

Anti Peptic Ulcer Activity of Tea Leaves

Prasenjit Mitra, Tanaya Ghosh and Prasanta Kumar Mitra*

Department of Biochemistry, North Bengal Medical College, Sushrutanagar - 734012 Dist. Darjeeling, West Bengal, India.

*Corresponding author : Dr. Prasanta Kumar Mitra, Present address: Professor & Head, Department of Medial Biotechnology, Sikkim Manipal University, Sikkim Manipal Institute of Medical Sciences, Gangtok, Sikkim, India. E.mail: dr pkmitra@rediffmail.com; Mobile Phone: +919434063026

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Abstract

Anti peptic ulcer activity of tea leaves was checked in cysteamine induced duodenal ulcer and ethanol, hydrochloric acid, aspirin, indomethacin, swimming stress, pyloric ligation and restraint induced gastric ulcer models in albino rats. Ranitidine, a conventional anti peptic ulcer drug, was used for comparison. Results showed that tea leaves powder could reduce ulcer index significantly (p<0.001) in all the experimental ulcer models studied and the results were comparable to that of ranitidine.

Keywords : Tea leaves, anti gastric ulcer activity, experimental gastric ulcer models.

Introduction

After water the cheapest beverage humans consume is tea. Almost two-thirds of the world's population take tea. This is due to pleasant taste, aroma and health benefits of tea. Tea is made from the processed leaf of *Camellia sinensis*. There are several health effects of tea. Regular

consumption of green tea lowered the risk of oesophageal cancer [1], lung cancer [2], colorectal and primary liver cancers [3], pancreatic cancer risk [4], gastric cancer [5], prostate cancer [6] and breast cancer [7]. In 2001 Hakim and Harris showed [8] that black tea consumption had a protective effect on cutaneous malignant melanoma risk.

Tea has beneficial effects against arthritic disease in humans. It is reported that those who drank tea had greater bone mineral density than those who did not drink tea [9]. It is also reported that women consuming 3 cups/day of tea had decreased risk of rheumatoid arthritis compared with women who do not drink tea [10].

In 2003 Tan and coworkers [11] showed that daily tea consumption protects neurological diseases. It was noted that persons taking 3 cups of tea/day had reduced risk of Parkinson's disease [12].

Tea consumption is good for diabetics, affects glucose metabolism and insulin signaling. Regular daily intake of black tea improves oxidative stress biomarkers and decreases serum C-reactive protein levels in type 2 diabetic patients. This gives good effects in health of diabetics [13]. In 2009 Nagao *et al* [14] showed that daily consumption of tea improves blood glucose control in patients with type 2 diabetes. Song *et al.* (2005) found that women who consumed more than 4 cups/day of tea had a 30% lower risk of developing type 2 diabetes than did those who did not consume tea [15].

Increased oxidative stress has been reported to be involved in cardiovascular disease development. In cardiovascular disease lipid metabolism is usually impaired [16]. Daily tea consumption reduces oxidative stress thereby modulates impaired lipid metabolism in cardio vascular patients. This is due to the presence of flavan-3-ols in tea leaves which are the anti oxidant molecules [17]. Further, endothelial function is recognized as a biomarker of cardiovascular health. Dysfunction of the endothelial layer is the etiological factor in atherogenesis [18]. In 2009 Park *et al.* noted that regular consumption of tea had positive impact

on endothelial and vascular functions in humans [19].

In spite of these medicinal values of tea, anti peptic ulcer activity of tea leaves is not well documented in literature. It was, therefore, thought worthwhile to study anti ulcerogenic effect of tea leaves in experimental peptic ulcer models.

Materials and Methods Tea leaves



Tea leaves (two leaves-one bud, *Camellia sinensis* L., Assam variety) were collected from Matigara Tea Estate, Siliguri, West Bengal, India in January – February, March- April, May - June, July - August, September - October, November - December, 2010. Leaves were identified by Taxonomist of the University of North Bengal, Siliguri, West Bengal and a voucher specimen was kept in the Department of Biochemistry, North Bengal Medical College, Siliguri, West Bengal for future reference. Leaves were shed dried and powdered. This powder was used as test drug.

Experimental animals

Wistar strain albino rats of both sex were used for the study. The animals were housed in colony

cages (4 rats/cage) and were kept for at least a week in the experimental wing of the animal house (room temperature 25–28 degree centigrade and humidity 60–65% with 12 h light and dark cycle) before experimentation. Animals were fed on laboratory diet with water *ad libitum*. For each set of experiment 8 animals were used. The animal experiment had approval of the institutional ethics committee.

Acute oral toxicity study

Acute toxicity studies were carried out on Swiss albino mice by the method of Ghosh MN, 2005 [20]. Tea leaves powder was given orally at doses of 100, 200, 500, 1000 and 3000 mg/kg to five groups of mice, each group containing six animals. After administration of the compound, the animals were observed for the first three hours for any toxic symptoms followed by observation at regular intervals for 24 hours up to seven days. At the end of the study, the animals were also observed for general organ toxicity, morphological behavior and mortality.

Chemicals

Cysteamine (Sigma Chemical Co.,USA), Ethanol (Baroda Chemical industries Ltd., Dabhoi), HCl LR (Thomas baker, Mumbai), Aspirin (Indosal Chemical Corporation), Indomethacin (Torrent Research Centre, Gandhinagar), Ranitidine (Cipla pharmaceuticals) were used in the study. All other chemicals used in this experiment were procured from Ranbaxy and SD Fine Chemicals, New Delhi, India.

Production of duodenal ulcers

This was done by the method of Parmar and Desai, 1993 [21]. To 18 h fasted rats (water was supplied *ad libitum*) cysteamine hydrochloride (400 mg/kg, p.o. in 10% aqueous solution) was administered in two doses at an interval of 4 h to produce duodenal ulcers. After 24 h of the first dose of cysteamine, animals were sacrificed by cervical dislocation and the duodenum was excised carefully and opened along the antimesenteric side. Duodenum was then examined for the presence of ulcers.

Production of gastric ulcers

Ethanol induced gastric ulcer

This was done by the method of Sairam *et al.*, 2001[22]. Rats were fasted for 18 h when no food but water was supplied *ad libitum*. Gastric ulcers were induced by administering ethanol (95%, 1 mL/200 g body weight) orally through a feeding tube. 1h after administration of ethanol, animals were sacrificed by cervical dislocation an the stomach was taken out and incised along the greater curvature. Stomach was then examined for the presence of ulcers.

HCl induced gastric ulcer

HCl induced gastric ulcer was developed in rats following the method of Parmar and Desai, 1993 [21]. 0.6M HCl (1 mL/200 g body weight) was orally administered to all rats. Rest part is same to that of ethanol induced gastric ulcer group.

Aspirin induced gastric ulcer

Aspirin induced gastric ulcer was produced by the method of Parmar and Desai, 1993 [21] with slight modification. Rats were fasted for 36 h when no food but water was supplied *ad libitum*. Aspirin is suspended in 1% carboxy methyl cellulose in water (20 mg/ml) and administered orally (gavage) in a dose of 500 mg/kg to the rats. Four hours after the pyloric ligation the animals were sacrificed by cervical dislocation. Rest part is same to that of ethanol induced gastric ulcer group.

Indomethacin induced gastric ulcer

This was done by the method of Parmar and Desai, 1993 [21]. Indomethacin(10 mg/kg) was given orally to rats in two doses at an interval of 15 hour. After the last dose of indomethacin, pylorus part of the animals was ligated. Four hours after the pyloric ligation the animals were sacrificed by cervical dislocation. The stomach was taken out, gastric juice collected, filtered and kept for subsequent investigations. Stomach was then incised along the greater curvature to examine ulcers.

Swimming stress induced gastric ulcer

Stress ulcer was induced in rats adopting the method of Alder R, 1984 [23]. Rats were fasted for 24h when no food but water was supplied *ad libitum*. Stress ulcer was induced by forced swimming in the glass cylinder (height 45 cm, diameter 25 cm) containing water to the height of 35 cm maintained at 25 degree centigrade for 3h. Rats were then sacrificed. Rest part was same to that of ethanol induced gastric ulcer group.

Induction of gastric ulcer by pyloric ligation method

This was done by the method of Parmar and Desai, 1993 [21]. Rats were fasted for 24h when no food but water was supplied *ad libitum*. Under light ether anesthesia, abdomen was opened and the pylorus was ligated. The abdomen was then sutured. After 4h the rats were sacrificed with excess of anesthetic ether and the stomach was dissected out. Rest part was same to that of ethanol induced gastric ulcer group.

Induction of gastric ulcer by restraint method

Restraint induced gastric ulcer was developed in rats following the method of Brodie and Hanson, 1960 [24]. Rats were fasted for 36 h when no food but water was supplied *ad libitum*. Each rat was then placed in single cage. Limbs of the rat were put together and tightened with adhesive tapes so that the animal could not move. Under this condition rats were kept for 24 hours and then sacrificed. Rest part was same to that of ethanol induced gastric ulcer group.

Evaluation of ulcer index

Evaluation of ulcer index was done by the method of Szelenyi and Thiemer, 1978 [25].

Gastric / duodenal lesions were counted and the mean ulcerative index was calculated as follows

- I Presence of edema, hyperemia and single sub mucosal punctiform hemorrhage.
- II Presence of sub mucosal hemorrhagic lesions with small erosions.
- III Presence of deep ulcer with erosions and invasive lesions.

Ulcer index = (number of lesion I) x1 + (number of lesion II) x2 + (number of lesion III) x3.

Anti gastric ulcer study of tea leaves & standardization of dose

Rats were divided into 7 groups ;

Group 1 : Control

Group 2 : Ethanol induced ulcer

Group 3 : Ethanol + Powder of tea leaves (0.5 g/kg)

Group 4 : Ethanol + Powder of tea leaves (0.75 g/kg)

Group 5: Ethanol + Powder of tea leaves (1.0 g/kg)

Group 6 : Ethanol + Powder of tea leaves (1.25 g/kg)

Group 7: Ethanol + Powder of tea leaves (1.5 g/kg)

(Powder of tea leaves, suspended in water, was given to rats orally through a feeding tube 30 minutes prior to administration of ethanol).

Results were as follow:

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
Ethanol	30.45 ± 3.19	
Ethanol + Tea leaves powder	24.43 ± 2.88	19.77
(0.5 g/kg)		
Ethanol + Tea leaves powder	20.48 ± 2.56	32.74
(0.75 g/kg)		
Ethanol + Tea leaves powder	$11.40 \pm 1.05 **$	62.56
(1.0 g/kg)		
Ethanol + Tea leaves powder	11.37 ± 1.04**	62.66
(1.25 g/kg)		
Ethanol + Tea leaves powder	11.28 ± 1.02 **	62.95
(1.5 g/kg)		

Values were mean \pm SEM of eight animals in each group. * p <0.05, **p < 0.001 when compared to drug control.

Results showed that anti gastric ulcer activity of tea leaves increased with dose, maximum activity was at 1g/kg thereafter activity remained same.

Anti gastric ulcer study of tea leaves in different experimental ulcer models

Rats were divided into 4 groups;

Group 1 : Control

Group 2 : Ulcerogenic drug or Method (Cysteamine hydrochloride / Ethanol / HCl / Aspirin/

Indomethacin / Swimming Stress / Pyloric ligation / Restraint Stress)

Group 3 : Ulcerogenic drug or method + Powder of tea leaves

(Powder of tea leaves, suspended in water, was given to rats orally in the dose of 1 g/kg through a feeding tube 30 minutes prior to administration of ulcerogenic agent / method)

Group 4 : Drug + Ranitidine : Ranitidine was given in the dose of 50 mg/kg p.o. 30 minutes before each dose of ulcerogenic drug / method. Dose of ranitidine was selected based on the report of Khare *et al.*, 2008 [26].

Statistical analysis

The values were expressed as mean \pm SEM and were analyzed using one-way analysis of variance (ANOVA) using Statistical Package for Social Sciences (SPSS) 20th versions. Differences between means were tested employing Duncan's multiple comparison test and significance was set at p < 0.05.

Results and Discussion

Acute toxicity studies

Acute toxicity studies revealed that tea leaves powder did not produce any toxic symptoms when administered orally to mice in doses of 100, 200, 500, 1000 and 3000 mg/kg. Animals were healthy, cheerful and behaved normal throughout the experimental period. No death of animal was recorded during seven days of experiment.

Effect of Tea Leaves on Cysteamine induced duodenal ulcer in albino rats.

Results are shown in Table -1.

Table-1. Effect of Tea Leaves (TL) on Cysteamine (CYS) induced duodenal ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
CYS	25.31 ± 1.32	
CYS + TL (1.0 g/kg)	$12.23 \pm 1.23 **$	51.67
CYS + Ranitidine (50mg/kg)	9.87 ± 1.17 **	61.00

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

Cysteamine hydrochloride (400 mg/kg, p.o. in 10% aqueous solution) when administered in two doses at an interval of 4 h produced duodenal ulcers in rats. There was bleeding in the duodenum. Adhesion and dilatation of the duodenum were seen. Ulcer index came, $25.31 \pm$ 1.32. When the rats were pretreated with tea leaves powder (1g/kg, orally), ulcer index came down to 12.23 ± 1.23 . Result was statistically significant (p<0.001). Pretreatment of rats with ranitidine (50 mg/kg) could decrease ulcer index to 9.87 ± 1.17 . Ulcer protection with tea leaves was 51.67% which was comparable to that of ranitidine group where protection was found 61%

Effect of Tea Leaves on Ethanol induced gastric ulcer in albino rats.

Results given in Table – 2 shows that ethanol produced massive gastric ulcers in all albino rats. Ulcers were mostly superficial. Bleeding of the stomach was followed by adhesion and dilatation. Ulcer index came 30.23 ± 2.17 . Pretreatment of rats with tea leaves powder (1g/kg)

Table-2. Effect of Tea Leaves (TL) on Ethanol (ETH) induced gastric ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
ETH	30.23 ± 2.17	
ETH + TL (1.0 g/kg)	11.43 ± 1.06 **	62.18
ETH + Ranitidine (50mg/kg)	9.45 ± 1.01 **	68.73

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

significantly reduced (p< 0.001) ulcer index induced by ethanol. Ulcer index came 11.43 ± 1.06 , Protection was 62.18%. This was comparable to that of ranitidine where protection came 68.73%.

Effect of Tea Leaves on HCl induced gastric ulcer in albino rats.

Results are shown in Table -3.

Table-3. Effect of Tea Leaves (TL) on HCl induced gastric ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
HCl	29.17 ± 1.86	
HCl + TL (1.0 g/kg)	13.22 ± 1.54 **	54.68
HCl + Ranitidine (50mg/kg)	11.13 ± 1.17 **	61.84

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

0.6 M HCl when administered to rats orally produced massive ulcers in stomach of all rats. Adhesion and dilatation of the stomach were seen. Ulcer index was 29.17 ± 1.86 . Pretreatment with tea leaves with the doses of 1 g/kg gave ulcer index 13.22 ± 1.54 . Result was statistically significant (p<0.001). There was protection (54.68%) with tea leaves from the production of HCl induced gastric ulceration which was comparable with ranitidine group where protection was found 61.84%.

Effect of Tea Leaves on Aspirin induced gastric ulcer in albino rats.

Table-4. Effect of Tea Leaves (TL) on Aspirin (ASP) induced gastric ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
ASP	32.42 ± 2.18	
ASP + TL (1.0 g/kg)	$15.28 \pm 1.75^{**}$	52.86
ASP + Ranitidine (50mg/kg)	$13.23 \pm 1.53 **$	59.19

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

Table – 4 shows that aspirin, suspended in 1% carboxy methyl cellulose in water (20 mg/ml), when administered orally in the dose of 500 mg/kg produced massive ulcers in stomach of the rats. Few ulcers were penetrating otherwise most of them were superficial in nature. There was profuse bleeding in the stomach. Adhesion and dilatation were seen in the stomach. Ulcer index came 32.42 ± 2.18 . Pretreatment of rats with tea leaves powder (1g/kg) significantly reduced ulcer index (15.28 ± 1.75). Ulcer protection was 52.86% which was comparable to that of ranitidine where protection came 59.19%.

Effect of Tea Leaves on Indomethacin induced gastric ulcer in albino rats.

Results are shown in Table -5.

Table-5. Effect of Tea Leaves (TL) on Indomethacin (INDO) induced gastric ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
INDO	29.44 ± 2.65	
INDO + TL (1.0 g/kg)	$10.13 \pm 1.05 **$	65.59
INDO + Ranitidine (50mg/kg)	9.21 ± 1.02**	68.71
		0.0.1

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

Indomethacin (10 mg/kg) when given orally to rats in two doses at an interval of 15 hours produced massive ulcers in glandular part of stomach of all rats. Out of 8 rats used in the experiment, 5 rats showed hemorrhage, 6 had acute dilation and 6 had adhesion in the stomach. Ulcer index was 29.44 ± 2.65 . Pretreatment with tea leaves with the doses of 1g/kg reduced ulcer index to 10.13 ± 1.05 . Result was statistically significant (p<0.001). In this group only 2 rats showed dilation in stomach Ulcer protection was 65.59%. Results were comparable with that of ranitidine group where ulcer protection came 68.71%.

Effect of Tea Leaves on swimming stress induced gastric ulcer in albino rats.

Table – 6 shows that forced swimming for 3 hours induced gastric ulcer in all rats. Ulcers were located in glandular part of the stomach. Most of the ulcers were superficial in nature. Bleeding,

Table-6. Effect of Tea Leaves (TL) on Stress (STR) induced gastric ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
STR	35.31 ± 2.45	
STR + TL (1.0 g/kg)	$17.22 \pm 1.75 **$	51.23
STR + Ranitidine (50mg/kg)	14.61 ± 1.43 **	58.62

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

adhesion and dilatation were found in the stomach. Ulcer index came 35.31 ± 2.45 . Pretreatment of rats with tea leaves powder (1g/kg) significantly reduced ulcer index (17.22 \pm 1.75). Ulcer protection was 51.23%. In ranitidine group, however, ulcer index came down to

 14.61 ± 1.43 with ulcer protection 58.62%.

Effect of Tea Leaves on pyloric ligation induced gastric ulcer in albino rats.

Results are shown in Table -7.

Table-7. Effect of Tea Leaves (TL) on pyloric ligation (PYL) induced gastric ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
PYL	31.33 ± 2.11	
PYL + TL (1.0 g/kg)	15.54 ± 1.34 **	50.39
PYL + Ranitidine (50mg/kg)	13.23 ± 1.14 **	57.77

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

Pyloric ligation for 4 hours produced ulcers in glandular part of stomach of all rats. Out of 8 rats used in the experiment, 2 rats showed acute dilation and 3 had adhesion in the stomach. Ulcer index was 31.33 ± 2.11 . Pretreatment with tea leaves with the doses of 1g/kg reduced ulcer index to 15.54 ± 1.34 . Result was statistically significant (p<0.001). In this group only 1 rat showed dilation in stomach Ulcer protection was 50.39%. In ranitidine group ulcer index was 13.23 ± 1.14 with ulcer protection 57.77%.

Effect of Tea Leaves on restraint induced gastric ulcer in albino rats.

Results are shown in Table -8.

Table-8. Effect of Tea Leaves (TL) on Restraint (RES) induced gastric ulcer in albino rats.

Group	Ulcer index (mean \pm SEM)	% Ulcer protection
Control	Nil	
RES	33.23 ± 2.42	
RES + TL (1.0 g/kg)	15.28 ± 1.07 **	54.02
RES + Ranitidine (50mg/kg)	14.37 ± 1.05 **	56.75

Results were in mean \pm SEM, Each group had eight rats, ** p<0.001

Restraint produced massive ulcers in glandular part of stomach of all rats. Out of 8 rats used in the experiment, 3 rats showed hemorrhage, 4 had acute dilation and 5 had adhesion in the stomach. Ulcer index was 33.23 ± 2.42 . Pretreatment with tea leaves with the doses of 1g/kg reduced ulcer index to 15.28 ± 1.07 . Result was statistically significant (p<0.001). In this group only 1 rats showed dilation in stomach Ulcer protection was 54.02%. Results were comparable to that of ranitidine group where ulcer index was 14.37 ± 1.05 and ulcer protection came 56.75%.

An ulcer is an open sore of the skin or mucus membrane characterized by sloughing of inflamed dead tissue [27]. Peptic ulcer refers to an ulcer in the lower oesophagus, stomach or duodenum, in the jejunum after surgical anastomosis to the stomach or, rarely in the ileum adjacent to a Meckel's diverticulum. When ulcer develops in stomach it is known as gastric ulcer. Likewise duodenal and esophageal ulcers occur in the duodenum and esophagus respectively. In 1963 Quincke [28] was probably the first to use the term 'Peptic ulcer'. Because of its frequency and worldwide distribution, peptic ulcer continues to be a subject of numerous investigations, both experimental and clinico pathological. In this respect peptic ulcer occupies a place secondary to carcinoma in the field of gastroenterology.

Peptic ulcer disease encompassing gastric and duodenal ulcer is the most prevalent

gastrointestinal disorder and affecting 10% of the world population [29]. Every year about fifteen thousand deaths occur as a consequence of peptic ulcer disease [30]. In India, peptic ulcer disease is also common.

Factors playing significant role in pathogenesis of peptic ulcer disease are: abnormalities in secretion of acid and pepsin, abnormalities of mucosal defense, delayed gastric emptying, reflux of bile and penetrate juice, emotional stress, role of microbes, genetic predisposition etc.[31]. However, the disease is generally characterized by the imbalance between gastric offensive factors like acid, pepsin secretion, *Helicobacter pylori*, nitric oxide, lipid peroxidation etc. and defensive mucosal factors like bicarbonate and mucin secretion, mucus proliferation, prostaglandins, glycoproteins, mucosal cell shedding and anti oxidating enzymes like super oxide dismutase, catalase as well as levels of glutathione [32]. Normally peptic ulcer develops when aggressive factors overcome the defensive factors.

Synthetic drugs are used for treatment of peptic ulcer [33]. In case, the ulcer is due to infection of *Helicobacter pylori* (*H. pylori*), the different medications are usually prescribed. This is known as "Triple therapy". This includes a proton pump inhibitor viz. omeprazole to reduce acid production and two antibiotics to get rid of the organism. Sometimes, instead of one of the antibiotics, bismuth salicylate may be the third medication recommended. This drug, available over the counter, coats and soothes the stomach, protecting it from the damaging effects of acid. Two, rather than three, drug regimens are currently being developed. For non *H. pylori* ulcers number of drugs are now available for treatment. These drugs are broadly classified into two categories [34].

- Those that decrease or counter acid pepsin secretion viz. ranitidine, famotidine etc. (H2 - blockers), pirenzepine, telenzepine etc. (M1 – blockers), omeprazole, lansaprazole etc. (proton pump inhibitors)
- 2) Those that affect cytoprotection by virtue of their effects in mucosal defense

factors like sucralfate, carbenoxolone etc.

No doubt the above said drugs have brought about remarkable changes in peptic ulcer therapy, the efficacy of these drugs is still debatable. Reports on clinical evaluation of these drugs show that there are incidences of relapses and adverse effects and danger of drug interactions during ulcer therapy [35, 36]. For example, proton pump inhibitors (omeprazole, lansoprazole) may cause nausea, abdominal pain, constipation, diarrhoea etc. and H2 receptor antagonists (cimetidine) may cause gynaecomastia as well as loss of libido.

Hence, search for an ideal anti – ulcer drug continues and has also been extended to medicinal plants / herbs in search for new and novel molecules, which afford better protection and decrease the incidence of relapse [37 - 44].

Beneficial effect of tea in peptic ulcer diseases is not clearly understood. Only few studies were undertaken to note anti peptic ulcer activity of tea. Maity et al. (1995) studied effect of the hot water extract of black tea (Camellia sinensis (L.) O. Kuntze, Theaceae) on ulceration induced by various ulcerogens and by cold restraint stress in albino rats [45]. They observed that the hot water extract of black tea possessed anti-ulcer activity and this activity was mediated through prostaglandins. In 2000, Yuk-kei yee & Marcel Wing-leung Koo showed [46] that tea has anti-Helocobacter pylori activity in a daily consumed concentration and epigallocatechin gallate present in tea was the active ingredient responsible for the action. Authors pointed out that H. pylori is the bacteria responsible for peptic ulcer diseases. In another study cytoprotective effect of tea root (Camellia sinensis var assamica) extract was studied in ethanol-induced rat gastric ulcer as an experimental model. The study provided evidence for possible involvement of both glutathione and nitric oxide in the tea root-mediated cytoprotection against ethanol-induced ulceration [47]. Hamaishi et al. in 2006 studied anti-ulcer effect of tea catechin in rats. Results showed that oral administration of tea catechin dose-dependently prevented absolute ethanolinduced or restraint plus water immersion stress-induced acute gastric mucosal injury in rats [48]. In 2009 Ratnasooriya et al. examined gastric ulcer healing potential of black tea (Camellia

sinensis) using Sri Lankan high grown Dust grade No: 1 black tea in rat acetic acid-induced

gastric ulcer model. Authors concluded that black tea possessed strong, oral gastric ulcer healing activity which was mediated via multiple mechanisms [49]. It was, therefore, thought worthwhile to study anti peptic ulcer activity of tea leaves in different experimental peptic ulcer models.

In present study we evaluated anti gastric ulcer activity of tea leaves in ethanol induced gastric ulcer in albino rats. Results showed that tea leaves powder could protect the rats from formation of gastric ulcer by ethanol. Result further showed that anti gastric ulcer activity of tea leaves increased with dose, maximum activity was at 1g/kg thereafter activity remained same (Fig – 1).





A: Ethanol, B: Ethanol & tea leaves (0.5 g/kg), C: Ethanol & tea leaves (0.75 g/kg) D: Ethanol & tea leaves (1.0 g/kg), E: Ethanol & tea leaves (1.25 g/kg), F: Ethanol & tea leaves (1.5 g/kg)

Since one experimental model is not sufficient to establish anti peptic ulcer activity of an agent,

it was thought worthwhile to study anti peptic ulcer activity of tea leaves in more experimental ulcer models. We, therefore, intended to study anti peptic ulcer activity of tea leaves in cysteamine induced duodenal ulcer and hydrochloric acid, aspirin, indomethacin, swimming stress, pyloric ligation and restraint induced gastric ulcer in albino rats. Results showed that in all the ulcer models studied tea leaves showed anti ulcer activity. The result was comparable to that of ranitidine, the standard drug for ulcer treatment (Fig - 3 & 4).





Figure - 4: Anti gastric ulcer activity of tea leaves powder against indomethacin, swimming stress, pyloric ligation and restraint induced gastric ulcer in albino rats.



Drug treated Drug and tea leaves Drug and ranitidine (Ulcer index)

In ulcer research emphasis has been given on rate of gastric secretion, pH [50], acidity and pepsin [33] as well as mucin [51]. Gastric mucosal lipid peroxidation has also been reported to increase incidence of experimental ulcers [22]. Work is now going on in this direction to evaluate the mechanism of anti peptic ulcer activity of tea leaves.

Conclusion

Anti peptic ulcer activity of tea leaves was studied in cysteamine induced duodenal ulcer and ethanol, hydrochloric acid, aspirin, indomethacin, swimming stress, pyloric ligation and restraint induced gastric ulcer models in albino rats. Results showed that tea leaves powder could reduce ulcer index significantly (p<0.001) in all the experimental ulcer models studied and the results were comparable to that of ranitidine, a conventional anti peptic ulcer drug,

From the present study it may be concluded that tea leaves may provide a scientific rationale for use as anti peptic ulcer drug.

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Authors Tolumn



Prof. (Dr.) Prasanta Kumar Mitra is a very senior medical teacher and researcher. He has completed thirty seven years in medical teaching and about forty years in research. His research area is 'Medicinal plants of India'. He has four Ph.D.s to his credit and published one hundred thirty nine research papers in national & international journals. Fifteen students did Ph.D. work under his guidance. He was co-supervisor of the research projects of five MD students. Prof. Mitra was Editor-in-Chief of the European Journal of Biotechnology and Biosciences. He is now Editor, Associate Editor and Member of Editorial Board of many national and international research journals. On behalf on Govt. of West Bengal Prof. Mitra worked as Coordinator of World Bank and GTZ projects for Health Sector Development in North Bengal. Prof. Mitra is a well known writer and science popularizer. He has written sixteen hundred ninety two popular science articles in different newspapers / magazines. He is the recipient of Rajiv Gandhi Excellence award for his academic excellence and outstanding contribution in the field of popularization of science in society.

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