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S2-011

Biological Effect of Some Fast Foods Fortified with Red Chili Pepper (RCP) on Peptic Ulcer, Lipids Profile, Liver and Kidneys Functions

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Abstract

There is urgent need to address illness problems caused by Salmonella enteric serotype Typhibacteria. The bacteria are deposited in water or food by human carrier and are then spread to other people in the area. In this research, a blood specimens were collected from typhoid fever patients, and serum levels of IFN-γ and IL-6 during the chronic and acute phase in typhoid patients group was determined according to protocol kit and calculation, results were higher levels in chronic phase (137.187±0.703.427±206.545 pg/ml respectively) and in acute phase were 128.787±2.522, 137.733±23.424 pg/ml, respectively with highly significant (P≤0.01) than those in healthy control group. Salmonella infects hosts as diversified as human, animal, and plants.

Keywords: *Salmonellatyphi*; Serology; IL6; INF-γ; *Salmonella* Infects Hosts as Plant

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Introduction

Fast food including spicy chicken and nuggets with red chili pepper are said to be not good for health because these foods rich in fat and cholesterol, and the high intake is associated with increased blood cholesterol levels and coronary heart disease (CHD). There is relation between red meat consumption and CHD risk factors. Dietary intake of saturated fat comes from fast food, snack food, oils and spreads, therefore in present study, biological studies were carried on selected fast foods using Sprague Dawley rats.

Chili peppers have been part of the human diet in the Americas since at least 7500 BC and perhaps earlier. There is archaeological evidence at sites located in southwestern Ecuador that chili peppers were well domesticated more than 6000 years ago [1].

In the previous century peptic ulcer was an uncommon disorder. However, during the early of the 1900s, the disease began to increase greatly. In the 1940s at least 10% to 15% of men were reported to have duodenal ulcers, with un- reported cases in women and children adding to those figures. This increasing seems to have peaked about the mid-1950s, and over the past 15 to 20 years. The knowledge on the disease process and its medical management has progressed rapidly.

Increasing of acid output, Helicobacter pylori (H. Pylori), non-steroidal anti- inflammatory drugs (NSAIDs) and stress are the basic risk factors in peptic ulcer disease [2]. Several studies found that capsaicin could have an anti-ulcer protective effect on stomach infected with H. pylori by affecting the chemicals the stomach secretes in response to infection [3-5].

The pain is the predominant symptom of peptic ulcer, although it may be absent in 25% of gastric ulcer patients. And upper gastro-intestinal hemorrhage may be the presenting sign of peptic ulcer disease, anemia from chronic blood loss, repeated vomiting and weight loss possibly happen [6].

There are many plants used for treatment of peptic ulcer, such as vegetables, fruits, and herbs. These substances had been experimented for treatment of peptic ulcer. But when we mention that spices use for healing ulcers, most people not only criticize us but also interpose with our speech. A lot of studies had been done on behalf of spices especially red chili pepper, some scientists did not find any medicinal effect of these spices on healing ulcers, as that of Myers BM, et al. (1987).

On the other hand, other scientists found that some spices especially red chili pepper has a good medicinal effect on healing ulcers. They found that red chili pepper involves functional substances called capsaicin, which has a gastro-protective effect against experimental gastric mucosal injury in animals [8-11].

Research in humans found that “after adding chili to the diet, the LDL, or bad cholesterol, actually resisted oxidation for a longer period of time, (delaying) the development of a major risk for cardiovascular disease” [12].

On the contrary, Alasdair GT, et al. (1981) remarked that hot and spicy food as long as uses aspirin is the main causes of chronic gastritis leading to ulcer disease. In this respect, red and black pepper incriminated in the increase of acid secretion [14,15].

All hot chili peppers contain phytochemicals known collectively as capsaicinoids.

- Capsaicin was shown, in laboratory settings, to cause cancer cell death in rats [16].
- Capsaicin in chilies has been found to inhibit chemically induced carcinogenesis and mutagenesis in various animal models and cell culture systems [1].

Research on mice showed that chili (capsaicin in particular) may offer some hope of weight loss for people suffering from obesity [17].

Researchers used capsaicin from chilies to kill nerve cells in the pancreases of mice with Type 1 diabetes, thus allowing the insulin producing cells to start producing insulin again [18].

Research on humans found that "after adding chili to the diet, the LDL, or bad cholesterol, actually resisted oxidation for a longer period of time, [delaying] the development of a major risk for cardiovascular disease" [12].

The amount of insulin required to lower blood sugar after a meal is reduced if the meal contains chili pepper (Blood sugar and spice Science News 2007).

Capsaicin could have an anti-ulcer protective effect on stomachs infected with H. pylori by affecting the chemicals the stomach secretes in response to infection.

By combining an anesthetic with capsaicin, researchers can block pain in rat paws without causing temporary paralysis. This anesthetic may allow patients to be conscious during surgery and may also lead to the development of more effective chronic pain treatments [19].

Objectives

The objectives of present work were to study:

- Biological effect of chili pepper (RCP), broast chicken and nuggets on rats with peptic ulcer.
- Biological effect of RCP, broast chicken and nuggets on fasting serum glucose, lipids profile, liver and kidneys functions for hypercholesterolemic rates.
- Histopathological effects of RCP, broast chicken and nuggets on internal organs of normal and rats with peptic ulcer.

Stages of the Work

The work of the current project is divided into 2 parts

- Biological effect of RCP, broast chicken and nuggets on rats with peptic ulcer. (included in these report).
- Biological effect of RCP, broast chicken and nuggets on hypercholesterolemic rates and hypoglycemic rates. (to be done in the next stage).

The first part (on rats with peptic ulcer)

Materials

- **Plants, spicy foods:** Red chili pepper (*Capsicum frutescens*), family were purchased from local market of KSA.
- **Aspirin:** Aspegic (Mmiriya Pharmaceutical Industries, Cairo) injection was prepared by dissolving one vial in 25mL distilled water to obtain solution. A volume of 1mL of this solution was orally given (at the level 200mg/kg body weight) for one day to induce acute gastric ulcer in male albino rats.
- **Diet:** The rats were fed as ratio (a basal diet devoid from starch) composed of wheat bran, soya bean powder 44%, fish meal, molasses, fibers 3.3%, sodium chloride, calcium carbonate, calcium phosphate, methionine and ash (net protein 22% and fats 4.7%). The diet was fed and water was provided *ad libitum* for the experimental period.
- **Rats:** sixty-six adult male albino rats (170+5g B.Wt., each) of Sprague Dawley Strain were obtained from animal house of the Faculty of Medicine, Umm Al-Qura University (Makkah, KSA).

Methods

Preparation of Aqueous Extracts

The clean RCP (*Capsicum frutescens*), was ground using porcelain grinder to pass through sieve-mesh pores of 1mm diameter. The extract of red chili was prepared by mixing 1g powdered leaves with 100 mL distilled water. The mixture was boiled for 10 min. and left to cool for 15 minutes. The aqueous extract was filtered using filter paper to remove the particulate matter (0.2 mm) then the filtrate was freely dried (lyophilized) and reconstituted in 1.5 mL of distilled water (100 mg/kg body weight).

Grouping Design and Feeding of Rats

The experiment was performed in animal house of the Faculty of Medicine, Umm Al-Qura University. Rats were housed in wire cages in a room maintained at 25±2°C and kept under normal healthy conditions for two weeks. All rats were fed for one week on basal diet before starting the experiment for acclimatization. After one-week period, rats were divided into two main groups. The first group (n=6 rats) was fed on the basal diet only as a control negative (healthy rats). All rats in the second main group (n=30 rats) were given orally aspirin at a dose of 200 mg/kg B.Wt., for induction of acute gastric ulcer according to Agrawal AK, et al. (2000). Rats with (aspirin-induced gastric ulcer) were disported into five groups (n=6 rats for each group) as the following:

Group 1: Control negative -ve group was fed on basal diet (non treated rats).

Group 2: Control positive +ve group fed on basal diet basal diet+Aspirin (Asp.) 200 mg/kg B.Wt.

Group 3: positive rats fed on basal diet containing 30% spicy nuggets.

Group 4: Positive rats fed on basal diet containing 30% spicy chicken.

Group 5: Positive rats fed on basal diet+oral RCP extract at doses of 300 mg/kg B.Wt.

Group 6: Positive rats fed on basal diet+oral RCP extract at doses of 600 mg/kg B.Wt.

The rats remained without food for one day prior to ether anesthesia (except for water) to avoid mixing of food with gastric secretions.

Measurement the length of gastric ulcer: At the last day of experimental period, all rats were fasted for 12-14 hrs and only allowed for drinking water. In the morning of the next day, all rats were sacrificed, and their stomachs were tied around both openings (cardiac & pyloric sphincters) and injected by distilled water (3 mL). The gastric juice was then collected in sterilized tube. The stomachs were opened longitudinally, washed with saline and examined under dissecting microscope for ulcer. The length of gastric ulcer was measured and expressed as mean±SD for each group. The curative ratio was then calculated for each treated group according to the method described by Akhtar AH, et al. (1995) using the following equation:

- Curative ratio (CR) = (LC - LT / LC) x 100

Where: LC = length of ulcer in control positive group

LT = length of ulcer in treated group

Measurement the Volume of Gastric Juice

Gastric juice was collected according to the methods of Hiromichi N, et al. (1991). Abdomen was incised and both the stomach and duodenum were exposed and a fistula made by a poly ethane tube inserted into the stomach from a small incision made in the duodenum and held in place by a ligature around pylorus. In addition, esophagus was clamped to prevent reflux and loss of the gastric mucosa in tubes and centrifuged at 500 rpm for 5 min. The volume of gastric juice was measured by graduated cylinder and expressed as mL.

Histopathological Study

Specimens from stomachs were collected from rats of all experimental groups at the end of the experimental period, fixed in 10% neutral buffered formalin (pH=7.0), dehydrated in ethyl alcohol, then cleared in xylol and embedded in paraffin. 4-6 microns thickness sections prepared and stained with heamtoxylin and eosin for examining both for and glandular parts of the stomach [23].

Statistical Analysis

Statistical analysis has been achieved using statistical package for social science program [24].

The present study was designated to clear out the effect of aqueous extracts of RCP and some spicy foods on healing acute gastric ulcer induced by aspirin in rats. The parameters were length of gastric ulcer, volume of gastric juice, pH, and histopathological examination of fore and glandular parts of stomachs. The effect of spicy nuggets, spicy chicken and aqueous extract of RCP (*Capsium frutescens*) at two doses on the length of gastric ulcer in rats were shown. The obtained results showed that:

- A single oral administration of aspirin (200mg/kg B.Wt.) induced gastric ulcer in rats.
- The mean length of gastric ulcer in control +ve group was higher compared with in control -ve group (normal rats) being 7.27±0.093 and zero mm, respectively.
- Oral administration of RCP extract at a dose of 600 mg/kg B.Wt., for 7 days after aspirin (200 mg/kg B.Wt.) caused high decrease in the length of gastric ulcer (1.71±0.0584mm). On the other hand, the lowest decrease of gastric ulcer length was happened in rats fed on 30% spicy nuggets, which was 4.35±0.088mm.
- The curative ratios of ulcerated rats with oral administration of RCP extract at a dose of 600 and 300mg/kg B.Wt., 30% spicy chicken and 30% spicy nuggets were 76.48, 71.14, 43.19 and 40.17 respectively. The highest curative ratio obtained in a dose of 600 mg/kg B.Wt., of RCP.
- The volume of gastric juice obtained from rats given aspirin-induced gastric ulcer (control +ve group) at a dose of 200 mg/kg B.Wt., was 0.69±0.008mL (P<0.001) compared with 0.23±0.005mL in normal rats (control -ve group).
- Oral administration with RCP extracts at a dose of 600 mg/kg B.Wt. for 7 days after given aspirin (200 mg/kg B.Wt.) caused the highest decrease in the volume of gastric juice in rats, but oral administration with 30% spicy nuggets for 7 days after given aspirin (200 mg/kg B.Wt.) caused the lowest decrease in the volume of gastric juice in rats. The gradual ascending data were 0.31±0.004mL (P<0.001), 0.34±0.006mL (P<0.001), 0.39±0.010mL (P<0.001) and 0.41±0.008mL (P<0.01) for 600 and 300 mg/kg B.wt. of RCP and 30% spicy chicken and nuggets compared with +ve 0.69±0.008mL respectively.
- Effect of spicy nuggets, spicy chicken and aqueous extract of RCP (Capsium frutescens) at two doses on the pH of gastric juice collected from stomachs of rats. It is clear from data illustrated that oral administration of aspirin at a dose of 200 mg/kg B.Wt., (control +ve) decreased the pH of gastric juice (mean 3.47±0.07) (p<0.001) compared with (4.87±0.049) of normal rats (control -ve).
- Data showed that oral administration of RCP extract at dose of 600 and 300 mg/kg B.Wt., for 7 days after aspirin (200 mg/kg B.Wt.) reflected maximal value of pH level 4.82±0.061 and 4.020.060 resp. (p<0.001) compared with control (+ve). However, oral feeding of 30% spicy nuggets for 7 days after aspirin (200mg/kg B.Wt.) caused no significant decrease value (3.48±0.07).

Histopathological Results

Microscopical examination of stomach from control untreated rats revealed the normal histological structure of the stomach which consists of four layers; mucosa, submucosa, musculosa and serosa. The surface epithelial cells which are mucus secreting cells formed a continuous epithelial sheet that covered the gastric surface and line pits of gastric glands. All the cells of the surface, gastric pits and gastric glands were intact and normal without evidence of erosion or hemorrhage in the gastric mucosa. Conversely, stomach of rat from group 2 showing necrosis of the glandular mucosa associated with sloughing of laminal epithelium these superficial erosions were multiple, small and not reaching the muscularis mucosa. The sub epithelial tissues showed hemorrhage and edema with increased number of the blood vessels. There are mononuclear cells infiltrating lamina propria. Also, the stomach of this group showed destruction of gastric glands, edema in lamina propria as well as mononuclear cells infiltration. Gastric glands are reduced in number, which are abnormal in morphology and distribution. Some areas showed hyperplasic gastric glands other areas showed gastric ulceration. Covering a bier like network of hyperplasic gastric pits with destruction of upper part of gastric glands. Stomach of rat from group 3 (Asp.) +30% spicy nuggets showing destruction of gastric glands, edema in lamina propria as well as mononuclear cells infiltration. Stomach of rat from group 4 (Asp.) +30% spicy chicken showing destruction of gastric glands, edema in lamina propria as well as mononuclear cells infiltration. In Stomach of rat form group 5 (Asp.) +RCP 300 showing the normal histological structure and edema in lamina propria. There was a renewal of epithelium to line the surface of the stomach and gastric pits again, which accompanied with significant increase in the number of gastric glands. Other areas showed return of

these gastric glands to the normal pattern (Tables 1- 3).

The Second Part (on Hypercholesterolemic Rates)

Work Design

Hypercholesterolemia grouping: The first group (n=6 rats) was fed on the basal diet only, as a control negative (C-) (healthy rats). The second group (n=36 rats) rats was feed for two weeks on the basal diet plus cholesterol 1.5% to induce hypercholesterolemia before starting the experiment (Figures 1-14). After two weeks feeding rats divided into 4 subgroups as follows:

Group 1: Control positive group C+, was fed on basal diet plus cholesterol 2% (non treated rats).

Group 2: C+ rats fed on (1.5% cholesterol diet) diet containing RCP extract at doses of 150 mg/kg B.Wt.

Group 3: C+ rats fed on (1.5% cholesterol diet) diet containing RCP extract at doses of 300 mg/kg B.Wt.

Group 4: C+ rats fed on (1.5% cholesterol diet) diet containing RCP extract at doses of 600 mg/kg B.Wt.,

Group 5: C+ rats fed on basal diet + 30% chicken.

Group 6: C+ rats fed on basal diet + 30% nuggets.

Blood sampling: At the end of the experiment (45 days), rats fasted overnight and anesthetized with chloroform. Blood samples

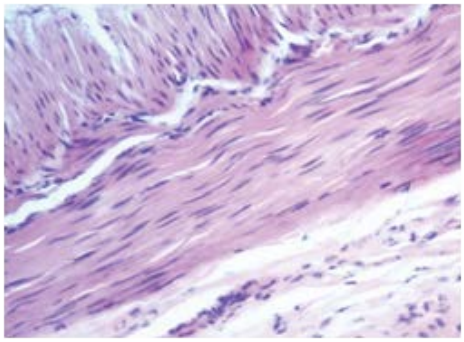

Figure 1: Ammannia baccifera (Monarch redstem) plant at Hot spring area.

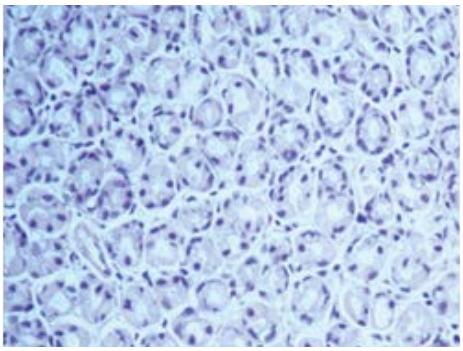

Figure 2: Stomach of rat form group 1 (control -ve) showing the normal histological structure (H & E x 200).

Table 1: Effect of spicy nuggets, spicy chicken and aqueous extract of RCP at two doses on the length of gastric ulcer in rats.

Groups		Aspirin, diet and extracts	Doses (mg/kg B.Wt.)	Gastric ulcer length (mm.)	CR (%)
				Mean±SE	
Control -ve	1	-	-	0.00***	-
Control +ve	2	Aspirin (Asp.)	200	7.27±0.093	-
Treated Groups	3	(Asp.) + 30% spicy nuggets		4.35±0.088**	40.17
	4	(Asp.) + 30% spicy chicken		4.13±0.042***	43.19
	5	(Asp.) + RCP	300	2.10±0.077***	71.14
	6	(Asp.) + RCP	600	1.71±0.0584***	76.48

CR: Curative Ratio *Differences are significant at 5% compared with +ve. **Differences are significant at 1% compared with +ve. ***Differences are significant at 0.1% compared with +ve.

Table 2: Effect of spicy nuggets, spicy chicken and aqueous extract of RCP at two doses on the volume of gastric juice collected from stomachs of rats.

Groups		Aspirin, diet, and extracts	Doses (mg/kg B.Wt.)	Volume of gastric juice (mL.)	DR (%)
				Mean±SE	
Control -ve	1	-	-	0.23±0.005***	-
Control +ve	2	Aspirin (Asp.)	200	0.69±0.008	-
Treated Groups	3	(Asp.) + 30% spicy nuggets		0.41±0.008**	40.58
	4	(Asp.) + 30% spicy chicken		0.39±0.010***	43.48
	5	(Asp.) + RCP	300	0.34±0.006***	50.72
	6	(Asp.) + RCP	600	0.31±0.004***	55.07

DR: Decrease Ratio *Differences are significant at 5% compared with +ve. **Differences are significant at 1% compared with +ve. ***Differences are significant at 0.1% compared with +ve.

Table 3: Effect of spicy nuggets, spicy chicken and aqueous extract of RCP at two doses on the pH of gastric juice collected from stomachs of rats.

Groups		Aspirin, diet, and extracts	Doses (mg/kg B.Wt.)	Volume of gastric juice (mL.)	DR (%)
				Mean±SE	
Control -ve	1	-	-	0.23±0.005***	-
Control +ve	2	Aspirin (Asp.)	200	0.69±0.008	-
Treated Groups	3	(Asp.) + 30% spicy nuggets		0.41±0.008**	40.58
	4	(Asp.) + 30% spicy chicken		0.39±0.010***	43.48
	5	(Asp.) + RCP	300	0.34±0.006***	50.72
	6	(Asp.) + RCP	600	0.31±0.004***	55.07

DR: Decrease Ratio *Differences are significant at 5% compared with +ve. **Differences are significant at 1% compared with +ve. ***Differences are significant at 0.1% compared with +ve.

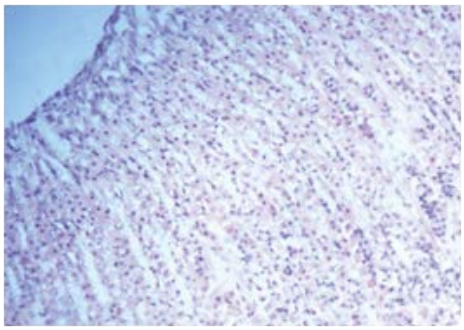

Figure 3: Stomach of rat form group 1 (control -ve) showing the normal histological structure (H & E x 200).

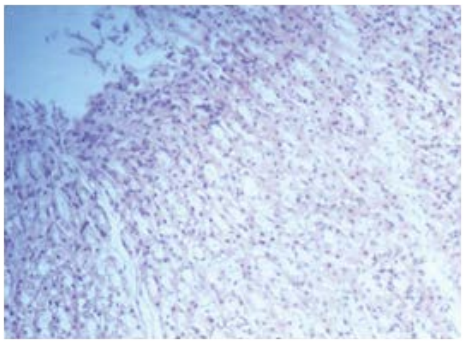

Figure 4: Stomach of rat from group 2 (control +ve) showing destruction of gastric glands, edema in lamina propria as well as mononuclear cells infiltration (H & E x 200).

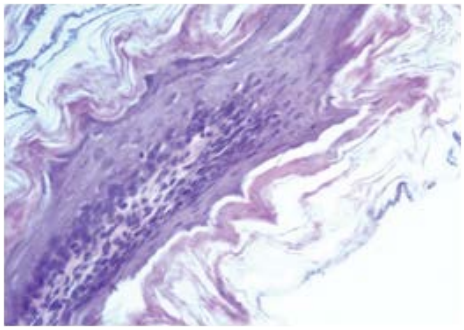

Figure 5: Stomach of rat from group 2 (control +ve) showing mononuclear cells infiltration (H and E x 200).

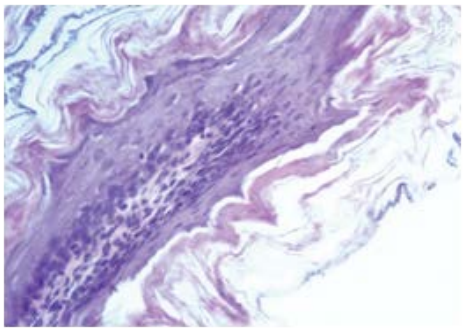

Figure 5: Stomach of rat from group 2 (control +ve) showing mononuclear cells infiltration (H and E x 200).

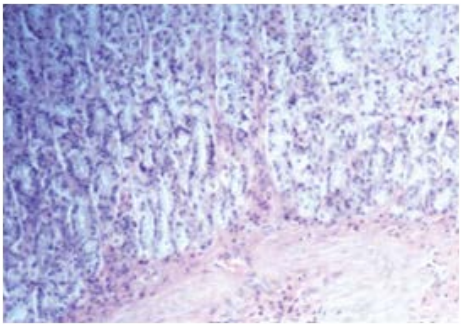

Figure 6: Stomach of rat from group 2 (control +ve) showing destruction of gastric glands and edema in lamina propria (H and E x 200).

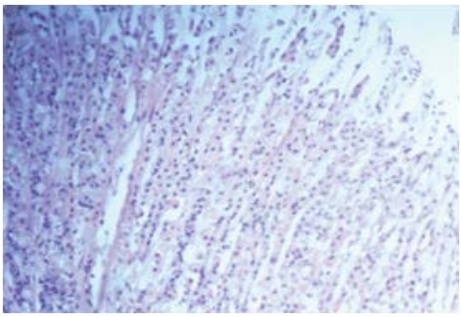

Figure 7: Stomach of rat from group 3 (Asp.) + 30% spicy nuggets showing destruction of gastric glands and edema in lamina propria (H and E x 200).

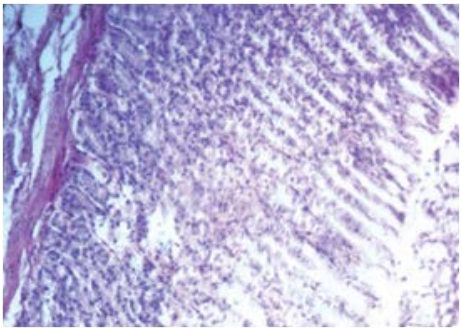

Figure 8: Stomach of rat from group 3 (Asp.) + 30% spicy nuggets showing destruction of gastric glands, edema in lamina propria as well as mononuclear cells infiltration (H and E x 200).

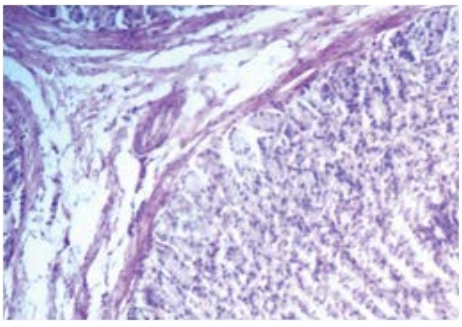

Figure 9: Stomach of rat from group 4 (Asp.) + 30% spicy chicken showing destruction of gastric glands, edema in lamina propria as well as mononuclear cells infiltration (H and E x 200).

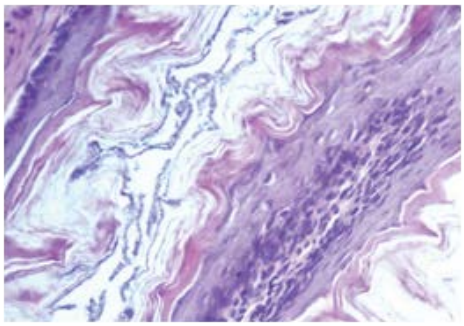

Figure 10: Stomach of rat from group 4 (Asp.) + 30% spicy chicken showing mononuclear cells infiltration (H and E x 200).

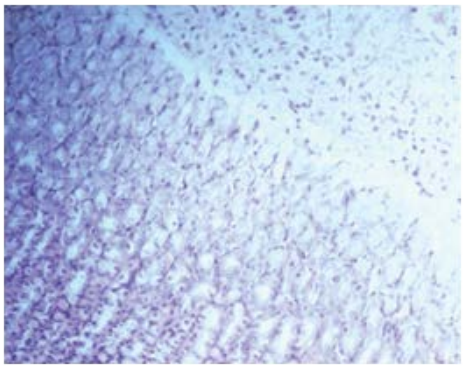

Figure 11: Stomach of rat form group 5 (Asp.) + RCP 300 showing the normal histological structure in gastric glands (H and E x200).

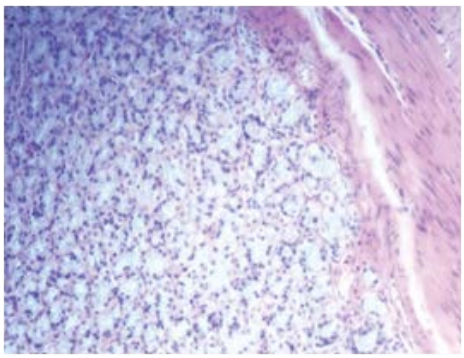

Figure 12: Stomach of rat form group 5(Asp.) + RCP 300 showing the normal histological structure and edema in lamina propria (H & E x 200).

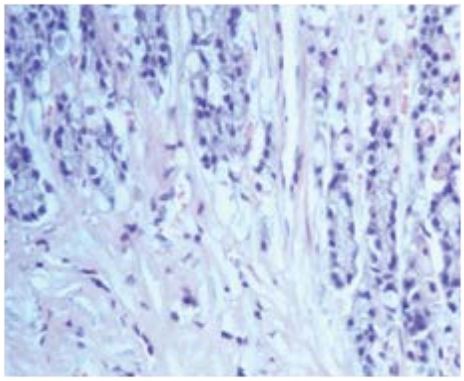

Figure 13: Stomach of rat form group 6 (Asp.) + RCP 600 showing the normal histological structure in gastric glands (H & E x200).

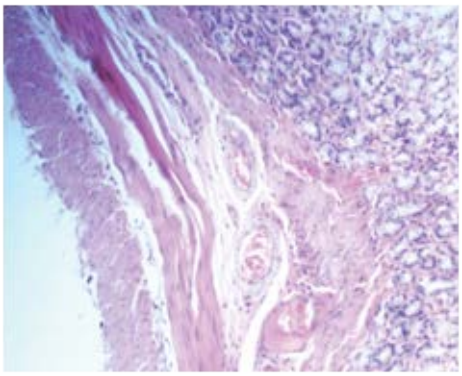

Figure 14: Stomach of rat form group 6 (Asp.) + RCP 600 showing the normal histological structure (H & E x 200).

collected be in clean dry centrifuge tubes from hepatic portal vein. Blood centrifuged for 10 minutes at 3000 rpm to separate the serum, which was kept in tubes at -18°C till analysis. Internal organs taken, washed with saline solution, and dried with filter paper, then weighted. All rats weighted once weekly. At the end of the experiment, biological evaluation of the different diets carried out by determination of body weight gain % (BWG %) and food efficiency ratio (FER) according to Chapman DG, et al. (1959) using the following formulas.

$$\text{BWG \%} = \frac{\text{Final weight} - \text{Initial weight}}{\text{Initial weight}} \times 100$$

Biochemical Analysis

Enzymatic colorimetric method was used to determine blood glucose according to Trinder P (1969). Colorimetric method was used for the determination of total cholesterol according to Allain CC, et al. (1974). Determination of HDLc were carried out according to the method of Friedewald WT (1972) and Gordon T, et al. (1977). Enzymatic colorimetric method was used to determine triglycerides according to Young D, et al. (1975). The determinations of VLDLc and LDLc were carried out according to the method of Lee R, et al. (1996) as follows: very low-density lipoprotein (VLDLc = triglycerides /5) and LDLc = (Total cholesterol – HDLc – VLDLc). Colorimetric method was used to determine AST and ALT according to Reitman S, et al. (1957); while determination of alkaline phosphates ALP activity according to Haussament TU (1977).

Results of the Second Part

Effect of RCP on Organs Weight to Body Weight Ratio

Data listed in Table 4 shows effect of RCP on organs weight to body weight ratio for control positive and different groups of hypercholesterolemic rats [34,38]. It could be noticed that the kidney and liver ratio were significant increase ($P<0.05$) for C +ve group rats compared with normal rats. These findings showed that there was significant increase ($P<0.05$) for liver weight to body weight ratio in control positive as compared to control negative group [39-41]. Hypercholesterolemic rats and fed on RCP 150 mg/kg B.Wt. and 30% nuggets showed significant ($P<0.05$) changes in liver weight to body weight ratio when compared to control positive [42].

$$\text{FER} = \frac{\text{Gain in body weight (g)}}{\text{Food Intake (g)}}$$

Table 4: Effect of RCP on organs weight to body weight ratio for control positive and different groups of hypercholesterolemic rats.

Groups Organs	Control (-)	Control (+)	RCP 150 mg/kg B.Wt.	RCP 300 mg/kg B.Wt.	RCP 600 mg/kg B.Wt	30% chicken	30% nuggets
Kidney	0.61±0.02*	0.69±0.01	0.65±0.03	0.71±0.05	0.64±0.02	0.69±0.08	0.72±0.02
Lungs	0.62±0.03	0.67±0.05	0.63±0.03	0.72±0.09	0.56±0.02	0.55±0.45	0.61±0.02
Spleen	0.26±0.01	0.26±0.01	0.28±0.02	0.3±0.04	0.33±0.04	0.9±0.49	0.39±0.03*
Liver	2.19±0.1*	2.76±0.08	3.41±18*	3.47±0.27	2.71±0.08	2.8±0.32	2.9±0.1*
Heart	0.35±0.02	0.36±0.01	0.28±0.02*	0.34±0.03	0.36±0.18	0.38±0.04	0.39±0.05
*Differences are significant at 5% (p<0.05). **Differences are significant at 1% (p<0.01). ***Differences are significant at 0.1% (p<0.001).							
RCP: red chills pepper extract.							

Table 5: Effect of RCP on lipid profile (mg/dl) for control positive and different groups of hypercholesterolemic rats.

Groups Parameters	Control (-)	Control (+)	RCP 150mg/kg B.Wt.	RCP 300 mg/kg B.Wt.	RCP 600 mg/kg B.Wt	30% Chicken	30% Nuggets
Triglycerides	29.7±2.9**	217.6±36.9	192.6±28.3	198.3±7.09	195.1±14.4	185.1±41.1	226.6±59.4
Cholesterol	84.4±5. 6**	195.75±9.1	178.9±16.9	182.4±16.9	166.5±22.8	197.3±6.42	206.6±11.9
HDL (mg/dl)	57.0±6.25*	43.58±9.24	51.4±6.3**	57.96±14.4	67.15±12.5	78.29±12.3	44.9±4.5**
VLDL (mg/dl)	5.94±0. 8**	43.52±7.67	38.52±17.9	39.7±2.9.4	39.02±2.87	37.1±8.2	45.32±11.9
LDL (mg/dl)	21.54±4.6*	108.65±7.4	88.9±5.65	84.74±14.2	60.32±16.1	81.91±11.2	116.4±23.1
*Differences are significant at 5% (p<0.05). **Differences are significant at 1% (p<0.01). ***Differences are significant at 0.1% (p<0.001).							
RCP: red chills pepper extract.							

Table 6: Atherogenic indices of for control positive and different groups of hypercholesterolemic rats.

Groups Parameters	Control (-)	Control (+)	RCP 150mg/kg B.Wt.	RCP 300 mg/kg B.Wt.	RCP 600 mg/kg B.Wt	30% Chicken	30% Nuggets
LDL/HDL ratio	0.39±0.06**	2.49±0.097	1.73±0.189	1046±0.22	0.89±0.12*	1.05±0.2	2.59±0.24
T.C/LDL ratio	3.92±1.82*	1.81±0.823	2.01±0.575	2.15±0.33	2.66±0.57	2.41±2.69	1.77±1.1
T.C/HDL ratio	1.48±0.24**	4.45±0.189	3.48±0.91	3.14±1.35	2.47±0.39	2.5±0.53	4.6±0.71
*Differences are significant at 5% (p<0.05). **Differences are significant at 1% (p<0.01). ***Differences are significant at 0.1% (p<0.001).							
RCP: red chills pepper extract							

Table 7: Effect of RCP on serum glucose (mg/dl) for control positive and different groups of hypercholesterolemic rats.

Groups	Control (-)	Control (+)	RCP 150mg/kg B.Wt.	RCP 300 mg/kg B.Wt.	RCP 600 mg/kg B.Wt	30% chicken	30% nuggets
Fasting Serum glucose	95.2±9.1*	114.8±25.2	110.5±10.8	102.5±18.2	107.9±19.2	112.2±33.7	123.8±26.2*

Effect of RCP on Lipid Profile (mg/dl)

Table 5 shows that, the effect of RCP on lipid profile (mg/dl) for control positive and different groups of hypercholesterolemic rats. The results revealed that there was a significant decrease in lipid profile for control negative compared with control positive. Meanwhile, there was a significant decrease (P<0.01) in HDL of experimental animals fed on RCP 150 mg/kg B.Wt. and 30% nuggets when compared to positive group [43-48].

Effect of Red Chills Pepper Extract on Atherogenic Indices for Hypercholesterolemic Rats

Atherogenic indices of control positive and different groups of hypercholesterolemic rats fed on RCP, broast chicken and nuggets were recorded (Table 6). Hypercholesterolemic rats (control positive groups) showed significant increase (P<0.01) in LDL/HDL ratio, while showed significant decrease (P<0.05) in T.C/LDL ratio and showed high significant increase (P<0.01) in TC/HDL ratio when compared to normal rats. In LDL/HDL ratio there were significant decrease (P<0.05) in RCP 600 mg/kg B.Wt while showed non-significant increase in TC/LDL ratio. At the same time, there were non-significant changes in breast chicken and nuggets when compared to control positive groups in neither LDL/HDL nor T.C/LDL ratio [49,50].

Effect of RCP on Serum Glucose (mg/dl)

Data in the Table 7 show the effect of RCP on serum glucose (mg/dl) for control positive and different groups of hypercholesterolemic rats. It is evident that the RCP 150, 300 and 600 mg /kg B.Wt. showed very slight decrease of the level of serum glucose. The mean values were (110.5, 102.5 and 107.9 mg/dl) respectively. Consequently, there were no significant difference in RCP 150, 300 and 600 mg /kg B.Wt. when compared to control positive. It was found that significant increase (P<0.05) of erum glucose could be noticed in the 30% nuggets treatment (123.8 mg/dl) compared to control positive group and the lower in 112.2 treatment (112.2 mg/dl) (Figures 15-18).

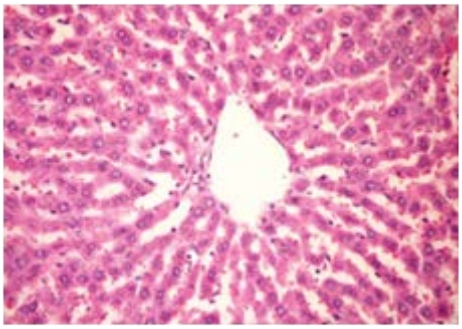

Figure 15: Liver of rat from control (–) showing the normal histological structure of hepatic lobule (H and E x 400).

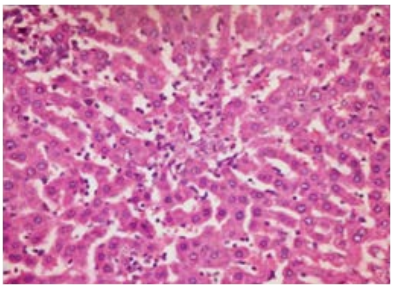

Figure 16: Liver of rat from control (+) showing sinusoidal leucocytosis (H and E x 400).

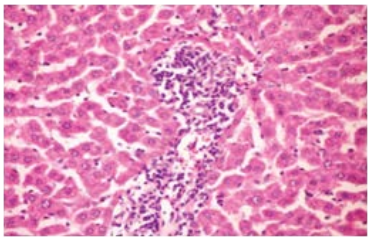

Figure 17: Liver of rat from control (+) showing portal infiltration with leucocytes (H and E x 400).

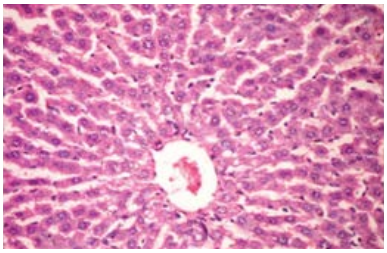

Figure 18: Liver of hypercholesterolemic rats from group (RCP 150 mg/kg B.Wt.) showing vacuolization of centrobular hepatocytes (H and E x 400).

Histopathological Results

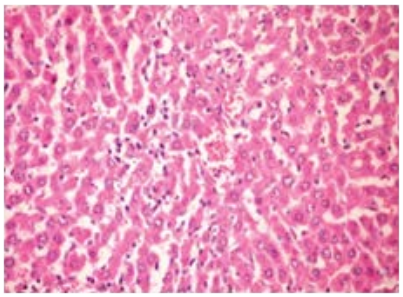

Figure 19: Liver of hypercholesterolemic rats from group (RCP 300 mg/kg B.Wt.) showing kupffer cells activation (H and E x 400).

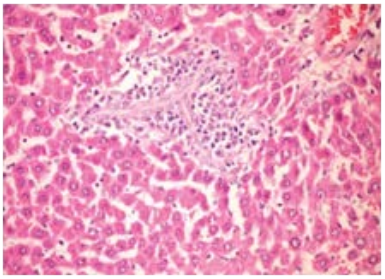

Figure 20: Liver of rat from group (RCP 600 mg/kg B.Wt.) showing portal infiltration with leucocytes (H and E x 400).

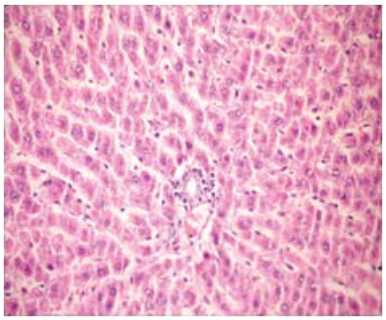

Figure 21: Liver of hypercholestrolemic rats fed on 30% chicken revealed kupffer cells activation, vacuolization of hepatocytes (H and E x 400).

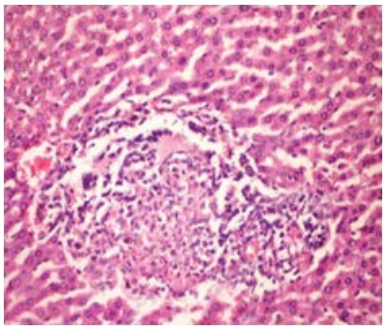

Figure 22: Liver of hypercholesterolemic rats from group (30% nuggets) showing focal hepatic necrosis associated with inflammatory cells infiltration (H and E x 400).

Recommendation

- Nutritional and health educational programs should be organized and directed for the public to protect themselves from peptic ulcer and its complications.
- Spices and herbs have an effective ingredient, which can protect from ulcers, so we should add them to the meal as it is possible.
- Patients with ulcers can use red chili pepper for healing the disease, which might be used as a medicine herbs or add to food with the same percentage.
- The plants had been experimented for rats can also be used for patients with ulcer in hospitals and evaluate their nutritional states.
- Further studies should be carried out to investigate the influence of red chili pepper on their ability in correcting peptic ulcer.
- Restrict the consumption of spice chicken nuggets and spice chicken prost due to high serum cholesterol, triglycerides, LDL, and VLDL.
- While no restriction of chicken products to healthy people is not inevitable, they are advised to consume only reasonable amounts at reasonable frequencies, to avoid at least the risk of obesity, taking into consideration that many fast foods are high fat, high calories, high cholesterol and of high NaCl content.

References

1. Perry L, Dickau R, Zarrillo S, Holst I, Pearsall DM, et al. (2007) Starch fossils and the domestication and dispersal of chili peppers (Capsicum spp. L.) in the Americas. Sci 315: 986-988.
2. Bak-Romaniszyn L, Wojtuń S, Gil J, Płaneta-Małecka I (2004) Peptic ulcer disease etiology, diagnosis is and treatment. J Pop Merkuriuz Lek 17: 128-132.
3. Lee IO, Lee KH, Pyo JH, Kim JH, Choi YJ, et al. (2007) Anti-inflammatory effect

of capsaicin in Helicobacter pylori-infected gastric epithelial cells. Helicobacter 12: 510-517.

4. Satyanarayana MN (2006) Capsaicin and gastric ulcers. Crit Rev Food Sci Nutr 46: 275-328.
5. O'Mahony R, Al-Khtheeri H, Weerasekera D, Fernando N, Vaira D, et al. (2005) Bactericidal and anti-adhesive properties of culinary and medicinal plants against Helicobacter pylori. World J Gastroenterol 11: 7499-7507.
6. Allen R, Myers MD (1986) Medicine: The national medical series for independent study. John Wiley and Sons Inc., United States.
7. Myers BM, Smith JL, Graham DY (1987) Effect of red chili pepper and black pepper on the stomach. J Gastroenterol 82: 211-214.
8. Kang JY, Alexander B, Barker F, Man WK, Williamson RC (1992) The effect of chili ingestion on gastrio-intestinal mucosal proliferation and azoxymethan-induced cancer in rat. J Gastro-enterol Hepatol 7: 194-198.
9. Kang JY, Teng CH, Wee A, Chen FC (1995) Effect of capsaicin and chili on ethanol induced gastric mucosal injury in the rat. J Gut 36: 664-669.
10. Yeoh KG, Kang JY, Yap IE, Guan R, Tan CC (1995) Chili protects against aspirin-induced gastro-duodenal mucosal injury in humans. J Dig Dis Sci 40: 580-583.
11. Cruz L, Castañeda-Hernández G, Navarrete A (1999) Ingestion of chili pepper (Capsicum annuum) reduces salicylate bio-availability after aspirin administration in the rat. J Physiol Pharmacol 77: 441-446.
12. Ahuja KD, Ball MJ (2006) Effects of daily ingestion of chili on serum lipoprotein oxidation in adult men and women. Br J Nutr 96: 239-242.
13. Alasdair GT, Peters M, Robin C (1981) Pathology Illustrated. Sheck Wah Tong Printing Press Ltd., Hong Kong, People's Republic of China.
14. Vasudevan K, Vembar S, Veeraraghavan K, Haranath PS (2000) Influence of intra-gastric perfusion of aqueous spice extracts on acid secretion in anesthetized albino rats. Indian J Gastroenterol 19: 53-56.
15. Ononiwu IM, Ibeneme CE, Ebong OO (2002) Effect of Piperine on gastrisuec acid secretion in albino rats. Afr J Med Med Sci 31: 293-295.
16. Athanasiou A, Smith PA, Vakilpour S, Kumaran NM, Turner AE, et al. (2007) Vanilloid receptor agonists and antagonists are mitochondrial inhibitors: how vanilloids cause non-vanilloid receptor mediated cell death. Biochem Biophys Res Commun 354: 50-55.
17. Hsu CL, Yen GC (2007) Effects of capsaicin on induction of apoptosis and inhibition of adipogenesis in 3T3-L1 cells. J Agric Food Chem 55: 1730-1736.
18. Razavi R, Chan Y, Afifiyan FN, Liu XJ, Wan X, et al. (2006) TRPV1+ sensory neurons control beta cell stress and islet inflammation in autoimmune diabetes". Cell 127: 1123-1135.
19. Binshtok AM, Bean BP, Woolf CJ (2007) Inhibition of nociceptors by TRPV1-mediated entry of impermeant sodium channel blockers. Nat 449: 607-610.
20. Agrawal AK, Rao CV, Sairam K, Joshi VK, Goel RK (2000) Effect of Piper longum linn, Zingiber officianails linn and Ferula species on gastric ulceration and secretion in rats. J of Exp Biol 38: 994-998.
21. Akhtar AH, Ahmad KU (1995) Anti-ulcerogenic evaluation of the methanolic extracts of some indigenous medicinal plants in Pakistan on aspirin-ulcerated rats. J Ethnopharmacol 46: 1-6.
22. Hiromichi N, Koji T, Susumu O (1991) Role of thyrotropin releasing hormone in acid secretory res response in rats. Eur J Pharmacol 198:137-142.
23. Carleton H (1976) Histopathological technique. (4th Edtn), Oxford University Press, London, United Kingdom.
24. SPSS (2008). Statistical Package for Social Science, Computer Software, IBM, SPSS Ver. 16.0 in 2008., SPSS Company, London, United Kingdom.
25. Chapman DG, Castillo R, Campbell JA (1959) Evaluation of protein in food: A method for the determination of protein efficiency ratio. Can J Biochem Physiol 37: 679-686.
26. Trinder P (1969) Determination of triglycerides. Ann Clin Biochem 6: 24-27.
27. Allain CC, Poon LS, Chan CS, Richmond WF, Fu PC (1974) Cholesterol enzymatic colorimetric method. J Clin Chem 20: 470.
28. Friedewald WT (1972) Determination of HDL. Clin Chem 18: 499.
29. Gordon T, Amer M (1977) Determination of HDL. J Med 62: 707.
30. Young D, Pestaner L (1975) Determination of triglycerides. J Clin Chem 21-25.
31. Lee R, Nieman D (1996) Nutritional Assessment. (2nd edtn), Mosby, USA.
32. Reitman S, Frankel S (1957) Determination of serum glutamic oxaloacetic transaminase and serum pyruvic transaminase. Am J Chem Path 28: 56-63.
33. Haussament TU (1977) Determination of alkaline phosphatase. Clin Chem Acta 35: 271-273.
34. Abdel-Salam OM, Szolcsanyi J, Mózsik G (1997) Capsaicin and the stomach. A review of experimental and clinical data. J Physiol Paris 91: 151-171.
35. Aguwa CN, Mittal GC (1981) Study of anti-ulcer activity of aqueous extract of leaves of (Pyrenacontha staudtii) family Icacinaceae, using various models of experimental gastric ulcer in rats. Eur J Pharmacol 74: 215-219.
36. Akah PA, Orisakwe OE, Gamaniel KS, Shittu A (1998) Evaluation of Nigerian traditional medicine: II. Effects of some Nigerian folk remedies on peptic ulcer. J Ethnopharmacol 62: 123-127.
37. Baĭbekov IM, Mavlian-Khodzhaev R (1986) Morphological research on the effect of vagotomy on the parietal micro-flora of the stomach and duodenum. J Biull EKP Biol Med 101: 496-498.
38. Bombik E, Bombik T, Saba L (2002) The effect of herb extracts on the level of selected micro-elements in blood serum of calves. J Biuletyn Informacyjny Instytut Zootechniki 40: 279-285.
39. Hung CR, Neu SL (1997) Acid-induced gastric damage in rats is aggravated by starvation and prevented by several nutrients. J Nutr 127: 630-636.
40. Khayyal MT, El-Ghazaly MA, Kenawy SA, Seif-El-Nasr M, Mahran LG, et al. (2001) Anti-ulcerogenic effect of some gastrio-intestinally acting plant extracts and their combination. J Arzneimittelforschung 51: 545-553.
41. Limlomwongse L, Chaitauchawong C, Tongyai S (1979) Effect of capsaicin on gastric acid secretion and mucosal blood flow in the rat. J Nutr 109: 773-777.
42. De B, Maiti RN, Joshi VK, Agrawal VK, Goel RK (1997) Effect of some sitavirya plants on gastric secretion and ulceration. Indian J Exp Biol 35: 1084-1087.
43. Mitra SK, Gopumadhavan S, Hemavathi TS, Muralidhar TS, Venkataranganna MV (1996) Protective effect of UL-409, a herbal formulation against physical and chemical factor induced gastric and duodenal ulcers in experimental animals. J Ethnopharmacol 52: 165-169.
44. Rao CV, Sairam K, Goel RK (2000) Experimental evaluation Bocopa Monniera on rats gastric ulceration and secretion. Indian J Physiol Pharmacol 44: 435-441.
45. Rees WD, Rhodes J, Wright JE, Stamford IF, Bennett A (1979) Effect of deglycyrrhizinated liquorice on gastric mucosal damage by aspirin. J Gastroenterol 14: 605-607.
46. Sairam K, Rao CV, Goel RK (2001) Effect of Convolvulus pluricaulis chais on gastric ulceration and secretion in rats. Indian J Exp Biol 39: 350-354.
47. Sen T, Basu A, Chaudhuri AN (1996) Studies on the possible mechanism of the gastric mucosal protection by calotropis procera-involvement of 5-lipoxygenase pathway. J Clin Pharmacol 12: 82-87.
48. Shobana S, Naidu KA (2000) Anti-oxidant activity of selected Indian spices. Indian J Prostaglandins Leukot Essent Fatty Acids 62: 107-110.
49. Standard Methods for the Examination of Dairy Products (1960). American health Association pub.
50. Zeiny AR, Shalaby AO, Zemzemi FM, Awad OA (1995) Basic Nutrition and Diet Therapy. (10th edtn), United States.