى الأخضر. أيضا أدت المعاملة بكل مـن الشاى الأسود والأخضر إلى نقص تركيز كل من كليستيرول بلازما الدم والدهون الثلاثية (TG) والليبوبروتينات منخفضة، وشديدة إنخفاض الكثافة (LDL, VLDL) على التوالى، بينما أدت إلى زيادة الليبوبروتينات عالية الكثافة (HDL) وخاصة فى المجموعة الخامسة. أوضحت النتـائج أن كل المجموعات المعاملة أدت إلى زيادة القدرة الكلية لمضادات الأكسدة بينما نقص معـدلها فـى المجموعة الضابطة الموجبة. فى نفس الوقت كان هناك نقص معنوى فى حمض الثيوباربتيوريـك (TBARS) فى كلا المجموعتين المتناولتين للشاى الأخضر.

من هذه الدراسة نستنتج أن المواد عديدة الفينول فى كل من الـشاى الأسـود أدت إلـى تحسين مستويات دهون الدم ، وقوت من زيادة القدرة الكلية لمضادات الأكسدة مما أدى إلى نقص مستوى أكسدة (TBARS) التى تثبط نقص التفاعلات المؤكسدة فى الجسم، ومن هذه الدراسـة أيضا إتضح أن إضافة الشاى بنوعيه له القدرة على إخماد تحول الأمينات الثانوية إلـى مركبـات النيتروزامين وهذا يوضح أهمية الشاى فى حماية الجسم ضد الأورام