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RESEARCH ARTICLE

Gastric antiulcer, antisecretory and cytoprotective properties of celery (*Apium graveolens*) in rats

Tawfeq Al-Howiriny[1], Abdulmalik Alsheikh[2], Saleh Alqasoumi[1], Mohammed Al-Yahya[1], Kamal ElTahir[3], and Syed Rafatullah[4]

[1]Departments of Pharmacognosy, College of Pharmacy, King Saud University, Riyadh, Saudi Arabia, [2]Department of Pathology, King Khalid University Hospital, King Saud University, Riyadh, Saudi Arabia, [3]Departments of Pharmacology, College of Pharmacy, King Saud University, Riyadh, Saudi Arabia, and [4]Medicinal, Aromatic and Poisonous Plants Research Center (MAPPRC), College of Pharmacy, King Saud University, Riyadh, Saudi Arabia

Abstract

In the present investigation, an ethanol extract of celery [*Apium graveolens* L. (Apiaceae/Umbelliferae)], at doses of 250 and 500 mg/kg body weight, was evaluated for antigastric ulcer activity using various experimental gastric ulcer models in rats. Ulcers were induced by indomethacin, cytodestructive agents (80% ethanol, 0.2 M NaOH and 25% NaCl) and cold restraint stress. Gastric secretory studies were undertaken by using pylorus ligation (Shay rat model). In addition to gastric wall mucus (GWM), non-protein sulfhydryl (NP-SH) and malondialdehyde (MDA) were also estimated in gastric tissues after 80% ethanol treatment. Pretreatment of celery extract produced dose-dependent reduction in all experimentally induced gastric lesions. Ethanol (80%) decreased the levels of GWM, NP-SH and increase in MDA concentration in gastric tissue. Celery extract showed the ability to significantly replenish the ethanol-induced depleted levels of GWM and gastric mucosal NP-SH. The gastric mucosal MDA level was also significantly lowered in extract pretreated rats. The celery extract showed stomach protection against the models used for ulcerogenesis. Results were further confirmed by using histopathological assessment. The phytochemical screening showed the presence of various chemical constituents such as flavonoids, tannins, volatile oils, alkaloids, sterols and/or triterpenes. Acute toxicity test revealed no deleterious or toxic symptoms or mortality over a period of 14 days. However, the LD_{50} was found to be 7.55 g/kg, and showed a large margin of safety. The results suggest that *Apium graveolens* extract significantly protects the gastric mucosa and suppresses the basal gastric secretion in rats, possibly through its antioxidant potential.

Keywords: Apium graveolens*; Arab traditional medicine; celery; cytoprotection; gastric mucus; gastric secretion; gastroprotection; lipid peroxidation*

Introduction

Celery, *Apium graveolens* L. (Apiaceae/Umbelliferae), is locally known as "karfas" or "ajmod". It is an aromatic biennial herb, almost the whole plant is used, including the roots, seeds, leaves, and oil. It is a bitter herb with a pleasant smell that relieves indigestion, reduces inflammation, and acts as a mild diuretic (Newall et al., 1996). Kamal (1975) has stated that celery is an aphrodisiac, emmenagogue, and carminative used by ancient Greco-Arab physicians; it is still used by Unani and Ayurvedic medical practitioners for stomach and kidney disorders. The whole plant is gently stimulant, nourishing, and restorative for weak conditions. The leaves and stalk of celery share the same medicinal properties of other parts of the plant. Eating fresh stalks can help stimulate milk flow, while seeds are mainly used as a diuretic, which help clear toxins from the system, so are especially good for gout and other joint diseases (Ody, 1993). Celery is known to possess antiflatulent and antispasmodic properties.

Address for Correspondence: Dr. Syed Rafatullah, MD(U); MFHom. Medicinal, Aromatic and Poisonous Plants Research Center, College of Pharmacy, King Saud University, P.O. Box 2457, Riyadh 11451, Saudi Arabia. Tel.: (+966) 1-46-77207; Fax: (+966) 1-46-6383; E-mail: srafat@ksu.edu.sa; srafatullah@yahoo.com

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It is used in bronchitis, asthma, and to some extent for liver and spleen diseases; it is also used as an antispasmodic and as an ingredient in salad (Kapoor, 1990).

Celery seed is generally regarded as safe (GRAS) in the US for human consumption as a spice, natural seasoning and plant extract/essential oil (Jellin et al., 2000). In Germany, celery preparations are used to treat loss of appetite, general and nervous exhaustion (Wren, 1988). Some pharmacological effects of celery have been reported, such as vasodilatory action in rat thoracic aorta (Ko et al., 1991) and mosquito repellent (Tuetun et al., 2005). Sultana et al. (2005) reported that celery is a potent plant against experimentally induced hepatocarcinogenesis in Wistar rats. The present study assessed the possible antigastric ulcer, cytoprotective, and antisecretory properties of the ethanol extract of *Apium graveolens* in rats in order to substantiate the traditional Unani, Arab, and Ayurvedic medicine practitioners' claim of its use in stomach disorders.

Materials and methods

Plant material and extraction

The aerial parts of fresh celery used in this study were purchased from the local vegetable market of Riyadh, and identified by an expert taxonomist, Atiqur Rahman. A voucher specimen (77262) was deposited in the Medicinal, Aromatic and Poisonous Plants Research Center of this college for future reference.

The shade-dried aerial parts (500 g) of celery were coarse powdered and macerated in 3 L of 96% ethanol for 72 h using the percolation method. The solvent was then removed at 40°C under reduced pressure in a Rotavapor (yield 5.2%). The extract was suspended in distilled water before administration.

Animal stock

Wistar albino rats of either sex (home bred), aged 7–8 weeks and weighing 150–200 g, were obtained from the Experimental Animal Care Center, King Saud University, Riyadh, Saudi Arabia. The animals were fed Purina chow diet and water ad libitum and were maintained under standard conditions of humidity (55% ± 5%), temperature (22° ± 2°C) and light (12 h light/12 h dark cycle). The rats were randomly assigned to different control and treatment groups each containing six animals. The conduct of experiments and the procedure of sacrifice (using ether) were approved by the Ethics Committee of the Experimental Animal Care Society, College of Pharmacy, King Saud University, Riyadh, Saudi Arabia.

The ulcerogenic drugs and necrotizing agents were freshly prepared before administration. The stomach was removed, opened along the greater curvature, washed with saline and the inner surface was examined with a 6.4 × binocular magnifier. Lesions were also assessed by two observers unaware of experimental protocol.

Gastric lesions induced by the drugs used in this study were multiple in each stomach. They were evaluated singly according to their dimensions and severity and scored between 0 (no visible ulcers) and 10 (deep lesion with diameter greater than 8 mm) in each stomach. The scores for each single lesion were then totaled (Valcavi et al., 1982).

Indomethacin-induced gastric ulcers

Indomethacin was suspended in 1% carboxymethylcellulose in water (6 mg/mL) and administered p.o. at a dose of 30 mg/kg (0.5 mL/100 g) to rats fasted for 36 h (Bhargava et al., 1973). Celery extract was administered orally (250 and 500 mg/kg body weight) 30 min before indomethacin. The rats were killed 6 h after indomethacin administration.

Gastric lesions induced by necrotizing agents (cytoprotection studies)

The experiments were done using Wistar male rats fasted for 36 h with access to drinking water ad libitum. The following necrotizing agents were administered orally in a volume of 1 mL: 80% ethanol, 0.2 M NaOH and 25% NaCl (Robert et al., 1983). The celery extract in doses of (250 and 500 mg/kg body weight) was administered orally 30 min before the necrotizing agents treatment.

Hypothermic restraint stress-induced ulcers

The method of Levine (1971) was followed. The animals were fasted for 36 h immobilized in restraint cages and placed inside a ventilated cold room, maintained at a temperature of 3° ± 1°C for 3 h. The rats were taken out of the cold room and sacrificed. Their stomachs were removed and examined for the severity of intraluminal bleeding according to the following arbitrary scale (Chiu et al., 1984): 0, no blood detectable; 1, thin blood follows the rugae; 2, thick blood follows the rugae; 3, thick blood follows the rugae with blood clots in certain areas; 4, extensive covering of the whole gastric mucosal surface with thick blood. After wiping off the blood with water, the ulcers in each stomach were scored as described in the protocol.

Antisecretory study

Gastric antisecretory activity was evaluated in rats according to Shay et al. (1945). The animals were deprived of food for 36 h with access to water ad libitum.

Under light ether anesthesia, a small midline abdominal incision was made and the pylorus ligated. The wound was closed using sterile suture. The plant extract or normal saline was administered intraperitoneally immediately after pylorus ligation. Six h after pylorus ligation the animals were sacrificed by cervical displacement. The stomachs were removed from both groups (treated and control) and the gastric contents collected and centrifuged. The volume of the supernatant was measured and titratable acidity of liquid gastric content was recorded in milliequivalents per liter (mEq/L) and the titratable acidity was calculated. Each stomach was scored for ulcers as described in the protocol.

Determination of gastric wall mucus (GWM)

Gastric wall mucus was determined according to the procedure of Corne et al. (1974). The glandular part of the stomach (0.5 g) was placed in 10 mL 1% Alcian blue solution in 0.16 M sodium acetate (pH 5.8) and left to stain for 2 h. The dye complex was extracted with 0.5 M $MgCl_2$ solution, centrifuged and measured spectrophotometrically at 580 nm using a standard curve of Alcian blue.

Estimation of non-protein sulfhydryl groups (NP–SH)

Gastric mucosal NP-SH was measured according to the method of Sedlak and Lindsay (1968). The glandular part of the stomach was homogenized in ice-cold 0.02 M ethylene diamine tetra-acetic acid (EDTA). Aliquots of 5 mL of the homogenates were mixed in 15 mL test tubes with 4 mL distilled water and 1 mL 50% trichloroacetic acid. The tubes were shaken intermittently for 10-15 min and centrifuged at 3000 *g*. An aliquot of 2 mL supernatant was mixed with 4 mL 0.4 M Tris buffer, pH 8.9 and 0.1 mL 0.4% DTNB [5,5-dithio-bis-(2-nitrobenzoic acid)] was added and the sample was shaken. The absorbance was read within 5 min of addition of DTNB at 412 nm against a reagent blank with no homogenate.

Determination of malondialdehyde

The method reported by Utley et al. (1967) was followed. The animals were killed 1 h after ethanol administration. The stomachs were removed and each tissue was homogenized in 0.15 M KCl (at 4°C, Potter-Elvehjem type C homogenizer) to give a 10% w/v homogenate. Aliquots of homogenate 1 mL in volume were incubated at 37°C for 3 h in a metabolic shaker. Then 1 mL of 10% aqueous trichloroacetic acid was added and mixed. The mixture was then centrifuged at 800 g for 10 min. An aliquot of 1 mL of the supernatant was removed and mixed with 1 mL of 0.67% thiobarbituric acid in water and placed in a boiling water bath for 10 min. The mixture was cooled and diluted with 1 mL of distilled water. The absorbance of the solution was then read at 535 nm. The content of malondialdehyde (nmole/g wet tissue) (index of the magnitude of lipid peroxidation) was then calculated, by reference to a standard curve of malondialdehyde solution.

Histopathological studies

The gastric tissue was fixed in 10% ethanol buffered formalin and processed through graded ethanol, xylene and impregnated with paraffin wax; sections were made by microtome. After staining with hemotoxylin and eosin stain (Culling, 1974), the sections were examined under a research microscope by a person who was not aware of experimental protocols. The different histopathological indices were screened.

Phytochemical screening

A preliminary phytochemical screening of the aerial parts of celery was conducted to determine the presence or absence of alkaloids, cardiac glycosides, flavonoids, tannins, coumarins, anthraquinones, saponins, volatile oil, volatile bases, cyanogenic glycosides, glucosinolates and sugars, according to the methods described by Farnsworth (1966).

Determination of acute toxicity and LD_{50} in mice

Swiss albino mice were divided into various groups (n = 10 animals per group). Each animal in each group was orally treated with a single dose of celery extract in the dose range 0.25g-12 g per kg. Following treatments, the animals were observed for 6 continuous h and thereafter at intervals of 12 h for up to 72 h. All behavioral changes and mortality during the observation period were recorded. The percentage of death in each group was then calculated. The LD_{50} was then determined using the methods outlined by Paget (1981) and Ghosh (1984).

Statistical analysis

The readings showed are mean ± standard error of mean. The means of treatment and control groups were statistically compared by using ANOVA, followed by Tukey-Kramer post-hoc test.

Results

Effect on indomethacin-induced gastric ulcers

Celery extract produced a dose-dependent significant protection against the ulcerogenic effect induced by indomethacin (Table 1).

Effect on necrotizing agents-induced gastric lesions

In the ethanol and strong alkali-induced ulcer protocol, it was observed that the treatment with ethanol extract of celery (250 and 500 mg/kg) significantly reduced the lesion index. Although, the ulcer intensity was found to be reduced in the animal groups that received (250 mg/kg) dose of extract in ethanol- and NaOH-induced mucosal damage, but this reduction of ulceration was statistically insignificant (Table 2).

Effect on hypothermic restraint stress ulcers

A highly significant reduction of ulceration in rats' stomachs and intraluminal bleeding was recorded after celery extract pretreatment at the dose of 500 mg/kg orally (Table 3).

Effect on pyloric ligation-induced gastric ulcers

In the gastric secretion determination model, using ligated pylorus for 6 h, the treatment with celery extract (250 and 500 mg/kg, i.p.), reduced the volume of basal gastric secretion, titratable acidity and ulceration significantly in comparison with the control group (Table 4).

Table 1. Effect of ethanol extract of celery on gastric lesion induced by indomethacin.

Treatment	Dose (mg/kg, orally)	Ulcer index
Control (indomethacin only)	30	40.0 ± 5.62
Celery extract + indomethacin	250 + 30	20.16 ± 4.35*
Celery extract + indomethacin	500 + 30	15.33 ± 3.93**

Values are expressed as mean ± SEM (n = 6); *p < 0.05, **p < 0.01 ANOVA followed by Tukey and Kramer post-hoc test.

Table 2. Effect of ethanol extract of celery on gastric lesion induced by various necrotizing agents.

Treatment	Dose (mg/kg, orally)	Ulcer index		
		80% EtOH	0.2 M NaOH	25% NaCl
Control	–	7.83 ± 0.4	7.66 ± 0.51	7.16 ± 0.75
Celery extract	250	5.66 ± 1.21	5.5 ± 1.04	3.5 ± 0.54**
Celery extract	500	4.5 ± 1.2*	4.0 ± 0.63***	2.16 ± 0.98**

Values are expressed as mean ± SEM (n = 6); *p < 0.05, **p < 0.01, ***p < 0.001 ANOVA followed by Tukey and Kramer post-hoc test.

Effect on gastric wall mucus

An aliquot of 1 mL 80% ethanol significantly decreased the gastric wall mucus (GWM) secretion of rats from 375.88 ± 51.13 to 194.22 ± 21.92 µg/g (wet glandular tissue). Acute oral pretreatment with celery extract significantly restored the lowered value of gastric wall content of mucus at both doses (250 and 500 mg/kg) in ethanol-induced depletion of gastric wall mucus from 194.22 ± 21.92 to 333.08 ± 38.54 and 354.66 ± 20.38 µg/g wet tissue (P < 0.01; P < 0.001). These results are presented in Table 5.

Estimation of NP-SH in gastric tissue

Ethanol 80% induced a significant decrease in gastric mucosal NP-SH level. Prior treatment of animals with ethanol extract of celery significantly replenished the depleted gastric NP-SH contents in both dose groups (Table 5).

Determination of MDA in gastric tissue

As tabulated in Table 6, MDA levels in the gastric mucosa used as an index of lipid peroxidation were significantly higher in the ethanol only treated group than the control group. On the other hand, celery extract decreased significantly (at 500 mg/kg dose) the MDA content; the lower dose (250 mg/kg), however, decreased the MDA content, but insignificantly.

Phytochemical screening

The preliminary qualitative phytochemical screening revealed the presence of volatile oils, alkaloids (positive reaction by spraying Dragendorff's reagent), flavonoids, saponins, tannins, coumarins, sterols, and/or triterpenes.

Table 3. Effect of ethanol extract of celery on hypothermic restraint stress induced intraluminal bleeding and gastric lesions in rats.

Treatment	Dose (mg/kg, orally)	Intraluminal bleeding	Gastric lesion
		Score	Ulcer index
Control	–	2.833 ± 0.75	32.16 ± 4.26
Celery extract	250	1.5 ± 0.54	16.66 ± 8.09
Celery extract	500	0.33 ± 0.51*	4.33 ± 6.12**

Values are expressed as mean ± SEM (n = 6); *p < 0.05, **p < 0.01 ANOVA followed by Tukey and Kramer post-hoc test.

Table 4. Effect of ethanol extract of celery on the gastric secretion, titratable acidity and lesion in 6 h pylorus ligated Shay rats.

Treatment	Dose (mg/kg, i.p.)	Gastric secretion at 6 h		
		Volume of gastric secretion (mL)	Titratable acidity (MEq/L)	Ulcer index
Control	–	11.66 ± 0.81	133.22 ± 4.9	2.83 ± 0.75
Celery extract	250	6.16 ± 0.75***	91.66 ± 5.47***	1.33 ± 0.81
Celery extract	500	4.66 ± 0.81***	62.22 ± 8.6***	0.5 ± 0.54*

Values are expressed as mean ± SEM (n = 6); *p < 0.05, ***p < 0.001, ANOVA followed by Tukey and Kramer post-hoc test.

Table 5. Effect of ethanol extract of celery on 80% ethanol-induced GWM, and levels of non-protein sulfhydryls (NP-SH) in rats.

Treatment	Dose (mg/kg, orally)	GWM (μg/g of wet glandular stomach tissue)	NP-SH (μmol/g of wet glandular stomach tissue)
Control	–	375.88 ± 51.13	5.02 ± 0.45
80% Ethanol only	–	194.22 ± 21.92[a]**	2.23 ± 0.41[a]***
Celery extract + 80% ethanol	250	333.08 ± 38.54[b]**	3.34 ± 0.17[b]*
Celery extract + 80% ethanol	500	354.66 ± 20.38[b]***	3.81 ± 0.18[b]**

Values are expressed as mean ± SEM (n = 6); *p < 0.05, **p < 0.01, ***p < 0.001 ANOVA followed by Tukey and Kramer post-hoc test.
[a]As compared with control group.
[b]As compared with 80% ethanol only treated group.

Table 6. Effect of ethanol extract of celery on the lipid peroxidation (MDA) level in the rat treated with 80% ethanol.

Treatment	Dose (mg/kg, orally)	MDA (μmol/g of tissue)	% Decrease
Control	–	1.44 ± 0.31	–
80% Ethanol only	–	4.54 ± 0.74[a]**	0
Celery extract + 80% ethanol	250	3.41 ± 0.4[b]	24.88
Celery extract + 80% ethanol	500	2.32 ± 0.52[b]*	48.89

Values are expressed as mean ± SEM (n = 6); *p < 0.05, **p<0.01 ANOVA followed by Tukey and Kramer post-hoc test.
[a]As compared with control group.
[b]As compared with 80% ethanol only treated group.

Determination of acute toxicity and LD_{50} in mice

No toxicity symptoms were observed. However, LD_{50} dose was found to be 7.55 g/kg, and showed a large margin of safety.

Histopathological studies

The histopathological assessment of gastric tissue substantiates the pharmacological and biochemical findings. Figure 1A shows the normal gastric mucosa. The oral treatment of 80% ethanol alone caused mucosal ulceration and hemorrhage (Figure 1B). Pretreatment of rats with celery extract in both doses (250 and 500 mg/kg) showed absence of mucosal ulceration and hemorrhage, as depicted in Figures 1C and 1D, respectively.

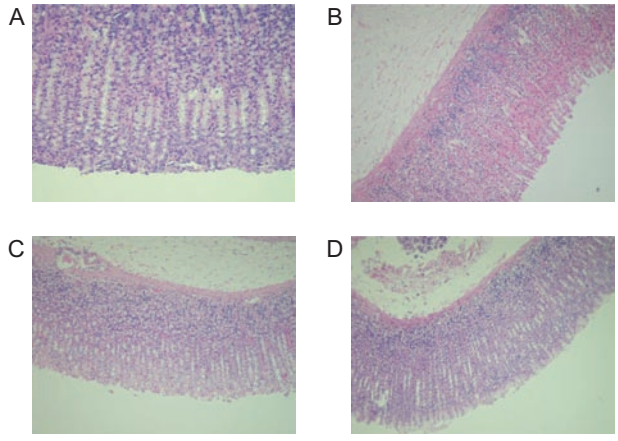

Figure 1. Light micrographs showing the effect of celery extract on ethanol- induced gastric lesions of rats. A: Normal mucosa; B: Ethanol-induced gastric mucosal congestion and necrosis; C: Pretreatment of rats with celery extract 250 mg/kg; D: Pretreatment of rats with celery extract 500 mg/kg.

Discussion

This study revealed a significant anti-ulcer effect of ethanol extract of *Apium graveolens* in experimental models of gastric lesions induced by NSAID indomethacin, various necrotizing agents, hypothermic restraint stress and pylorus ligated Shay rats.

It is well known that ulcers result from an imbalance of the interactive process of aggressive and defensive factors of the stomach (Bandyopadhyay et al., 2000). The primary pathology of NSAID-induced acute gastric mucosal damage is likely to be mucosal lesions due to inhibition of cyclooxygenase that prevents prostaglandin biosynthesis which in turn inhibits the release of mucus (Kauffman, 1989; Hudson et al., 1992), and reduction in gastric mucosal damage is likely to be mucosal lesions due to ischemia. The main mechanism by which NSAIDs reduce gastric mucosal blood flow is thought to be inhibition of COX–1 (Funatsu et al., 2007). This inhibition leads to a deficiency in endogenous prostaglandin E_2 and PGI_2 which in turn cause microcirculatory disturbances in the gastric mucosa (Shorrock & Rees, 1989). The mechanisms involved in prostaglandin action are multiple, including stimulation of mucus and bicarbonate output, enhancement of gastric mucosal blood flow, decreasing gastric motility, and stimulation of cellular growth and repair (Goulart et al., 2005). Our observations that the celery extract prevented gastric lesions induced by indomethacin, may be explained by the ability of celery extract to generate endogenous prostaglandins in stomach tissue.

In the present study, oral administration of celery extract inhibited the gastric damage caused by ethanol and strong alkali, the most commonly employed tests in the evaluation of anti-ulcer/cytoprotective activity (Oliveira et al., 2004). It is suggested that oxygen radicals may contribute to the induction of ethanol and other necrotizing agents-induced gastric mucosal lesions (Trier et al., 1987; Matsumoto et al., 1992) and anti-oxidants are protective against the damage caused by oxidants (Mizui et al., 1987; Farina et al., 1998). Ethanol has been shown to deplete the level of non-protein sulfhydryl content in stomach tissues and its restoration appears to be important in gastroprotection since they provide a substrate for free radicals to replenish GSH stores (Trier et al., 1987). The celery extract in the present investigation significantly restored the NP-SH contents of rat stomach. Therefore, it is suggested that celery extract prevention of the ethanol-induced gastric damage may at least, in part, be due to its anti-oxidant mechanism. The chemical constituents of celery responsible for antiulcer activity are not known. However, many naturally occurring flavonoids and tannins have been shown to possess anti-gastric ulcer activity (Islam et al., 2002). The preliminary qualitative phytochemical evaluation of celery showed the presence of flavonoids, tannins, volatile oils, saponins, alkaloids, sterols and/or triterpenes (Al-Howiriny et al., 2003). The antioxidant properties of flavonoids and tannins have been related to antiulcer activity (Hodek et al., 2002) since free radicals are developed in gastric mucosal lesions (Pihan et al., 1987). Flavonoids and tannins have shown cytoprotective activity in various models (Al-Rehaily et al., 2002; Al-Howiriny et al., 2003). Some authors (Rao et al., 1976; Sairam et al., 2001) have reported an antiulcer effect for saponins.

Celery extract significantly reduced the intensity of gastric ulceration induced by hypothermic restraint-stress. Gastric ulceration induced by stress is probably mediated by histamine release and vagal over-activity (Grijalva & Novin, 1990) with enhancement in acid secretion and a reduction in mucus production (Senay & Levine, 1967).

Moreover, gastric acid is an important factor for the genesis of ulceration in pylorus-ligated rats (Shay et al., 1945). Vagal activation by stimulation of pressure receptors in the antral gastric mucosa in the hypersecretion model of pylorus ligature is believed to increase gastric acid secretion (Baggio et al., 2003). The current data clearly demonstrated that celery extract not only reduced basal gastric acid secretory volume but also the titratable acidity and ulceration. Gastric wall mucus is thought to play an important role as a defensive factor against gastrointestinal damage (Marhuenda et al., 1993). Gastric wall mucus was used as an indicator for gastric wall mucus secretion (Mersereau & Hinchey, 1982). It was observed in the present investigation that celery extract has caused a significant elevation of gastric wall mucus that has been depleted by 80% ethanol in rats. This further confirms the capacity of celery to prevent and/or ameliorate the effects of damaging agents. This finding indicates that celery extract preserves gastric mucus secretion and strengthens the defense factors of gastric mucosa in experimental rats (Davenport, 1968; Guth, 1972).

Our results showed a significant reduction in NP-SH content of gastric mucosa after ethanol administration. Sulfhydryl compounds have been significantly implicated in maintenance of gastric integrity, particularly when reactive oxygen species are involved in the pathophysiology of tissue damage (Kimura et al., 2001; Natale et al., 2004). Pretreatment of rats with celery extract significantly prevented NP-SH depletion. Non-protein sulfhydryls are known to be involved in protecting gastric mucosa against various noxious chemicals (Szabo et al., 1981). On the other hand, celery extract exhibited a marked reduction of lipid peroxidation induced by ethanol administration. It significantly decreased malondialdehyde concentration in the rat stomach tissue. MDA is one of the end products resulting from peroxidation of polyunsaturated fatty acids and related esters within cell membranes, and measurement of this substance represents a suitable index of lipid peroxidation (Kwicien et al., 2002). Recent reports indicate that lipid peroxidation was prevented by flavonoids (Kahraman et al., 2003; Alqasoumi et al., 2008).

In order to confirm the results of the *in vivo* experiment, the stomachs were also evaluated by histopathological means. In histological examination, the gastric mucosa of rats revealed that the ethanol treatment caused hemorrhagic necrosis. Celery extract pretreatment exerted no ulceration or hemorrhage in the gastric antrum. These findings are supportive to the results obtained in pharmacological and biochemical parameters.

In conclusion, the results of the present study show that the ethanol extract of *Apium graveolens* displays gastroprotective activity, as demonstrated by its significant inhibition of the formation of ulcers induced by different experimental models, and its ability to decrease basal gastric acid secretion. This gastric antiulcer capacity of celery extract could be related to its antioxidant properties, resulting in reduction of the lipid peroxidation and elevation of the NP-SH contents, in addition to improving the mucus coat of the stomach. Therefore, we suggest that due to its antioxidative effects, it may be useful in the prevention of gastric disorders.

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Declaration of interest

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