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BRIEF ARTICLE

Studies on activity of various extracts of *Mentha arvensis* Linn against drug induced gastric ulcer in mammals

Ramesh L Londonkar, Pramod V Poddar

Ramesh L Londonkar, Department of Studies and Research in Biotechnology, Gulbarga University Gulbarga 585106, Karnataka, India
Pramod V Poddar, Department of Studies and Research in Biotechnology, Gulbarga University Gulbarga 585106, Karnataka, India
Author contributions: Londonkar RL and Poddar PV contributed equally to this work; Londonkar RL designed research; Poddar PV performed research, contributed new reagents/analytical tools; Londonkar RL and Poddar PV analyzed data and wrote the paper.
Correspondence to: Ramesh L Londonkar, Associate Professor, Department of Studies and Research in Biotechnology, Gulbarga University Gulbarga 585106, Karnataka, India. londonkarramesh53@reddiffmail.com
Telephone: +84-72-263290 Fax: +84-72-263206
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Abstract

AIM: To examine the antiulcerogenic effects of various extracts of *Mentha arvensis* Linn on acid, ethanol and pylorus ligated ulcer models in rats and mice.

METHODS: Various crude extracts of petroleum ether, chloroform, or aqueous at a dose of 2 g/kg po did not produce any signs or symptoms of toxicity in treated animals. In the pyloric ligation model oral administration of different extracts such as petroleum ether, chloroform and aqueous at 375 mg/kg *po*, standard drug ranitidine 60 mg/kg *po* and control group 1% Tween 80, 5 mL/kg *po* to separate groups of Wister rats of either sex (n = 6) was performed. Total acidity, ulcer number, scoring, incidence, area, and ulcer index were assessed.

RESULTS: There was a decrease in gastric secretion and ulcer index among the treated groups i.e. petroleum ether (53.4%), chloroform (59.2%), aqueous (67.0%) and in standard drug (68.7%) when compared to the negative control. In the 0.6 mol/L HCl induced ulcer model in rats (n = 6) there was a reduction in ulcerative score in animals receiving petroleum ether (50.5%), chloroform (57.4%), aqueous (67.5%) and standard. drug (71.2%) when compared to the negative control. In the case of the 90% ethanol-induced ulceration model (n = 6) in mice, there was a decrease in ulcer score in test groups of petroleum ether (53.11%), chloroform (62.9%), aqueous (65.4%) and standard drug ranitidine (69.7%) when compared to the negative control. It was found that pre-treatment with various extracts of *Mentha arvensis* Linn in three rat/mice ulcer models ie ibuprofen plus pyloric ligation, 0.6 mol/L HCl and 90% ethanol produced significant action against acid secretion (49.3 ± 0.49 *vs* 12.0 ± 0.57, $P < 0.001$). Pre-treatment with various extracts of *Mentha arvensis* Linn showed highly -significant activity against gastric ulcers (37.1 ± 0.87 *vs* 12.0 ± 0.57, $P < 0.001$).

CONCLUSION: Various extracts of *Mentha arvensis* Linn. 375 mg/kg body weight clearly shows a protective effect against acid secretion and gastric ulcers in ibuprofen plus pyloric ligation, 0.6 mol/L HCl induced and 90% ethanol-induced ulcer models.

Key words: *Mentha arvensis*; Anti ulcer; Gastro-protection; Medicinal plant

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INTRODUCTION

Ulcers are a crater-like erosion or sore that occur in the upper gastrointestinal tract of the body. Stomach ulcers are also called peptic ulcers[1]. The word peptic refers to pepsin, a stomach enzyme that breaks down protein. A peptic ulcer located in the stomach is called a gastric ulcer. An ulcer is the result of an imbalance between aggressive and defensive factors. On one hand, too much acid and pepsin can damage the stomach lining and cause ulceration. On the other hand, the damage comes first from some other cause making the stomach lining susceptible to even an ordinary level of gastric acid[2]. Peptic ulceration is a very common disease and it is estimated that approximately 10%-20% of the adult male population in western countries will experience a peptic ulcer at some stage in their lives[3]. It produces considerable pain and illness. In 1970 in the USA 3.5 million people suffered from peptic ulcer and 8600 deaths were attributed to this disease. At present nearly 15 million people are suffering from peptic ulcer diseases and 6000 deaths per year are reported across the world. According to physicians more than 90% of duodenal ulcers are caused by *Helicobacter pylori (H. pylori)* infection[4]. Long term use of NSAIDs can also cause gastric ulcer. Treatment cost is estimated more than $2 to $4 billion per year. Most of the ulcers heal by using synthetic drugs. After 6-8 wk there is a problem of recurrence of side effects. Therefore, people prefer natural product drugs for disease treatment. Over three quarters of the world population relies mainly on plants and plant extracts for health care. More than 30% of the entire plant species at one time or other, have been used for medicinal purposes.

It is estimated that the world market for plant derived drugs may account for about Rs. 200 000 crores. The annual production of medicinal and aromatic plant raw material is worth about Rs. 200 crores. This is likely to touch at US $5 trillion by 2050. WHO reported that 70% of the population of developing countries depends on natural product drugs for health care[5]. *Mentha arvensis* L. (Lamiaceaea) is distributed throughout the western Himalayas and is cultivated throughout world for use as a vegetable. It is an erect aromatic herb that grows up to 60 cm in height with suckers; the stem is cylindrical and the leaves are simple and opposing type. *Mentha arvensis* L. is used as a carminative, anti-spasmodic, anti peptic ulcer agent, and has been given to treat indigestion, skin diseases, coughs and colds in folk medicine. According to several researchers the plant contains 90% mint oil. It contains monoterpenes such as (menthone, menthofuran, methyl acetate cineole and limonene); sesquiterpenes (viridiflorol); flavonoids (luteolin, menthoside, isorhoi folin, rutin hesperidin); phenolic acids (caffeic, chlorogenic and rosmarinic); triterpenes (squalene, a-amyrin, urosolic acid; sitosterol); phytol; tocopherols; carotenoids; choline; betaine; cyclenes; rosmarinic acid; tannin; and minerals[6-8]. Hence the present study is aimed to investigate the antiulcer effect of *Mentha arvensis*.

MATERIALS AND METHODS

Animals

Healthy Swiss Albino rats and mice of the Wister strain weighing 150-200 g and 25-30 g respectively were used for the study. The animals were used with the approval of the Institute animal ethics committee and obtained from Sri Raghavendra enterprises, Bangalore. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28℃ temperature relative humidity; 60-70℃, standard light cycle (12 h light, 12 h dark) and water adlibitum (as described by CFTRI, Mysore). Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation.

Plant material and extraction

The whole herb of *Mentha arvensis* L. were collected from the fields around Gulbarga City, Karnataka in the month of February 2008 and authenticated at the department of Botany, Gulbarga University, Gulbarga. The voucher specimen was kept. The whole plants of *Mentha arvensis* were washed with tap water and shade dried at room temperature in the animal and pharmaceutical Biotechnology laboratory. After 7 d of drying the plant material ground in a pestle and mortar to obtain a fine powder. The powder was weighed and plant powder material was extracted successively using solvents ranging from non-polar to polar i.e. petroleum ether (60-70℃), chloroform (60-70℃) and aqueous (90-100℃), in a soxhlet apparatus for 18 h.

The powdered plant material (120 g) was extracted with 500 mL of petroleum ether. The filtrate gave a light green jelly syrup (3 g) with (w/w) yield of 2.5%. One hundred and twenty grams of plant material was extracted with 500 mL of chloroform. The filtrate gave a green jelly syrup (4.85 g) with (w/w) yield of 4.04%. Similarly, 120 g of powdered material was extracted with 500 mL of water. The filtrate gave a brown jelly syrup (11.5 g) with (w/w) yield of 9.58% en and was administered suspended in 1% Tween 80.

Phytochemical screening

The various extracts of *Mentha arvensis* L. were subjected to qualitative tests for preliminary phytochemical screening. This was carried out by the method described by Harborne and Kokate[9,10] to show the presence of various compounds (flavonoids, alkaloids, tannins, saponins, carbohydrates *etc*) and by Thin Layer Chromatography (TLC). TLC plates were made of silica gel G on glass 20 cm × 20 cm using a solvent system mixture composed of chloroform: methanol:n-propanol:water 5:6:1:4 v/v ratio[11]. The plates were sprayed with a specific reagent and to observe the mobile phase of constituents we used Dragendorff's reagents (alkaloids), polyethylene glycol reagent (flavonoids), 5% ferric chloride solution in methanol with 1% gelatin solution and iodine vapours (tannins), anisaldehyde - sulphuric acid reagent (saponins and triterpenes).

Drugs and chemicals

The chemical used and other solutions were all of analytical grade. All drugs and reagents were prepared immediately before use. The following drugs/chemicals were used: absolute ethanol, hydrochloric acid, solvents, ranitidine. All drugs/chemicals were purchased from Sri. Venkateshwar Chemicals, Gulbarga.

Acute toxicity studies

The method of Lorke[12] was modified to evaluate the oral acute toxicity (LD_{50}) of the various extracts of *Mentha arvensis*. The Swiss albino mice used in the study were starved of food but allowed an excess of water 24 h prior to the study. The oral administration of widely differing doses of the various extracts of *Mentha arvensis* L., (10, 100, 1000, 2000 mg/kg *po*) to four groups of mice (n = 4) was done to establish the range of doses of extracts that would produce toxic effects. This was done by observing the mice over a 72 h period post-treatment for behavioral signs such as excitement, nervousness, dullness, alertness, ataxia or even death. The adopted method estimated LD_{50} by calculating the geometric mean of the dose that caused 100% mortality and the dose which caused no lethality at all.

Induction of acute gastric mucosal damage

Antiulcerogenic activity was evaluated by using three different assay models. Ibuprofen plus pylorus-ligation, administration of 0.6 mol/L HCl and administration of ethanol in different set of rats/mice were employed for induction of acute gastric mucosal lesion.

Antiulcerogenic activity

Ibuprofen plus pyloric ligation ulcer model[13]: In this ulcer model rats weighing 150-200 g were used. They were divided into 5 groups, each group containing six rats. Group 1: This group received 1% Tween 80 5 mL/kg body weight and ibuprofen 200 mg/kg for 5 consecutive days and were considered as a negative control group. Group 2: This group received 375 mg/kg body weight petroleum ether extract and 200 mg/kg body weight ibuprofen for 5 d and were considered as test group 1. Group 3: This group received 375 mg/kg body weight chloroform extract and 200 mg/kg body weight ibuprofen for 5 d and were considered as test group 2. Group 4: This group received 375 mg/kg body weight aqueous extract and 200 mg/kg body weight ibuprofen for 5 d and considered as test group 3. Group 5: This group received the standard drug ranitidine 60 mg/kg body weight and 200 mg/kg body weight ibuprofen for 5 d and were considered as a control group.

Ulcer were induced by the NSAID ibuprofen in all groups of rats 30 min after treatment with 1% Tween 80 (negative control). Different plant extracts suspended in 1% Tween 80 and standard ranitidine were administered orally by a intragastric catheter tube for 5 d. On the 6th day fasted rats were subjected to pylorus ligation under ether anaesthesia. Water was withheld during the postoperative period. The abdomen was opened and the pyloric end of the stomach was ligated and replaced. The abdomen wall was closed by 2-3 interrupted sutures. A 6th dose of 1% Tween 80/extracts/standard drugs was given 30 min prior to pylorus ligation. After 4 h of pylorus ligation the animals were sacrificed and the stomach was isolated after suturing the lower esophageal end. Gastric juice was collected and filtered through glass wool in a measuring cylinder and the stomach was opened along the greater curvature. The gastric contents were centrifuged at 3000 rpm for 5 min, and the supernatant was used for the estimation of total acidity (pH). The volume of gastric juice was expressed as mL/100 g of body weight[14].

For estimation of total acidity[15], 1 mL of supernatant was diluted to 10 mL with distilled water. The solution was titrated against the 0.05 mL/L NaOH using phenolphthalein as an indicator[16]. Titration was continued until the color changed to light pink. The volume of NaOH required was noted and was taken as corresponding to the total acidity. Acidity was expressed as Total acidity = (Volume of NaOH × Normality × 100)/0.1 (mEgq/L). The ulcer score was determined by using a 10 × magnifying hand lens. The scoring of severity of ulceration was as follows[17,18]: 1 mm (pin point) = 1; 1-2 mm = 2; > 2 mm = 3; > 3 mm = 4. The mean ulcer score was determined by dividing the total ulcer indices in a group by the total number of animals in that group[19]. Ulcer Score = Total ulcer index (UI) in a group/Total number of animals in that group. The curative ratio of an ulcer was determined by subtracting the test ulcer score from the control ulcer score divided by the control ulcer score. The result was multiplied by 100[20]. Curative ratio = [(Control ulcer score)-(Test ulcer score)]/(Control ulcer score) × 100.

0.6 mol/L HCl induced ulcer model[21]: Thirty rats were randomly divided into 5 groups; each group contained six rats. Prior to the experiment animals were fasted for 24 h, and fed water ad libitum. Animals in group 1 were treated with 1% Tween 80 (5 mL/kg body weight) and served as untreated control. Animals in Group 2, Group 3, and Group 4 were treated with petroleum ether extract, chloroform extract and aqueous extract (375 mg/kg body weight) respectively. Group 5 animals were treated with the standard drug ranitidine (60 mg/kg body weight). All treatments were performed intragastrically *via* stomach tube. Thirty minutes after pre-treatment with 1% Tween 80, extract, ranitidine treated animals were again intubated using a stomach tube and were given 0.6 mol/L HCl *via* the stomach tube (1 mL/rat). The animals were sacrificed 4 h after the induction of ulcer with HCl in a chloroform chamber. Each animal's abdomen was opened; the stomach was exteriorized and opened through the greater curvature, rinsed under a stream of water, laid out on a flat surface and examined for the presence of mucosal lesions (on ulceration using 10 × magnifying hand lens). Ulcer scores were determined as described in Ibuprofen plus pylorus ligated rats.

Ethanol induced ulcer model[22]: Mice were divided into 5 groups; each group consisted of six animals. Group 1:

Table 1 Effect of *Mentha arvensis* on ibuprofen plus pyloric ligation ulcer model (n = 6, mean ± SE)

Group No.	Treatment	Dosage (mg/kg) B.wt	Gastric contents				% of inhibition
			Vol. of gastric juice (mL/100 g)	pH	Total acidity (mEq)	Ulcer score	
1	Control 1% Tween 80	5 mL	2.08 ± 0.05	2.01 ± 0.04	49.3 ± 0.49	37.1 ± 0.87	-
2	Petroleum ether extract	375	1.93[a] ± 0.06	2.65[b] ± 0.22	40.0[a] ± 0.57	17.3[b] ± 0.42	53.4
3	Chloroform extract	375	1.58[a] ± 0.04	2.93[a] ± 0.03	30.5[b] ± 0.56	15.1[b] ± 0.79	59.2
4	Aqueous extract	375	1.23[d] ± 0.09	3.33[b] ± 0.05	20.3[b] ± 0.33	12.0[d] ± 0.57	67.0
5	Ranitidine	60	0.96[d] ± 0.05	3.78[b] ± 0.13	11.1[b] ± 0.30	11.1[d] ± 0.30	68.7

[a]$P < 0.05$, [b]$P < 0.01$, [d]$P < 0.001$ *vs* Group 1.

This group received 1% Tween 80 (5 mL/kg body weight) orally. Group 2: this group was treated with petroleum ether extract of plant *M. arvensis* 375 mg/kg body weight. Group 3: this group was treated with chloroform extract 375 mg/kg body weight. Group 4: this group received aqueous extract at a dose of 375 mg/kg body weight. Group 5: animals received standard drug rantidine at a dose of 60 mg/kg body weight. After 30 min of treatment with 1% Tween 80, plant extracts or ranitidine, animals received 1 mL of 90% ethanol *via* an intragastric catheter tube. After 4 h of induction animals were scarified by excess anesthesia. A ventral medline incision was made on each animal and the stomach exteriorized, opened through the greater curvature, rinsed, laid out on a flat surface and examined for the presence of mucosal lesions by 10 × magnifying lens. Ulcer score and curative ratio were determined as described in previous ulcer models.

Statistical analysis

All values were expressed as mean ± SE. Statistical analyses of data were performed using a one-way analysis of variance (ANOVA). A value of $P < 0.005$ was considered statistically significant.

RESULTS

As part of this pharmacological study, the various extracts of *M. arvensis* were first investigated for toxicity in mice. A single oral dose of 2 g/kg of each different plant extract of *M. arvensis* did not produce any signs or symptoms of toxicity in treated animals. Seventy-two hours after administration, no animal died and no significant macroscopic changes occurred. This result probably indicates that the plant extract has no acute toxicity in mice. For this reason, a 5-fold lower dose was used as the maximum dose in all experiments to determine the general antiulcer profile of various extracts of the plant *M. arvensis*.

Effect on ibuprofen plus pyloric ligated ulcer model

The results are depicted in Table 1, which shows a decrease in ulcer score, volume of acid secretion, total acidity and pH in various extracts of *Mentha arvensis* i.e. aqueous extract, petroleum ether extract and chloroform extract. There was also a significant reduction in these parameters in the ranitidine treated ulcer group when compared to the negative control group.

In the group of animals in which ulcers were induced using ibuprofen and pylorus ligation, the aqueous extract showed significant activity in all the selected parameters with 67% inhibition of ulcers and a significant reduction in total acidity, ulcer score and gastric secretion ($P < 0.001$). Standard drug treatment with ranitidine (60 mg/kg) also showed significant reductions in acidity, gastric secretion and ulcer score with a curative ratio of 68.7% when compared to negative control group. The petroleum ether extract and chloroform extract produced curative ratios of 53.4% and 59.2%, respectively (Table 1).

Effect on 0.6 mol/L HCl induced ulcer model

The ulcer indices in rats treated with ranitidine and aqueous extract of *Mentha arvensis* were found to be significantly ($P < 0.001$) lower than that of control treated rats with a curative ratio of 71.2% and 67.5%, respectively. In addition rats that received the chloroform and petroleum ether extracts showed reductions in ulcer indices with a curative ratio of 57.4% and 50.5% respectively compared with 1% Tween 80 treated control rats (Table 2).

Effect on ethanol induced ulcer model

The aqueous extract of *Mentha arvensis* and ranitidine significantly increased the macroscopic curative ratio to 65.4% and 69.75%, respectively, compared to control groups. The ulcer index was significantly reduced in mice pretreated with chloroform and petroleum ether extract, with a curative ratio 62.9% and 53.11%, respectively, compared to the negative control group (Table 3).

DISCUSSION

Peptic ulcer disease is a chronic inflammatory disease characterized by ulceration in the upper gastro-intestinal tract[23]. The pathophysiology of ulcers is due to an imbalance between aggressive factors (acid, pepsin, *H. pylori* and NSAIDs) and local mucosal defensive factors (mucus bicarbonate, blood flow and prostaglandins). The integrity of the gastroduodenal mucosa is maintained through a hemostatic balance between these aggressive and defensive factors. The major cause of gastric ulcer is the chronic use of NSAIDs. Therapeutic and adverse effects of NSAIDs have been attributed to the ability of these drugs to inhibit the action of cyclooxygenase (COX). COX is responsible for the synthesis of prostaglandins

Table 2 Effect of *Mentha arvensis* on 0.6 mol/L HCl induced ulcer model (n = 6, mean ± SE)

Group No.	Treatment	Dosage (mg/kg) B. wt	Ulcer score	% of inhibition
1	Control 1% Tween 80	5 mL	31.3 ± 0.42	-
2	Petroleum ether extract	375	15.5[a] ± 0.50	50.5
3	Chloroform extract	375	13.3[a] ± 0.42	57.4
4	Aqueous extract	375	10.1[b] ± 0.47	67.5
5	Ranitidine	60	9.0[b] ± 0.44	71.2

[a]P < 0.05, [b]P < 0.001 *vs* Group 1.

Table 3 Effect of *Mentha arvensis* on 90% Ethanol induced ulcer model (n = 6, mean ± SE)

Group No.	Treatment	Dosage (mg/kg) B.wt	Ulcer score	% of inhibition
1	Control 1% Tween 80	5 mL	27.0 ± 0.36	-
2	Petroleum ether extract	375	12.6[a] ± 0.95	53.1
3	Chloroform extract	375	10.0[b] ± 0.68	62.9
4	Aqueous extract	375	9.33[d] ± 0.76	65.4
5	Ranitidine	60	8.16[d] ± 0.47	69.7

[a]P < 0.05, [b]P < 0.01, [d]P < 0.001 *vs* Group 1.

that normally inhibit acid secretion, as well as having a protective effect on the gastric mucosa. Infection of the stomach mucosa with *H. pylori* - a gram-negative spiral shaped bacterium - is now generally considered to be a major cause of gastrointestinal ulcers. Treatment includes H2-receptor antagonists (cimetidine), proton pump inhibitors (omeprazole) and cytoprotectives (misoprostol). Antacids, like aluminum hydroxide and magnesium hydroxide, are used often to neutralize excess gastric acidity in the stomach. Due to problems associated with recurrence after treatment, there is the need to seek an alternative drug against gastrointestinal ulcers[24].

The present investigation demonstrated the efficacy of *Mentha arvensis* plant extract against gastric ulceration induced by 3 experimental models viz., ibuprofen induced gastric ulceration, 0.6 mol/L HCl induced ulceration and 90% ethanol induced ulceration. The plant extract *Mentha arvensis* and standard drugs produces a decrease in the ulcer number, total acidity, volume of gastric juice and pH in the ibuprofen induced pyloric ligation ulcer model in rats. The curative ratio in this pyloric ligation model was 53.4%, 59.2%, 67% and 68.7% using petroleum ether, chloroform, aqueous extract and standard drug ranitidine, respectively. This indicates that the plant has antiulcerogenic, antisecretory and cytoprotective actions. Several investigators have reported the same results after plant extract treatment. Gastric mucus is known to protect the gastric mucosa against tissue damage by HCl produced by parietal cells. It consists of viscous, elastic, adherent and transparent gel formed by 95% water and 5% glycoproteins that covers the entire gastrointestinal mucosa. Moreover, mucus is capable of acting as an antioxidant thus can reduce mucosal damage mediated by oxygen free radicals. The protective properties of the mucus barrier depend not only on gel structures but also on the thickness of the layer covering the mucosal layer[25]. A decrease in gastric mucus renders the mucosa susceptible to injuries induced mainly by acids, NSAIDs and alcohol.

The effect of Mentha aervensis extracts on the mucosal damage in the 0.6 mol/L HCl induced gastric ulcer model in rats reveals the decreases in ulcer scores. Treatment with successive extracts and standard drug shows the decreases i.e. petroleum ether (50.5%), chloroform (57.4%), aqueous (67.5 %) and ranitidine (71.2%). This indicates that the extracts have cytoprotective effects against the irritant actions caused by acids. Studies of plant extract *Tephrosia* purpurea treated ulcerogenic rats have also shown cytoprotective activity[26].

Peptic ulcer is an imbalance between gastroduodenal mucosal defense mechanisms and offensive factors. Some studies have revealed that reactive oxygen species (ROS) and lipid peroxidation are implicated in the pathogenesis of ethanol induced gastric lesions and gastrointestinal damage and that they attack and damage many biological molecules such as prostaglandins. After an initial reaction with ROS, a continuing chain reaction causes cell injury and ultimately cell death[27-31]. Therefore, treatment with antioxidants and free radical scavengers can decrease ethanol induced gastric mucosal damage. In the present study, a reduction in ulcer number in ethanol induced gastric ulceration in mice was found after various extract treatments, such as petroleum ether (53.1%), chloroform (62.9%), aqueous extract (65.4%) of *Mentha arvensis* and the standard drug ranitidine (69.7%). This indicates cytoprotective actions in the plant extracts. Plant chemical substances such as flavonoids, tannins, terpenoids *etc* have been shown to scavenger free radicals and therefore are viewed as promising therapeutic drugs for free radical pathologies. Phytochemical tests revealed the presence of flavonoids and terpenoids in the extracts of *Mentha arvensis*. Some of the triterpenes are known as an antiulcer agents and their action has been mentioned to be due to activation of cellular proteins, reduction of mucosal prostaglandin metabolism, cytoprotective actions and reduction of gastric vascular permeability[32-34]. However, the mechanism by which this extract produces an antiulcer effect is not entirely clear. The results in present study seems to provide support for the use of *Mentha arvensis* as an antiulcer drug in folk medicine. Therefore, also in view of its large use in India more detailed phytochemical and pharmacological investigations on the antiulcer effects and toxicity studies are required. In all three ulcer experimental models the aqueous extract shows the best antiulcerogenic action, due to the presence of tannins and flavonoids, as in literature references.

The present data obtained from various extracts

of *Mentha arvensis* L. showed the presence of a gastro-protective effect and improved ulcer healing properties. The data also confirmed the traditional claim on the use of *M. arvensis* for treating gastric ulcers in the Indian subcontinent. Although at this time it is difficult to explain the exact mechanism involved with these crude extracts, the effects obtained on acute and chronic gastric lesions suggest a multifactorial mechanism, involving *M. arvensis* influence on free-radical scavenging properties, on endogenous prostaglandins and sulphydryl groups.

COMMENTS

Background

The stomach defends itself from hydrochloric acid and pepsin by creating a mucus coating (that shields stomach tissue), by producing bicarbonate and by circulating blood to the stomach lining to aid in cell renewal and repair. When these functions are impaired it can lead to the formation of an ulcer. Mentha arvensis has plenty of medicinal property which contains corn mint used for the cure of stomach cancer and peptic ulcer.

Research frontiers

Mentha arvensis is a carminative and antispasmodic herb has excellent good results in the treatment of indigestion, biliousness, flatulent colic, *etc*. the present study is used for the prevention of the peptic ulcer with mentha arvensis, the present research shows the efficiency of the menthe arvensis for the treatment of the peptic ulcer.

Innovations and breakthroughs

Earlier the mentha arvensis has a carminative, cholatotic, secretolytic and cooling properties. Mint oil has antimicrobial activity and contain corn mint which is effectively used for the treatment of stomach cancer. The present investigation is aimed to study the antiulcer activity of mentha arvensis. In this research three solvent's extract were used such as petroleum ether, chloroform and aqueous. Among all the three extract, aqueous extract has shown/given good result for the cure of ulcer when it compared with the standard drug available in the market.

Applications

Pyloric ligation ulcer model in rats. The results of the present article suggest that the plant mentha arvensis has a bioactive compounds which can dissolve in solvents like petroleum ether, chloroform and aqueous which can be used in the preparation of drugs for the treatment of intestinal ulcer.

Terminology

The intestine has mucus coating, a sort of defensive /protective coverage against intestinal enzymes, Mentha arvensis has flavanoids, alkaloids, and terpenoids which will acts as gastro-protective agent's. These compounds are more potent than the standard drug available in the market.

Peer review

The present investigation on bioactive compounds of mentha arvensis plant in which authors have analyzed the preventive property of peptic ulcer by using secondary metabolites of the plant. The results reveals that there is significant efficacy of mentha arvensis plant extract against gastric ulceration induced by three experimental model viz Ibuprofen induced gastric ulceration, 0.6 mol/L HCl and 90% ethanol induced ulceration. Various extracts of mentha arvensis plant and standard drug both have shown decrease in the ulceration, total acidity, volume of gastric juice and pH in Ibuprofen induced pyloric ligation ulcer model in rat. Therefore, This study helps to use phyto compounds as novel. Drug for the treatment of gastric ulcer.

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