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Biological Study of the Protective Effects of Fennel, Chamomile and Cardamom Oils Against Aspirin Induced Gastric Ulcers in Male Albino Rats

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Abstract: The present study is conducted to investigate the effect of protective effects of fennel, chamomile and cardamom oils against Aspirin Induced Gastric Ulcers in Male Albino Rats. Forty two male albino rats were classified into normal control group and five aspirin induced gastric ulcer groups. The results revealed that, the treatment with these oils can protect from aspirin induced gastric ulceration. The mechanism of its gastro protective activity may be attributed to significant reduction in volume of gastric juice, total acidity of gastric juice, gastric ulcer index, serum total oxidative capacity , serum interleukin-1 IL-1, serum tumor necrosis factor-alpha, gastric COX-2 activity and gastric total nitric oxide concentration along with significant elevation in serum total antioxidant capacity , blood hemoglobin, gastric prostaglandin PGE2 level and gastric cytochrome P_{450} reductase activity compared with ulcerated control (+ve) group. Curative ratio percentage showed insignificant difference of oils treatment groups compared with RAN drug group. These obtained results are confirmed by the histopathological examination. The protection afforded by treatment of fennel , Chamomile and Cardamom oils was found to be effective. In conclusion, the fennel , Chamomile and Cardamom oils possess antiulcer potential due to its antioxidant and anti-effect. The healing activity may be due to its cytoprotectivity effect coupled with anti-secretory activity.

Key words: Fennel oil, Chamomile oil, Cardamom oil, Aspirin, Gastric ulcer.

Introduction:

Peptic ulcer disease (PUD), encompassing gastric and duodenal ulcers is the most prevalent gastrointestinal disorder. The pathophysiology of PUD involves an imbalance between offensive factors like acid, pepsin and defensive factors like nitric oxide and growth factors **(Sloan, 2010)**. Always there has been renewed interest in identifying new antiulcer drugs from natural sources **(Brito *et al*., 1997)** since chemical compounds are known to have undesirable side-effects. Gastric ulcers are believed to be due to an imbalance between acid and pepsin along with weakness of the mucosal barrier. The gastric mucosa is continuously exposed to potential injurious agents such as acid, pepsin, bile acid, bacterial products and drugs **(Goncalves *et al*.,2006)**. In recent studies, many medicinal plants continue to provide valuable therapeutic agents for the treatment of ulcers both in modern medicine and by the traditional system throughout the world. Besides, some medicinal plants have been found to have both preventive and/or therapeutic effects on ulcers**(Gshanthi *et al*.,2009).**

The essential oil of fennel is obtained through the steam distillation of crushed fennel seeds, a herb which bears the scientific name of (*Foeniculum Vulgare)*. Fennel seeds have been in use in culinary applications and as a mouth freshener since ancient times. They are also widely used for medicinal purposes. These medicinal properties come from the various components of the essential oil of fennel, including alpha pinene, anisic aldehyde, cineole, fenchone, limonene, myrcene, methyl chavicol and trans anethole. Fennel oil helps to keep the stomach healthy and functioning properly, and maintains the normal function of all the secretions of digestive acids and bile. Furthermore, it protects the stomach from infections and ulcers, thus ensuring the all around health and wellness of the stomach **(Sabiha *et al* .,2011)** .

Chamomile oil is extracted from the flowers of the chamomile plant, which is very popular as a flowering plant. There are two types of

chamomile, the roman chamomile, which is scientifically known as *Anthemis Nobilis* and the german chamomile, whose scientific name is (*Matricaria Chamomilla).* Although the essential oils extracted from both varieties are quite similar in some medicinal properties, their composition is different and they do possess certain specific qualities that are worth noting. Roman chamomile oil is composed of alpha pinene, beta pinene, camphene, caryophyllene, sabinene, myrcene, gamma terpinene, pinocarvone, farsenol, cineole, propyl angelate and butyl angelate. German chamomile oil, on the other hand, is composed of azulene (also called *Chamazulene),* alpha bisabolol, bisabolol oxide-A & B and bisabolene oxide-A. Being a stomachic, they tone up the stomach and ensure its proper function. They also promote the secretion of digestive juices into the stomach and facilitate digestion. Only about 0.5% to 1.9% of the chamomile plant is essential oil. Chamomile essential oil is a mixture of a number of separate oils, and roughly 120 secondary metabolites have been identified in it. Twenty eight terpenoids and thirty six flavinoids have been also identified. **(Denkova *et al*., 2009)**

Cardamom essential oil is extracted from the seeds of cardamom, whose scientific name is(*Elettaria Cardamomum).* It is extensively used and admired as a versatile spice around the world. It is the essential oil in cardamom that makes it such a good digestive. This oil boosts digestion by stimulating the whole digestive system. It is also stomachic in nature, which means that it keeps the stomach healthy and functioning properly. It helps maintain the proper secretion of gastric juices, acids and bile in the stomach. It also protects the stomach from infections **(Jafri *et al*., 2001).** Cardamom essential oil stimulates the entire system. This stimulating effect also boosts the spirits in cases of depression or fatigue. It also stimulates the secretion of various enzymes and hormones, gastric juices, peristaltic motion, circulation, and excretion, thus maintaining proper metabolic action throughout the body **(Marhuenda *et al*,. 1993).**

Aspirin is a potent nonsteroidal anti-inflammatory drug (NSAID) that is used for the treatment of rheumatoid arthritis and related diseases as well as the prevention of cardiovascular thrombotic diseases **(Laine *et al*., 2008)**. Gastric ulcer associated with the use of aspirin is a major problem. Many factors such as gastric acid and pepsin secretion, gastric microcirculation prostaglandin E2 (PGE2) content , pro-inflammatory cytokines interleukin (IL)-1 and tumor necrosis factor (TNF) play important roles in the genesis of gastric mucosal damage and its subsequent development **(Wang *et al*., 2007 and Wallace, 2008)** .It has been reported that the increases in NO synthase (NOS) activity is involved in the gastrointestinal mucosal defense and also in the pathogenesis of mucosal damage **(Wallace *et al*.,2000 and Mari *et al*.,2007).** Recent studies found that different substances from medicinal plant sources not only offer gastric protection but also accelerate ulcer healing **(Darbar, 2010 and Magaji *et al*., 2007) .** Thus, the aim of this study was designed to investigate the antiulcer effects of fennel , chamomile and cardamom oils in aspirin-induced gastric ulcer model rats.

Materials And Methods :

Materials :

The herbal oils drug constituents, fennel oil (*Foeniculum Vulgare*) , chamomile oil (*Anthemis Nobilis*) and cardamom oil (*Elettaria Cardamomum*) were obtained from Agricultural Research Center, Giza, Egypt. Each one of them was administered daily at dose 1ml/kg body weight of rats orally by stomach tube.

Forty tow male albino rats of Sprague Dawley strain were purchased from Laboratory Animal Colonies, Helwan, Egypt. The average weight was 145±10g. One gram vials of aspegic drug were obtained from Sigma Pharmaceutical Industries, Cairo, Egypt. It was synthesized from salicylic acid, acetic anhydride and non corrosive 12- tungstophosphoric acid. One vial was dissolved in 10 ml distilled water and administered orally as a single dose every week of freshly prepared aspirin solution in

dose 400 mg/kg body weight of rats to induce gastric ulcer according to **Main and Whittle (1975)**. Ranitidine tablets were obtained from SEDICO Pharmaceutical Company, Giza, Egypt. Each tablet contains 150 mg of ranitidine hydrochloride that inhibits gastric ulcer. Ranitak drug was dissolved in distilled water in dose 30 mg/kg of rat using a stomach tube. The basal diet was performed according to **(NRC., 1995).**

Methods :

Experimental Animals: Rats were fed on the basal diet for one week before starting the experiment for adaptation , then the rats were allocated into six equal groups. Normal control group fed on the basal diet only while the other five groups were administered orally of freshly prepared aspegic orally to induce gastric ulcer then classified into non treated control (+ve) and treated groups that were drug group, 1 ml fennel oil, 1 ml chamomile oil and 1 ml cardamom oil treatment groups. Daily food intake and weekly body weight gain were calculated. Food efficiency ratio (FER) was determined according to the method of **(Chapman *et al*.,1959).** At the end of the experiment period (6 weeks), rats were sacrificed after overnight fasting under ether anesthesia. Blood samples were taken from hepatic portal vein, small part was taken into heparinised tube and the remainder were left to clot by standing at room temperature for 15 minutes and then centrifuged at 3000 rpm for 20 minutes. Serum was carefully separated and transferred into clean quite fit plastic tubes and kept frozen at - 20°C until the time of biological analysis.

Biochemical Estimations:

Determination of Gastric Volume: After sacrificing the rat, the stomach portion was removed. The gastric contents were transferred in to centrifuge tube and centrifuged at 1000 rpm for 10 minutes. The supernatant liquid was then transferred to a measuring cylinder, and the volume was measured. **(Beckers *et al*.,1988).**

Determination of Free Acidity and Total Acidity: One mL of gastric juice was pipette out into a 100 mL conical flask. 2 to 3 drops of

Topfer's reagent were added and titrated with 0.01 N NaOH (which was previously standardized with 0.01 N of oxalic acid) until all the trace of the red color disappeared and the color of the solution was yellowish orange. The volume of the alkali added was noted. This volume corresponds to free acidity. Acidity was calculated by using the formula:

Acidity= Vol.ofNaOH × Actualnormality of NaOH0.1×100mEq/L/100g.

The acidity of gastric juice was calculated as total acid content/gastric juice volume in mEq/l **(Khushtar *et al*., 2009)**

Determination of Gastric Ulceration: The stomachs were opened longitudinally, washed with saline solution and examined under dissecting microscope for gastric ulcer . The length of gastric ulcer was measured for each group to determine the way of ulcer index (UI) and the curative ratio according to **Parmar and Desai (1993).** The ulcerative index was calculated by severity of gastric mucosal lesions 1mm or less, 1-2 mm and more than 2 mm and graded as 1, 2 and 3 score, respectively. Then the UI was calculated by using the formula: UI = 1 x (number of lesions of grade 1) + 2 x (number of lesions of grade 2) + 3 x (number of lesions of grade 3).Then the overall score was divided by a factor 10, which was designated as ulcer index.

The curative ratio was calculated for each group as following: Curative ratio = (length of gastric ulcer in control positive group - length of gastric ulcer in treated group/length of gastric ulcer in control positive group) x100.

Serum analysis : Total antioxidants capacity (TAC), Total oxidative capacity (TOC), Interleukin-1(IL-1), Tumor necrosis factor-alpha (TNF-α) were determined according to **Cao *et al*.,(1993), Flohe and Gunzler (1984), Grassi *et al*.,(1991) and Beutler *et al*., (1985)** respectively.

Determination of Blood Hemoglobin: Blood hemoglobin was estimated according to **Drabkin (1949).**

Determination of Gastric Mucosa: Gastric mucosal of cyclooxygenase (Cox-) activity, prostaglandin E (PGE) level, cytochrome P reductase (Cyto- P) activity and total nitric oxide (TNO) concentration were determined according to **Hemler and Lands (1976), Hamberg and Samuelsson (1973), Mc-Lean and Day (1974) and Griess *et al*., (1982)** respectively.

Histological Examination: Stomach and duodenum obtained from pharmacological studies were immersed in 10% formalin as fixative and sent to Cancer Institute for histopathological examination according to **Bancroft *et al*., (1996).**

Statistical Analysis: The obtained results (mean ± SEM) were analyzed using one-way ANOVA followed by Duncan's test. The minimum level of significance was set at □<0.05.(SPSS, 2008).

Results And Discussion :

Data presented in Table (1) show that, control (+ve) rat group exhibited a significant decrease in final body weight, body weight gain (%), food intake and food efficiency ratio while, rat groups fed on drug, fennel oil, chamomile oil and cardamom oil rat groups showed insignificant difference in food intake and FER compared with control (-ve) group. Treatment with drug, fennel oil, chamomile oil and cardamom oil showed significant increases in final weight, weight gain (%), food intake and FER, compared with control (+ve) rat group.

The results in Table (2) indicated that aspirin administration caused significant increase in volume and total acidity of gastric juice associated with significant decrease in acidity of control (+ve) group. While, the treatment with RAN drug or fennel , chamomile and cardamom oils produced a significant reduction in volume of gastric juice and total acidity and significant increases in acidity, compared with control (+ve) group. The values of gastric ulcer index were significantly decreased in all treated rat groups compared with control (+ve) group. The treated groups with fennel oil, chamomile oil and cardamom oil showed

insignificant difference in curative ratio percentage compared with RAN drug group. In accordance with the obtained results, aspirin has been reported to reduce the gastric juice pH and increase the volume of gastric juice **(Wang *et al*., 2007).** The anti-ulcerative effects capability of fennel, chamomile and cardamom oils were investigated in aspirin-induced gastric ulcer model rats. It is evident from the present results that all these oils have potent ulcer protective activity at 1ml/kg. There was a significant decrease in ulcer index in these oils treated rats as ranitidine treated rats. Also, there was a significant decrease in the volume of gastric content and total acidity in all oils treated rat groups as compared with ulcerated control rats. The curative ratio (%) in all oils treated groups showed insignificant difference compared to ranitidine group.

Table (3) revealed that, aspirin administration led to significant increases in serum total oxidative capacity, interleukin-1 and tumor necrosis factor-alpha levels while, there were significant decreases in serum total antioxidant capacity level and blood hemoglobin content. Rat groups fed on drug, fennel, chamomile and cardamom oils showed significant decrease in total oxidative capacity, interleukin-1 and tumor necrosis factor-alpha while there were significant increase in total antioxidative capacity and hemoglobin compared to ulcerated positive control group.

The obtained results gave scientific evidence that fennel, chamomile and cardamom oils may contain biological substances with potential anti-ulcer properties. This gastro protective effect may be due to the high flavonoids content of these oils. The obtained results are consistent with those of **Khushtar *et al*., (2009)** who indicate that fennel oil has a protective action against gastric ulcers induced by aspirin in rats. The fennel oil has antisecretory activity, as observed by the decrease in total acidity and volume of gastric juice. Further, the fennel oil treatment offers cytoprotection by increasing mucus inhibition. Also, **Magaji *et al.*, (2007)** reported that chamomile extract showed good gastro protective anti-ulcerogenic activity and they attributed this effect to the

anti-oxidativ activity of flavonoids found in the extract. Furthermore, **Rachhadiya *et al.*, (2011)** demonstrated that cardamom oil possess antiulcer activity against the ulceration caused by aspirin. Aspirin a well-known NSAID which induces erosions and ulcers in gastroduodenal tract through different processes, such as inhibition of cyclooxygenase mediated prostaglandin synthesis, generation of reactive oxygen species (ROS) and induction of apoptosis. Inflammatory injury is associated in part with increased generation of ROS **(Sarkar and Buha, 2008).** Moreover, **Shyamal and Chandan (2012)** reported that aspirin significantly decreased the levels of SOD,CAT and GSH activities as well as increased lipid oxidation MDA in aspirin- treated experimental group, compared with control group. The inhibition of TAC level and the elevation of TOC concentration in the present study during aspirin induced ulcer due to the increased generation of reactive free radicals, which can create an oxidative stress in the cells. The administration of RAN drug and using herbal oils inversed these results which through protection from the free radical induced oxidative stress. This result supports that, these oils have potential antioxidant action on gastric ulcer rat model through phenolic compounds that scavenges peroxyl radicals and have a powerful antiulcer activities. These compounds contain an OH group linked with the aromatic ring and thus may possess potential antioxidant and antiulcer activities (**Shyamal and Chandan,2012)**.

Inflammation and neutrophil infiltration are also important in the pathogenesis of the gastric damage induced by NSAIDs **(Souza *et al*., 2004).** The inflammation induced in the gastric mucosa by aspirin is accompanied by increased production **(Naito *et al* ., 2001) and (Jainu and Devi, 2006),** which augments neutrophil-derived superoxide generation **(Kwiecien *et al*., 2002)** and stimulates IL-1 production, leading to neutrophil accumulation **(Kokura *et al*., 2000 and Odashima *et al*., 2006)**. Over production of TNF- α increases the risk of gastric ulcer and cancer **(Mitsushige *et al*., 2007)** . In the present study, the levels of TNF-α active and IL-1 were increased by aspirin administration and the treatment with these oils inhibited the increases in TNF- α and

IL-1 levels. These results are in agreement with those obtained by **(Wang *et al*., 2011)** who reported that fennel powder inhibited the increasing in TNF- α and IL-1 levels without ulcer formation progressing. Suppression of TNF- α and IL-1 production this may be attributed to the anti-inflammatory activity of these treatment oils. The TNF- α and neutrophil infiltration will ultimately inhibit tissue destruction by reactive oxygen species **(Kwiecien *et al*., 2002)**.

The statistical analysis in Table 4 presented that, control (+ve) rat group showed significant increase in gastric cyclooxygenase (COX2) activity and total level nitric oxide compared to (-ve) control group, while it showed significant decrease in gastric prostaglandin E2 and cytochrome P_{450} reductase activity compared to (-ve) control group. The groups fed on drug, fennel oil, chamomile oil and cardamom oil showed a significant decrease in gastric cyclooxygenase (COX2) activity and total nitric oxide compared to (+ve) control group, while they showed a significant increase in gastric prostaglandin E2 and cytochrome P_{450} reductase activity compared to (+ve) control group. Prostaglandins have protective effects against various gastric injury models. Aspirin has been shown to reduce the mucosal PGE2 content **(Kontureck *et al*,.2006).** Prostaglandin, a key molecule that stimulates the complex array of ulcer healing mechanism, gets synthesized in the mucosal cells by cyclooxygenase (COX) enzymes. It stimulates the secretion of biocarbonate and mucus, maintains mucosal blood flow and regulates mucosal turn over and repair. Suppression of prostaglandins synthesis by aspirin may be led to increase the susceptibility of stomach to mucosal injury and gastroduodenal ulceration **(Anoop and Jegadeesam, 2003 and Hiruma-Lima *et al*., 2006)** . The data were obtained in the ongoing investigation into a parallel line with previous studies, aspirin significantly reduced gastric mucosal prostaglandin E2 (PGE2) level and gastric cyto P reductase activity while elevated COX-2 activity compared to control. Treatment with oils significantly inversed these results when compared to aspirin treated groups. This finding was explained by **Borrelli and Izzo (2000)** who reported that flavonoids

may protect the gastric mucosa from damage by increasing the mucosal prostaglandin content and by inhibiting histamine secretion from mast cells by inhibition of histidine decarboxylase. Fennel appears to exert anti-inflammatory effects by suppressing COX-2 activity **.(Frondoza *et al*., 2004)** . One of the mechanisms by which aspirin damages the gastric mucosa is the increased production of NO due to the over expression of inducible nitric oxide synthetase (iNOS) **(Kontureck *et al*., 2006)**. NO is a mediator not only of gastrointestinal mucosal defense, but also of its damage. It has been shown that different concentrations of NO have completely an opposite effects in the same tissue. In general, the mucosal and endothelial NOS isoforms produce low amounts of NO. However, the high quantity of NO produced by iNOS damages the epithelium **(Wallace and Miller, 2000)**. The excessive release of NO from gastric epithelial cells induced by aspirin has been reported to exert detrimental effects **(Whittle, 2003)** and **(Hsu and Liu, 2004)**. Inhibiting aspirin induced increases in iNOS expression in the gastric mucosa leading a reduction in gastric mucosal damage **(Kontureck *et al*., 2006)**. In the present study, the results of fennel, chamomile and cardamom oils , treatment groups agree with **Wang *et al*.**, **(2011)**, who reported that fennel powder reduced iNOS activity and inhibited the production of gastric ulcers, even in the presence of aspirin. Both constituents of fennel, chamomile and cardamom showed protective effects against aspirin-induced gastric ulcers together with anti-inflammatory effects by reducing iNOS activity in the gastric mucosa and inflammatory cytokine (TNF and IL-1) expression. These effects of fennel powder seem to be derived from the actions of chavicol and cineole, the main ingredients of fennel as reported by **(Wang *et al*.,2011; Lantz *et al*., 2007 and Isa *et al*., 2008).** Another study carried out by **Mari *et al*., (2007)**, who explained that anti-inflammatory compounds of flavonoids as flavone, the isoflavones, the flavonols isorhamnetin, kaempferol, quercetin and the anthocyanin inhibited iNOS expression.

Histopathological Results: The obtained results are confirmed by the histopathological examination. Stomach of control (-ve) group showed normal histological gastric mucosa (Figure1). While, stomach of control

(+ve) group showed focal necrosis of gastric mucosa associated with mucosal and submucosal eosinophilic cells infiltration (Figure2). Stomachs of drug, fennel, chamomile and cardamom oils treatment groups showed no histopathological changes (Figure 3-6, respectively).

Histopathological studies on the gastric mucosa revealed that aspirin administration induced a mucosal ulceration, associated with significant increase in lipid peroxidation. This was manifested as lamina epithelial necrosis, blood vessels congestion and leukocytic infiltration **(Valcheva-Kuzmanova *et al*., 2007 and EL-Moselhy *et al*., 2009),** this effect on histological derangement was in accordance with our results. Moreover, all treatment groups with oils showed protective effect against aspirin induced inflammatory infiltration and congestion at the ulcer sites. They prevented gastric mucosal lesions through their flavonoids content. Flavonoids could scavenge free radicals, inhibit lipid peroxidation and increase prostaglandins and mucosal content of the gastric mucosa; showing cyto-protective effects **(Alanko *et al*., 1999).**

Conclusion:

Fennel, chamomile and cardamom oils possess antioxidant, anti-inflammatory, which may be responsible for its antiulcerogenic activity the presence of phyto-constituents in these medicinal oils, particularly flavonoids might be responsible for these pharmacological actions. These phyto-constituents provide protection against gastric mucosal damage induced by aspirin. The healing activity may be due to its cyto-protective effect coupled with anti-secretory activity.

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Tables:

Table (1) : Effect of fennel, chamomile, and cardamom oils treatment on nutritional parameters against aspirin induced gastric ulcers in rats.

Groups	Parameters				
	Initial weight(g)	**Final weight (g)**	**Weight gain (%)**	**Food intake (g/d)**	**FER**
Normal control group (-ve)	148.34 ±9.03[a]	235.32 ± 12.48[a]	58.51 ±9.58[a]	17.22 ±2.01[a]	0.174 ±0.001[a]
ASP. control group (+ve)	149.31 ±8.84[a]	194.31 ±9.89[d]	34.33 ±5.66[c]	12 .67 ±2.13[b]	0.097 ±0.002[b]
ASP+RAN drug group	152.26 ±11.22[a]	217.01 ±12.14[b]	44.61 ±6.43[b]	15.23 ±2.11[a]	0.162 ±0.003[a]
ASP+ 1ml/kg fennel oil	145.61 ±8.07[a]	211.00 ±13.71[c]	48.35 ±8.13[b]	16.45 ±2.87[a]	0.168 ±0.001[a]
ASP+ 1ml/kg chamomile oil	149.72 ±9.76[a]	218.00 ±10.84[b]	45.26 ±6.17[b]	16.77 ±2.16[a]	0.172 ±0.001[a]
ASP+ 1ml/kg cardamom oil	151.31 ±9.58[a]	213.00 ±10.27[bc]	43.98 ±7.93[b]	16.94 ±2.69[a]	0.174 ±0.003[a]

Values with the same letters indicate insignificant difference and vice versa ($P<0.05$).

ASP: aspirin, RAN: Ranitidine, FER: Food efficiency ratio

Table (2) : Effect of fennel, chamomile, and cardamom oils treatment on gastric juice analysis and gastric ulceration against aspirin induced gastric ulcers in rats.

Groups	Parameters						
	Volume of gastric juice (1ml)	**Decrease volume of gastric juice (%)**	**Acidity (meq/l)**	**Total acidity of gastric juice (meq/l)**	**Decrease in gastric total acidity (%)**	**Ulcer index (mm)**	**Curative ratio (%)**
Normal control group (-ve)	1.12 ±0.15^{d}	-	5.11 ±0.53^{a}	5.42 ±0.38^{d}	-	-	-
ASP. control group (+ve)	6.94 ±0.13^{a}	-	2.31 ±0.13^{d}	24.56 ±1.96^{a}	-	9.78 ±1.37^{a}	-
ASP+RAN drug group	3.71 ±0.23^{c}	51.62 ±3.13^{a}	4.37 ±0.11^{b}	15.03 ±1.51^{b}	43.81 ±3.76^{b}	2.37 ±0.53^{b}	77.51 ±7.82^{a}
ASP+ 1ml/kg fennel oil	4.82 ±0.25^{b}	42.23 ±3.51^{c}	3.68 ±0.16^{c}	12.55 ±1.35^{c}	57.86 ±4.74^{a}	2.76 ±0.92^{b}	74.56 ±8.11^{a}
ASP+ 1ml/kg chamomile oil	3.93 ±0.27^{c}	46.75 ±3.66^{b}	3.79 ±0.11^{bc}	14.32 ±1.22^{b}	42.19 ±4.83^{b}	2.01 ±0.86^{b}	79.42 ±9.41^{a}
ASP+ 1ml/kg cardamom oil	4.16 ±0.32^{b}	43.16 ±2.97^{c}	3.52 ±0.19^{c}	11.81 ±1.17^{c}	55.17 ±4.75^{a}	2.73 ±0.58^{b}	75.58 ±8.27^{a}

Values with the same letters indicate insignificant difference and vice versa. ($P<0.05$).

ASP: aspirin RAN: Ranitidine

Table (3): Effect of fennel, chamomile, and cardamom oils treatment on serum levels of total antioxidative capacity, total oxidative capacity, Interleukin-1, tumor necrosis factor-alpha and blood hemoglobin against aspirin induced gastric ulcers in rats.

Groups	Parameters				
	TAC mmol/L	**TOC mmol/L**	**IL-1 pg/ml**	**TNF-α pg/ml**	**Hb g/dl**
Normal control group (-ve)	1.86 ±0.05^{a}	0.31 ±0.05^{c}	13.54 ±1.31^{f}	3.12 ±0.35^{e}	13.72 ±1.41^{a}
ASP. control group (+ ve)	0.81 ±0.02^{e}	1.04 ±0.09^{a}	53.09 ±8.56^{a}	12.33 ±2.43^{a}	9.22 ±0.21^{b}
ASP+RAN drug group	1.46 ±0.07^{b}	0.51 ±0.04^{c}	25.17 ±2.23^{e}	6.14 ±1.25^{d}	13.33 ±1.84^{a}
ASP+ 1ml/kg fennel oil	1.22 ±0.01^{c}	0.76 ±0.03^{b}	34.18 ±2.73^{d}	8.87 ±1.33^{c}	13.76 ±1.76^{a}
ASP+ 1ml/kg chamomile oil	1.13 ±0.04^{d}	0.84 ±0.02^{b}	45.31 ±3.34^{d}	9.91 ±1.71^{bc}	13.85 ±1.57^{a}
ASP+ 1ml/kg cardamom oil	1.11 ±0.08^{cd}	0.80 ±0.01^{b}	35.12 ±2.95 ^{d}	9.18 ±1.73^{c}	13.01 ±1.07^{a}

Values with the same letters indicate insignificant difference and vice versa. ASP: aspirin, RAN: Ranitidine, TAC: total antioxidative capacity, TOC: total oxidative capacity, IL-1: Interleukin-1, TNF- α : tumor necrosis factor-alpha, Hb: hemoglobin.

Table (4): Effect of fennel, chamomile, and cardamom oils treatment on gastric tissues of cyclooxygenase, prostaglandin E2, cytochrome P_{450} reductase and total nitric oxide against aspirin- induced gastric ulcers in rats

Groups	Parameters			
	Cox-2 ng/mg	**PGE2 pg/mg**	**Cyto P_{450} reductase ng/mg**	**TNO pg/mg**
Normal control group (-ve)	5.13 ±0.031^{e}	505.61 ±17.33^{a}	1.91 ±0.016^{a}	33.79 ±5.16^{d}
ASP. control group (+ve)	16.89 ±0.29^{a}	304.25 ±11.46^{e}	0.62 ±0.010^{e}	71.43 ±8.76^{a}
ASP+RAN drug group	6.67 ±0.78^{d}	453.7 ±16.94^{b}	1.52 ±0.011^{b}	48.21 ±6.31^{c}
ASP+ 1ml/kg fennel oil	9.72 ±0.18^{c}	425.23 ±19.49^{c}	1.18 ±0.021^{c}	50.91 ±6.75^{c}
ASP+ 1ml/kg chamomile oil	12.48 ±0.57^{b}	384.57 ±15.12^{d}	0.91 ±0.010^{d}	59.87 ±5.12^{b}
ASP+ 1ml/kg cardamom oil	13.96 ±0.71^{b}	378.17 ±13.77^{d}	0.82 ±0.013^{d}	55.52 ±7.53 ^{bc}

Values with the same letters indicate insignificant difference and vice versa. ASP: aspirin, RAN: Ranitidine, Cox: cyclooxygenase, PG: prostaglandin, Cyto: cytochrome, TNO: total nitric oxide

Figures:

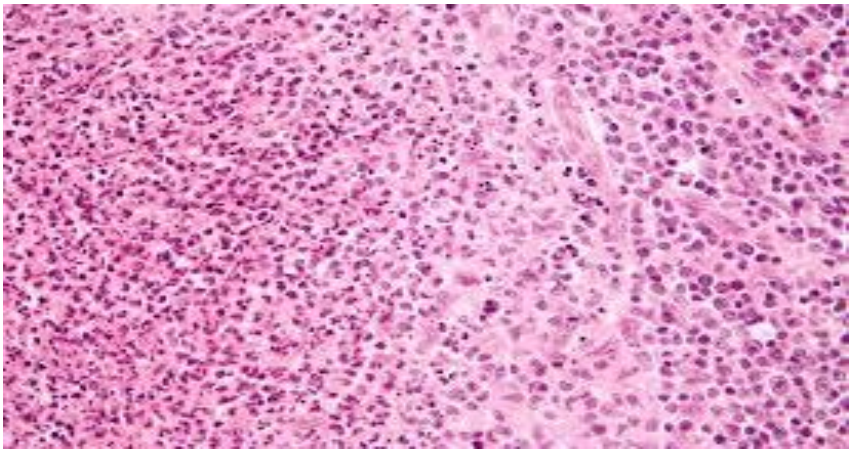

Fig. (1): Stomach of rat from control group showing normal gastric mucosa (H and E X 200)

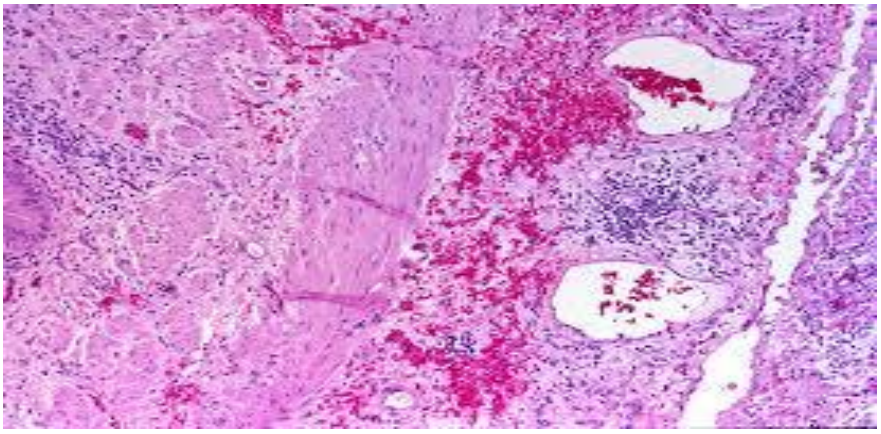

Fig. (2): Stomach of rat from positive control group As shown in Table 3, aspirin administration led to

- showing focal necrosis of gastric mucosa significant increases in serum total oxidative capacity,
- associated with mucosal and submucosal eosinophilic cells infiltration (H and E X 200)

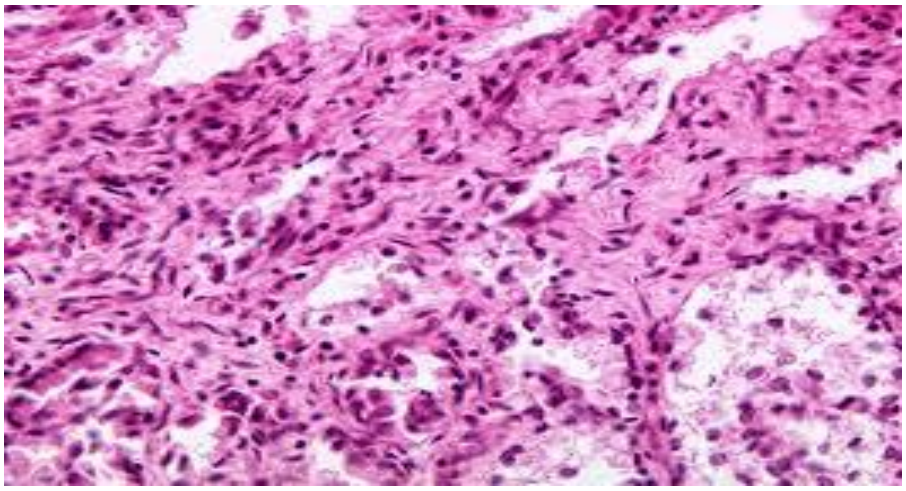

Fig.(3): Stomach of rat from drug group showing no histopathological changes (H and E X 200)

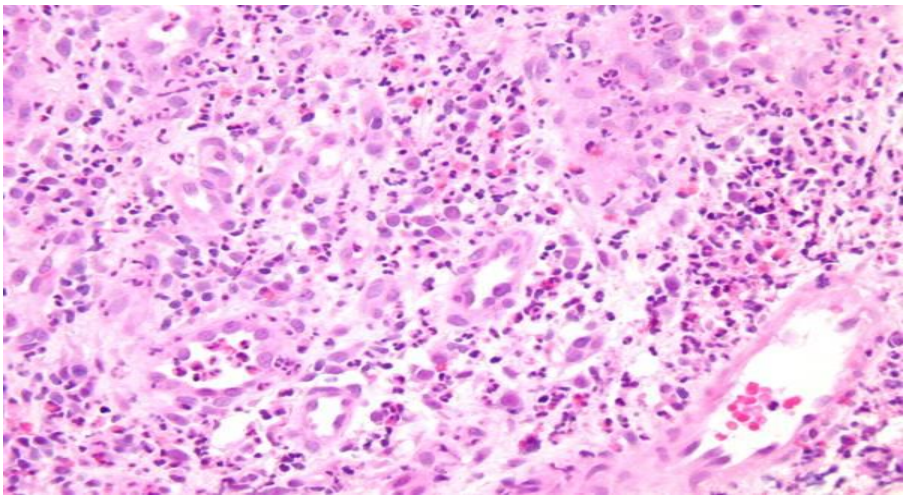

Fig.(4): Stomach of rat from group 1ml fennel oil showing no histopathological changes (H and E X 200)

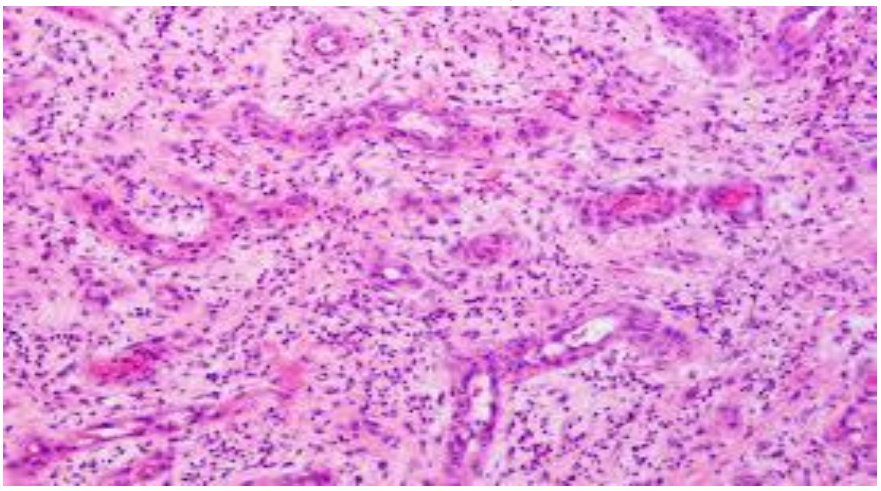

Fig. (5): Stomach of rat from group 1ml chamomile oil showing no histopathological changes (H and E X 200)

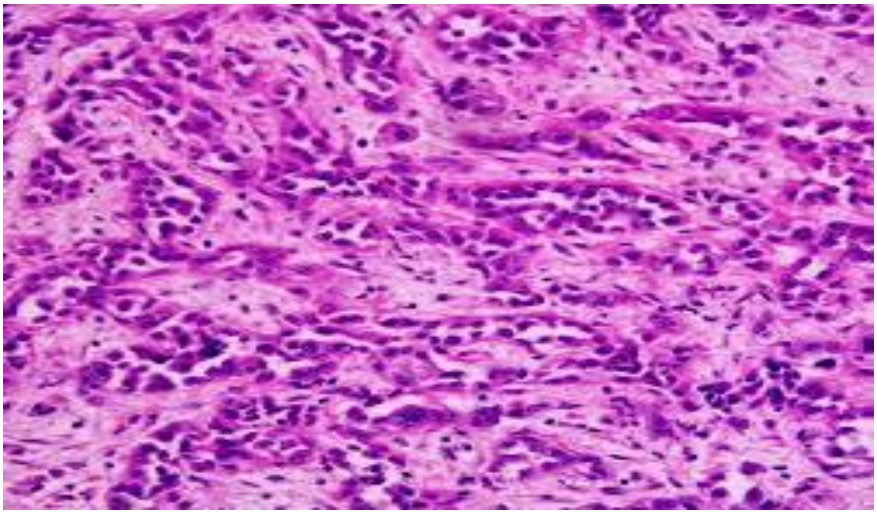

Fig. (6): Stomach of rat from group 1ml cardamom oil showing no histopathological changes (H and E X 200)

مجلة الاقتصاد المنزلي - مجلد ٢٤- العدد الثالث – ٢٠١٤م

دراسة بيولوجية للتأثير الوقائي لزيت كل من الشمر و البابونج و الهيل علي قرحة المعدة المستحثة بالأسبرين في ذكور فئران الألبينو

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الملخص العربي:

تهدف الدراسة إلي التحقق من التأثير الوقائي لزيوت الشمرو البابونج والهيل علي قرحة المعدة المستحثة بالاسبرين في ذكور فئران الألبينو. تم تصنيف اثنين وأربعين من ذكور الفئران الي مجموعة ضابطة وخمسة مجموعات مصابة بقرحة المعدة المستحثة بالأسبرين .

وأظهرت النتائج أن العلاج بهذه الزيوت يمكن أن يحمي من تقرحات المعدة الناجمة عن الأسبرين. ويمكن أن يعزى آلية نشاطها الوقائي إلى انخفاض كبير في حجم عصارة المعدة و الحموضة الكلية لعصارة المعدة ومؤشر قرحة المعدة ،و قدرة الأكسدة الكلية، والعوامل المسببة لأورام المعدة، و تركيز أكسيد النيتريك جنبا إلى جنب مع ارتفاع ملحوظ في القدرة المضادة للأكسدة في مصل الدم ،وهيموجلوبين الدم ،ومستوى البروستاجلاندين PGE2 بالمعدة والسيتوكروم P_{450} المعدي مقارنة مع المجموعة الضابطة الموجبة.

كما أظهرت النتائج أن الفرق في التأثير الوقائي ضئيل بين مجموعات العلاج بالزيوت بالمقارنة مع مجموعة الدواء RAN. وتأكدت هذه النتائج من خلال فحص الأنسجة. ووجد أن التأثير العلاجي لزيوت كل من الشمر، والبابونج والهيل فعالة لما لها من تأثير مضاد للأكسدة، ومضاد للالتهابات.

الكلمات المفتاحية : زيت الشمر - زيت البابونج - زيت الهيل- الأسبرين- قرحة المعدة.