ANTI - ULCER ACTIVITY OF *Ocimum sanctum* (Thulasi) *Musa* (AAB Group, "Nendran") AND *Withania somnifera* (Amukkiram) IN RATS

By

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THESIS

Submitted in partial fulfilment of the requirement for the degree of

Master of Veterinary Science

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DECLARATION

I hereby declare that this thesis entitled "**Anti-ulcer activity of *Ocimum sanctum* (Thulasi), *Musa* (AAB group "Nendran") and *Withania somnifera* (Amukkiram) in rats**" is a bonafide record of research work done by me during the course of research and that this thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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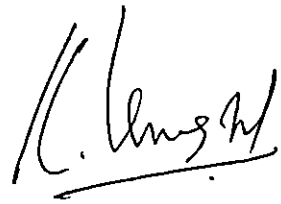
Certified that the thesis, entitled "**Anti-ulcer activity of *Ocimum sanctum* (Thulasi), *Musa* (AAB group "Nendran") and *Withania somnifera* (Amukkiram) in rats**" is a record of research work done independently by **Sri. D. Sanjay**, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.



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D. SANJAY

Dedicated To My Parents

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Introduction

INTRODUCTION

Peptic ulcer is a common condition which kills few but troubles many and has been estimated to affect about 10 per cent of the human population (of the western world). A mucosal interruption in the stomach and/or duodenum is called a peptic ulcer, only if it is deep enough to penetrate through the entire mucosa. Any lesser penetration is called an erosion. Grossly, an ulcer is a discontinuity of mucosal surface with an inflammed base.

A peptic ulcer develops as a result of localised areas of necrosis and digestion of the lining of the digestive tract. The process is a penetrating one, beginning in the mucosa and gradually extending through the muscularis mucosa in to or through the muscularis propria. In some cases the ulcer penetratesinto blood vessels resulting in haemorrhage or completely through the gut wall into adjacent organs or as a free perforation into the peritoneal cavity. Healing starts from below and extends upward with the growth of granulation tissue and fibroblast. In small superficial lesion, healing is complete. In large chronic ulcer healing is slower, new glands are not formed and tissue is replaced by fibrous and elastic tissue.

Clinically chronic peptic ulcer occur only in those portions of the digestive tract exposed to the action of acid juice, the lower portion of the esophagus, the stomach, the upper portion of the small intestine or the small bowel adjacent to a patent gastroenterostomy or a Meckel's diverticulum containing ectopic gastric glands. The majority of peptic ulcers however occur along the lesser curvature of the stomach and in the first three to four centimeters of the duodenum.

It was observed that in 45-70 per cent patients with duodenal ulcers, acid secretion was within normal limits whereas in gastric ulcer patients acid secretion was either normal or even subnormal. It was therefore apparent that peptic ulceration was not solely induced by the offensive factors of acid and pepsin but by break down of mucosal defence mechanisms such as mucus-bicarbonate barrier, endogenous prostaglandins, epithelial protection, mucosal blood flow and sulfhydryl compounds (Rosa *et al.*, 1991).

Many commonly used drugs are ulcerogenic of which the most important drugs are the non-steroidal anti-inflammatory agents such as aspirin, flunixin, phenylbutazone, indomethacin, naproxen, etc. Most of these drugs exert an antiprostaglandin effect by blocking prostaglandin cyclooxygenase activity. Corticosteroids appear to play a

causative role in gastric ulceration. Corticosteroids decrease mucosal cell turnover and mucus production and increase gastrin level and thus gastric acid output. Corticosteroids alone has very less chance of producing gastric ulceration but they have been shown to enhance the ulcerogenic effect of non-steroidal anti-inflammatory agents significantly. Other agents such as alcohol, hypertonic agents, certain detergents and heavy metals can also cause gastric lesions (Schimmer and Parker, 1996).

Conditions of stress can result in acute erosion of the gastric mucosa. The precise mechanism is unknown but current evidence indicates that stress impairs epithelial cell replication of gastric mucosa.

Neurologic disease has been associated with the formation of gastric ulceration in both man and animals.

Metabolic disease frequently result in secondary gastric ulceration. In renal failure, uraemic toxins directly injure the gastric mucosa and vessels of the gastric wall. The gastro-intestinal hormone, gastrin is metabolised in kidney, in turn increases gastric acid output resulting in mucosal damage. Gastric ulceration secondary to liver failure occur due to decreased mucosal blood flow associated with portal hypotension, vascular shunting and altered mucosal renewal due hypoproteinemia and elevated histamine concentration causing

excess acid production. Gastric lesions have also been observed in some animals with adrenocortical insufficiency possibly resulting from hypotension and abnormal vascular tone (Strombeck and Guilford, 1991).

Tumours such as mastocytoma and gastrinoma are frequently associated with gastric ulceration.

Helicobacter pylori, a cytopathogenic bacteria is distributed globally in 25-50 per cent of normal population. The eradication of this organism can effectively reduce ulcer recurrence (Goel and Bhattacharya, 1991).

Research advances during the last few years have offered new insights in the therapy and prevention of gastro-duodenal ulceration by measures directed at neutralising acid-pepsin and by strengthening the mucosal defense system also.

Approach for the treatment of peptic ulcers are:

1. Neutralisation of gastric acid by systemic and non-systemic antacids.
2. Reduction of gastric acid secretion by H_2 receptor antagonist, anticholinergics, proton pump inhibitors and prostaglandin analogues
3. Ulcer protectives
4. Ulcer healing drugs.

Global estimate indicate that 80 per cent of total population cannot afford the products of the western pharmaceutical industry and have to rely upon the use of traditional medicines, which are mainly derived from plant material.

As the modern life saving drugs are beyond the reach of more than three quarter of the third world population, use of herbal drugs remain as a viable alternative for the future.

India is well known for the use of medicinal plants. Besides the vedas, the ancient scholars like Charaka, Susruta, Vaghbatta and others brought out texts containing the description of various plants used in several preparation for the treatment of various diseases.

Anti-ulcer effect of *Ocimum sanctum* leaves has been reported in rats (Mandal *et al.*, 1993). Similar effect of *Withania somnifera* was also reported in rats (Sahni and Srivastava, 1993). The anti-ulcerogenic activity of the unripe dried plantain banana, *Musa paradisiaca* was reported in rats by Best *et al.* (1984).

In the present study an attempt has been made to evaluate the anti-ulcer potentialities of powder and alcoholic extract of *Ocimum sanctum* leaves, *Withania somnifera* root and *Musa* (AAB group "Nendran") unripe fruit at various dose levels and durations in aspirin induced gastric ulcer in adult albino rats of either sex.

Review of Literature

REVIEW OF LITERATURE

2.1 Ulcer induction

Hemmati *et al.* (1973) induced gastric ulcers in rats using drugs such as aspirin at 200 mg/kg single oral dose, phenyl butazone at 100 mg/kg single oral dose, indomethacin at 10 mg/kg in two doses, ibuprofen at 200 mg/kg in two doses and single injection of reserpine at 5 mg/kg 1/m.

Manekar and Waghmare (1980) reported that several factors like, increase in the serotonin level, damage to the mucosal barrier, changes in mucosal permeability and mucosal blood flow and inhibition of prostaglandin synthesis could be responsible individually or collectively for aspirin induced gastric damage. It was also found that rabbits, guinea-pigs and rats were sensitive to aspirin induced gastric ulceration in a decreasing order.

Gupta *et al.* (1988) had deviced an arbitrary scoring system for grading the severity of gastric lesions, (i) denuded epithelium = 10 (ii) petechial and frank haemorrhages = 20; (iii) one or two ulcers = 30; (iv) multiple ulcers = 40; (v) perforated ulcers = 50. The severity of ulceration was expressed in terms of ulcer index which was the mean score of all the animals in a group.

Goel *et al.* (1990) has induced gastric ulcers by giving 200 mg/kg of aspirin orally for 5 days.

2.2 Famotidine

Famotidine is a H_2 receptor antagonist used as an anti-ulcer drug. The antiulcer activity of famotidine is attributed not only to suppression of acid secretion but also to activation of the gastric mucosal defensive mechanism (Miyata *et al.*, 1991).

2.3 *Ocimum sanctum*

O. sanctum is a small herb found throughout India. All parts are used for medicinal purposes (Nadkarni, 1954). In Ayurvedic system of medicine, the juice and decoction of the *Ocimum sanctum* leaves are used as diaphoretic and antipyretic. The leaves possess expectorant properties and their juice is used in catarrh and bronchitis (Kirtikar and Basu, 1935).

Chopra *et al.* (1958) has reported that the *O. sanctum* leaves yield 0.7 per cent essential oil containing 71.3 per cent eugenol, 3.2 per cent carvacrol, 20.4 per cent methyl eugenol and 1.7 per cent caryophyllene.

Besides the volatile oils, the plant is also reported to contain alkaloids, glycosides and tannins. The leaves contain

ascorbic acid (83 mg/100 mg of leaves) and carotene (2.5 mg/100 gm of leaves). An infusion of the leaves is used as a stomachic in gastric disorders in children (The Wealth of India, 1966).

A study conducted by Bhargava and Singh (1981) found that the alcoholic extract of whole plant of *Ocimum sanctum* increased the physical endurance of swimming in mice and prevented stress induced ulcers in rats at a dose level of 100 mg/kg given intraperitoneally one hour before the experiment. He also found that the alcoholic extract of whole plant prevented aspirin induced gastric ulcer at the dose rate of 100 mg/kg intraperitoneally.

According to the observations made by Seethalekshmi *et al.* (1982) *O. sanctum* enhanced the physical endurance in mice and prevented stress induced gastric ulcers in rats.

Mediratta *et al.* (1988) studied the effect of steam distilled extract of *O. sanctum* leaves on the humoral immune response in experimental animals. The study clearly suggested that *O. sanctum* modulate the humoral immune response by acting at various level in the immune mechanism such as antibody production, release of mediators of hyper sensitivity reaction and tissue response to these mediators on the target organs. He also found that *O. sanctum* significantly inhibited antigen induced histamine release from the peritoneal mast cells of

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sensitised rats *in vitro*. The *O. sanctum* also antagonised the effects of various spasmogens viz., histamine, serotonin and acetyl choline on the isolated guinea pig ileum.

Akhtar and Munir (1989) found that the *Ocimum basilicum* powder and its aqueous and methanolic extracts decreased the ulcer index. Moreover the acid output was also decreased by its methanolic extract while hexosamine secretion was enhanced.

Singh and Agrawal (1991) found that fixed oil from *O. sanctum* leaves inhibit aspirin, indomethacin and alcohol-induced ulceration which could be due to leukotriene antagonistic activity.

Akhtar *et al.* (1992) studied the anti-ulcerogenic effects of different type of extracts, volatile oils and flavanoid glycoside of leaves of *O. basilicum* in normal and ulcer induced rats. He found that the aqueous, methanol and water-methanol extracts and flavanoid glycoside reduced the ulcer index, inhibited gastric acid and pepsin secretion and enhanced hexosamine in aspirin treated rats. He also found that volatile oils of the plant were ineffective against stress induced ulcers.

Mandal *et al.* (1993) found that *O. sanctum* leaves alcoholic extract at the dose rate of 100 mg/kg orally has

anti-ulcerogenic property against experimental ulcers and it is due to its ability to reduce acid secretion and increase mucous secretion. It was also evident from the study that *O. sanctum* required a few days of pretreatment to increase mucus secretion, whereas its effect on acid secretion was almost immediate.

"Zeetress", an anti-stress poly-herbal ayurvedic formulation (M/s Indian herbs), contains chiefly the extracts of *O. sanctum* and *W. somnifera*, has been found to decrease the incidence, number and severity of gastric ulcers induced by cold-immobilisation stress (Bhattacharya and Ghosal, 1994).

Singh and Majumdar (1995) found that fixed oil of *O. sanctum* seeds possessed significant anti-ulcer activity. Anti-ulcer activity against histamine, reserpine and stress induced ulceration and anti-secretory activity of the oil could be attributed to its histamine antagonistic and anticholinergic effects.

Vanisree *et al.* (1995) studied the effect of pretreatment with a suspension of powdered *O. sanctum* leaves at the dose rate of 200 mg/kg orally for 8 days on hydrochloric acid ethanol induced gastric lesion in rats. An increase in volume and acidity of the gastric juice, decrease in peptic activity, increase in lipid peroxidation, decrease in activity of antioxidants and decrease in protein and glycoprotein

contents which occurred in ulcerated mucosa, was not seen in the *O. sanctum* treated experimental group.

2.4 *Withania somnifera*

Withania somnifera is a shrub found in different parts of India. In Ayurvedic medicine the root is regarded as tonic, alterative and aphrodisiac and is used in different ailments both in children and aged patients. The ground root and bruished leaves employed as a local application to treat carbuncles, ulcers and painful swelling (Kirtikar and Basu, 1935).

Root and leaves are used as a hypnotic and anti-helminthic. The fruits and seeds are used as diuretic (Nadkarni, 1954).

The hypnotic and sedative properties of *W. somnifera* is due to the presence of an alkaloid *Somniferine*. Root contain traces of essential oil. This plant also contain a reducing sugar, phytosterol, ipuranol, mixture of saturated and unsaturated fatty acids (Chopra *et al.*, 1958).

The anti-ulcerogenic activity of *W. somnifera* has been reported by C.S.I.R. (1986).

Bhattacharya *et al.* (1987) also indicated the anti-ulcerogenic effect of *W. somnifera* on restrained stress-induced gastric ulcers in rats, where it produced a significant reduction in number of ulcers per stomach from 10.6 ± 1.9 to 1.2 ± 0.9 and ulcer index from 46.0 to 11.6.

W. somnifera powder at the oral dose of 500 and 1000 mg/kg produced dose-dependent significant anti-ulcerogenic effect on aspirin (200 mg/kg oral) induced gastric ulcer in albino rats. *W. somnifera* also reduced the ulcer index of aspirin from 22.04 to 17.34 and produced the healing index (% improvement) of 21.32 and 42.37 per cent at the dose of 500 and 1000 mg/kg respectively (Sahni and Srivastava, 1993).

Anti-inflammatory and anti-ulcerogenic activity of *W. somnifera* was evaluated in rats. *W. somnifera* at 1 gm/kg oral produced significant anti-inflammatory activity of 60.09 per cent at 2nd hour of its administration against carrageenin induced paw edema in rats. Promethazine enhanced the anti-inflammatory activity of *W. somnifera* by 54.75 per cent at first hour of treatment and cyproheptadine potentiated its anti-inflammatory activity by 63.27 per cent at 22nd hour of treatment. *W. somnifera* at the dose of rate of 1 gm/kg body weight orally produced protective effect on aspirin (200 mg/kg) induced gastric ulcers in rats (Sahni and Srivastava, 1994).

Kiran Nashine *et al.* (1995) observed that the aqueous extract of *W. somnifera* exhibits its anti-inflammatory activity on carrageenin induced rat paw edema by blocking histamine H_1 and H_2 receptors, 5-HT receptors in early phase and inhibition of prostaglandin synthesis in delayed phase of inflammation.

Two withanolides, withaferin A and withanone, both steroidal lactones, have been isolated and purified from the alcoholic extract of root. Withaferin A showed anti tumor properties (Umadevi, 1996).

2.5 *Musa* (AAB group) "Nendran"

Musa sapientum is a herb with thick stems cultivated throughout India and in other tropical countries. The unripe fruit is acrid, cooling, tonic, astringent to the bowels. The ripe fruit is sweet, acrid, tonic, aphrodisiac and increases appetite (Kirtikar and Basu, 1935).

Musa sapientum or *M. paradisiaca* contains about 37 per cent of dry matter. Growing parts of the plant contain much tannic and gallic acid. The Ripe fruit contains 22 per cent sugar, starch, albuminoids, fats, non-nitrogenous extracts and ash containing phosphoric anhydride, lime, alkalies, iron, chlorine etc. Large quantities of Vitamin C and a certain

amount of Vitamin B are also present. Green plantain contains a large amount of tannin (Nadkarni, 1954).

Waalkes *et al.* (1958) reported that banana has high content of 5-hydroxy tryptamine and norepinephrine. He estimated the serotonin, norepinephrine and dopamine content of *Musa* pulp (avg. wt-130 gm, without peel) and found that pulp contains 28 μg/gm of serotonin, 1.9 μg/gm of norepinephrine, 7.9 μg/gm of dopamine, 3.7 mg of serotonin, and he postulated that serotonin was known to inhibit gastric secretion and due to this high serotonin content banana can be useful in peptic ulcer.

Vegetable banana (*M. paradisiaca*) has been extensively investigated since 1960's based on the report of Waalkes (1958). The anti-ulcerogenic activity of unripe green banana was first reported in 1961 by Sinha *et al.*, 1961).

Sanyal *et al.* (1961) found that banana emulsions introduced directly into the stomach reduce gastric acid secretion and also prevent chronic ulceration and perforation induced by repeated injections of histamine.

Banana contained comparatively large amounts of two physiologically important compounds, namely serotonin (5-HT) and nor-epinephrine in addition to dopamine (3, 4, dihydroxy phenyl alanine) and an unidentified catecholamine. The pulp

contained 8-50 μg/gm of serotonin, 7.9 μg/gm of dopamine, 1-9 μg/gm of nor-epinephrine. The therapeutic use of banana in coeliac diseases, constipation, peptic ulcer may be due to the presence of these active principles. Banana is a rich source of potassium, magnesium, sodium, phosphorus and a fair source of calcium (The Wealth of India, 1962).

The study conducted by Sanyal *et al.* (1963) clearly indicated that unripe plantain banana helps in prevention and healing of the phenyl butazone induced ulcers at a dose rate of 1 gm/kg body weight daily in guinea-pigs. It was suggested that the anti-ulcerogenic properties of plantain banana may be due to a demulcent or antacid effect similar to that of Aluminium hydroxide.

The anti-ulcerogenic activity of *M. paradisiaca* was later confirmed on a variety of experimental animals such as rat, mice and guinea pigs (Sanyal *et al.*, 1964, 1965).

Best *et al.* (1984) found that various preparations of dried unripe plantain banana were found to be anti-ulcerogenic against aspirin-induced ulceration in rats and were effective both as a prophylactic treatment and help in healing of ulcers already induced by aspirin. Ripe fruit bananas were found to be ineffective. The active factor was in the water soluble fraction and was concentrated by extraction to approximately

300 times that of the dried banana powder. The anti-ulcerogenic action of banana preparation appear to be due to their ability to stimulate the growth of gastric mucosa. He also found that Aluminium hydroxide, cimetidine, prostaglandin E_2 except 5-hydroxy tryptamine showed anti-ulcerogenic when used prophylactically in rats but were ineffective in healing ulcers already formed by aspirin. These substances did not stimulate the growth of gastric mucosa.

Ghosal and Saini (1984) identified two new stenyl-acyl glucosides from fruits of *Musa paradisiaca* to show protection against peptic and duodenal ulcers. The structure of these compounds have been established from their spectral data.

Goel *et al.* (1985) established that effective dose of banana did not contain sufficient serotonin to justify the postulate that serotonin in banana was responsible for its anti-ulcerogenic action.

Goel *et al.* (1986) later observed that anti-ulcerogenic action of unripe banana was not due to inhibition of acid-pepsin but was associated with augmentation of gastro-duodenal mucosal protective factors. He noticed that banana increased mucus secretion, enhanced repair and restituition and inhibited mucosal cell shedding.

Chattopadhyay *et al.* (1987) isolated sitoindoside IV, an anti-ulcerogenic acyl steryl glycoside from *M. paradisiaca* and administered at the dose rate of 100-400 μg/mouse over a span of time (3-7 days) produced a statistically significant mobilisation and activation of peritoneal macrophages.

Double blind studies in four centres found that about 70 per cent of endoscopically proved duodenal ulcers healed after 12 weeks of treatment with unripe banana powder compared to about 16 per cent with placebo (Mukhopadhyaya *et al.*, 1987).

The effect of extracts of unripe plantain banana (*Musa sapientum* Linn var *M. paradisiaca*) on the accumulation of eicosanoids in incubates of human gastric and colonic mucosa was studied. The ethanolic extract caused a concentration dependent increase in the eicosonoid accumulation but the water extract was ineffective. Since all the eicosanoid studied tended to increase, banana may act by increasing the availability of arachidonate (Goel *et al.*, 1989).

The introduction of vegetable banana as a drug, MUSAPEP from Reckitt and Colman thus herald the introduction of natural mucosal protective agent (Goel *et al.*, 1991).

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The study was conducted in adult albino rats weighing 100-150 gm of either sex. Rats were maintained under identical feeding and management practices in the laboratory for one week.

Two hundred and thirty two rats were used for the study. They were divided into twenty nine groups with each group consisting of eight rats each.

Aspirin was administered orally for the induction of gastric ulcers at a single dose rate of 200 mg/kg orally for seven days.

The experimental rats were given powder and alcoholic extract of *Ocimum sanctum* leaves, *Withania somnifera* root and *Musa* (AAB group "Nendran") matured and unripe fruit orally.

Famotidine, a standard anti-ulcer drug was given at the dose rate of 40 mg/kg orally for comparing the anti-ulcer activity of *O. sanctum* leaves, *W. somnifera* root and *Musa* (AAB group "Nendran") matured and unripe fruit.

The doses and duration of administration of different plant preparations were fixed arbitrarily (Table 1).

Table 1. Doses of *Ocimum sanctum* leaves, *Musa* (AAB group "Nendran") mature and unripe fruit, *Withania somnifera* root and Famotidine (in mg/kg orally) were fixed as below

Plants	Alcoholic extracts				Powder			
	10 days treatment		20 days treatment		10 days treatment		20 days treatment	
Ocimum sanctum	G(1) 250	G(2) 500	G(3) 250	G(4) 500	G(5) 500	G(6) 1000	G(7) 500	G(8) 1000
Musa (AAB group Nendran)	G(9) 500	G(10) 1000	G(11) 500	G(12) 1000	G(13) 1000	G(14) 2000	G(15) 1000	G(16) 2000
Withania somnifera	G(17) 250	G(18) 500	G(19) 250	G(20) 500	G(21) 500	G(22) 1000	G(23) 500	G(24) 1000
Famotidine	10 days treatment F(10) 40 mg/kg p.o				20 days treatment F(20) 40 mg/kg p.o			

Control group A [CG(A)] - Aspirin treated controls

Control group B [CG(B)] - Natural healing for 10 days

Control group C [CG(C)] - Natural healing for 20 days

Preparation of Aspirin for ulcer induction

Aspirin (Pravin Pharma), (300 mg) tablet was purchased locally and suspended in 5 ml of 2 per cent gum acacia. The suspension contained 60 mg/ml of aspirin and was given orally at the rate of 200 mg/kg body weight, for each rat.

Preparation of Famotidine for oral administration

Famotidine (Torrent Pharmaceuticals Ltd.), (40 mg) tablet was purchased locally and suspended in 10 ml of 2 per cent gum acacia. The suspension contained 4 mg of famotidine/ml and was given orally at the dose rate of 40 mg/kg body weight for each rat.

Preparation of powder of *Ocimum sanctum* (leaves)

O. sanctum leaves collected from KAU campus were air dried at room temperature and powdered. 3 gm powder was suspended in 50 ml of 2 per cent gum acacia. The suspension contained 60 mg of *O. sanctum* leaf powder/ml and was given orally.

Preparation of powder of *Musa* AAB group (Nendran)

Musa (AAB group "Nendran") matured and unripe fruits were purchased locally, peeled, sliced and air dried at room temperature and powdered. 6 gm powder was suspended in 50 ml

of 2 per cent gum acacia. The suspension contained 120 mg of *Musa* powder/ml and was given orally.

Preparation of powder of *Withania somnifera* (root)

W. somnifera roots were purchased locally, air dried at room temperature and powdered. 3 gm powder was suspended in 50 ml of 2 per cent gum acacia. The suspension contained 60 mg of *W. somnifera* powder/ml suspension for oral administration.

Preparation of alcoholic extract of *Ocimum sanctum* leaves

Sixty gms of *O. sanctum* leaf powder was taken in a soxhlet apparatus. The extraction was done using 70 per cent ethyl alcohol. The extract was evaporated to dryness at room temperature. On an average 60 gm of the *O. sanctum* leave powder gave 9 gms of dry extract. This was suspended in 180 ml of 2 per cent gum acacia. The suspension contained 50 mg of *O. sanctum* dry extract/ml for oral administration.

Preparation of alcoholic extract of *Musa* (AAB group, "Nendran") matured and unripe fruit

Hundred grams of matured and unripe banana (AAB group, "Nendran") fruit powder was taken in a soxhlet apparatus. The extraction was done using 70 per cent ethyl alcohol. The residue was evaporated to dryness at room temperature.

Hundred gms of the *Musa* powder gave 5 gms of dry extract. Five gms of dry extract was suspended in 25 ml of 2 per cent gum acacia. The suspension contained 200 mg of *Musa* dry extract/ml for oral administration.

Preparation of alcoholic extract of *Withania somnifera* (root)

Seventy gms of root powder of *W. somnifera* was taken in a soxhlet apparatus. The extraction was done using 70 per cent ethyl alcohol. Seventy gms of root powder yielded 8 gms of dry extract. Eight gms of dry extract was suspended in 200 ml of 2 per cent gum acacia, thus making it a 40 mg of *W. somnifera* dry extract/ml suspension for oral administration.

The Aspirin, Famotidine and plant preparations (both powder and alcoholic extract) were administered by oral intubation using a curved 16" gauge needle with blunt tip and a syringe.

Experimental design

Group	Treatment
Control group A (CGA)	Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib*. On 8th day rats were sacrificed and ulcer index was assessed.

Control group B (CGB)	Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib*. From 8th day onwards food and water were given *ad lib*. On 18th day, rats were sacrificed and assessed the natural healing
Control group C (CGC)	Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. Food and water were given ad lib for next 20 days. On 28th day, rats were sacrificed and the natural healing was assessed
Experimental-GI (*O. santcum* leaf)	Aspirin alone was given orally at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. Eighth day onwards, alcoholic extract of leaves of *O. sanctum* at the rate of 250 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and assessed the healing index.

Experimental-G2 (*O. sanctum* leaf)	Aspirin alone was given at the rate of 200 mg/kg body weight orally for 7 days. Food was restricted and water given *ad lib* for 7 days. 8th day onwards, alcoholic extract of *O. sanctum* leaves at the rate of 500 mg/kg was given orally for 10 days. On 18th day, rats were sacrificed and assessed the healing index.
Experimental-G3 (*O. sanctum* leaf)	Aspirin alone was given orally at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. 8th day onwards, alcoholic extract of *O. sanctum* leaves at the rate of 250 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and assessed the healing index.
Experimental-G4 (*O. sanctum* leaf)	Aspirin alone was given orally at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. 8th

day onwards, alcoholic extract of *O. sanctum* leaves at the rate of 500 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and assessed the healing index.

Experimental-G5 (*O. sanctum* leaf)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *O. sanctum* leaf powder was given at the rate of 500 mg/kg body weight orally for 10 days. On 18th day, rats were sacrificed and assessed healing index.

Experimental-G6 (*O. sanctum* leaf)

Aspirin alone was given at the rate of 200 mg/kg body weight orally for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *O. sanctum* leaves powder was given at the rate of 1000 mg/kg body weight orally for 10 days. On 18th day, rats were sacrificed and assessed healing index.

Experimental-G7 (*O. sanctum* leaf)	Aspirin alone was given at the rate of 200 mg/kg body weight orally for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *O. sanctum* leaf powder was given at the rate of 500 mg/kg body weight orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.
Experimental-G8 (*O. sanctum* leaf)	Aspirin alone was given at the rate of 200 mg/kg body weight orally for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *O. sanctum* leaf powder was given at the dose rate of 1000 mg/kg body weight orally for 20 days. On 28th day, rats were sacrificed and the healing index was assessed.
Experimental-G9 (*Musa* AAB group, "Nendran")	Aspirin alone was given at the rate of 200 mg/kg body weight orally for 7 days. Food was restricted and water given *ad lib* for 7 days. On

8th day onwards alcoholic extract of *Musa* unripe fruit at the rate of 500 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and the healing index was assessed.

Experimental-G10 (*Musa* AAB group, "Nendran")

Aspirin alone was given at the rate of 200 mg/kg body weight orally for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards alcoholic extract of *Musa* unripe fruit at the rate of 1000 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.

Experimental-G11 (*Musa* AAB group, "Nendran")

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards alcoholic extract of *Musa* unripe fruit at the rate of 500 mg/kg body weight was given orally for 20 days. On 28th day, rats were

sacrificed and healing index was assessed.

Experimental-G12 (*Musa* AAB group, "Nendran")

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards alcoholic extract of *Musa* unripe fruit at the rate of 1000 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.

Experimental-G13 (*Musa* AAB group, "Nendran")

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *Musa* unripe fruit powder at the rate of 1000 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.

Experimental-G14 (*Musa* AAB group, "Nendran")

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days.

Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *Musa* unripe fruit powder at the rate of 2000 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.

Experimental-G15 (*Musa* AAB group, "Nendran")

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *Musa* unripe fruit powder at the rate of 1000 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.

Experimental-G16 (*Musa* AAB group, "Nendran")

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards *Musa* unripe fruit powder at the rate of 2000 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.

Experimental-G17 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards alcoholic extract of *W. somnifera* (root) at the rate of 250 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.

Experimental-G18 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards alcoholic extract of *W. somnifera* (root) at the rate of 500 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.

Experimental-G19 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day

onwards alcoholic extract of *W. somnifera* root at the rate of 250 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.

Experimental-G20 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards alcoholic extract of *W. somnifera* root at the rate of 500 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.

Experimental-G21 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards powder of *W. somnifera* (root) at the rate of 500 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.

Experimental-G22 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day *onwards* powder of *W. somnifera* (root) at the rate of 1000 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.

Experimental-G23 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards powder of *W. somnifera* (root) at the rate of 500 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.

Experimental-G24 (*W. somnifera* root)

Aspirin alone was given at the rate of 200 mg/kg body weight for 7 days. Food was restricted and water given *ad lib* for 7 days. On 8th day onwards powder of *W. somnifera*

	(root) at the rate of 1000 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.
Standard drug Famotidine (F10)	Aspirin alone was given orally at the rate of 200 mg/kg body weight for 7 days. On 8th day onwards, Famotidine at the rate of 40 mg/kg body weight was given orally for 10 days. On 18th day, rats were sacrificed and healing index was assessed.
Standard drug Famotidine (F20)	Aspirin alone was given orally at the rate of 200 mg/kg body weight for 7 days. On 8th day onwards, Famotidine at the rate of 40 mg/kg body weight was given orally for 20 days. On 28th day, rats were sacrificed and healing index was assessed.

The stomachs were dissected along the greater curvature and examined under a magnifying lens to determine the incidence and severity of ulceration.

The ulcer index and healing index was calculated after sacrificing the rats using the method employed by Sahni *et al.* (1990).

$$\text{Ulcer index} = \text{Number of ulcers} + \text{Severity of ulceration} + \frac{\text{\% incidence}}{\text{No. of rats}}$$

$$\text{Healing index} = \frac{\text{Ulcer index (control)*} - \text{Ulcer index (drug)}}{\text{Ulcer index (control)*}} \times 100$$

* Control group A [CG(A)] - Rats given aspirin alone for seven days and sacrificed on eight day

Severity of ulceration was assessed by a method employed by Gupta *et al.* (1988) (arbitrary scoring system).

(i)	Denuded epithelium	= 10
(ii)	Petechial and frank haemorrhage	= 20
(iii)	One or two ulcers	= 30
(iv)	Multiple ulcers	= 40
(v)	Perforated ulcers	= 50

The data obtained were compared with those of standard anti-ulcer drug, famotidine.

Haematological study

Blood samples were collected from the orbital plexus of rats at the end of the experiment for the estimation of haematological parameters viz. erythrocyte count, total leucocyte count, differential count and haemoglobin concentration as described by Schalm (1975).

Histopathological examination was done to demonstrate the ulcer lesion and healing, according to the method described by George and Somvanshi (1987). Three mm thick piece of stomach tissue was taken and fixed in 10 per cent formaline and processed through routine paraffin-embedding process using turpentine oil for clearing and stained with haematoxylin and eosin and studied the histopathology.

Results

RESULTS

Results obtained were tabulated and presented in tables 2 to 36 and the statistical analysis was done using the method suggested by Snedecor and Cochran (1980).

All the plant preparations under study significantly reduced the ulcer index of aspirin treated control [CG(A)]. The ulcer index and healing index of all the plant preparations, famotidine and control groups were determined, tabulated and presented in Tables 2 to 30 and graphically represented in Figures 1 to 6.

4.1 *Ocimum sanctum* leaf

None of the experimental groups under study with the plant, produced statistically significant healing effects comparable to famotidine group given at the dose rate of 40 mg/kg body weight for 10 days and 20 days respectively.

Healing obtained with *O. sanctum* alcoholic extract at 500 mg/kg body weight for 10 days (28.72 ± 6.44) and alcoholic extract of 250 mg/kg body weight for 20 days (31.42 ± 5.61) are comparable only with the control groups for 10 days and 20 days (28.59 ± 4.47 and 32.77 ± 5.41) respectively while *O. sanctum*, alcoholic extract at the dose rate of 250 mg/kg body weight for 10 days (36.5 ± 6.37), alcoholic extract at

500 mg/kg body weight for 20 days (35.48 ± 6.41), powder at 500 mg/kg body weight for 20 days (36.98 ± 4.37), powder at 1000 mg/kg body weight for 20 days (39.69 ± 4.46), powder at 500 mg/kg for 10 days (35.04 ± 4.85) and powder at 1000 mg/kg body weight for 10 days (37.68 ± 4.99) produced significantly higher healing efficiency than control groups for 10 days and 20 days (28.59 ± 4.47 and 32.77 ± 5.41) respectively.

Summary of ulcer index and healing index produced by *O. sanctum* leaves in comparison with famotidine and control groups are presented in Table 31 and 32 and represented graphically in Fig.1 and 4 respectively.

4.2 *Musa* (AAB group 'Nendran') mature and unripe fruit

All experimental groups under study with this plant have significantly higher healing effects than control groups for 10 days.

The alcoholic extract of *Musa* (AAB group 'Nendran') at the dose rate of 1000 mg/kg body weight for 10 days (55.22 ± 6.75), 500 mg/kg body weight for 20 days (47.312 ± 4.13); and 1000 mg/kg body weight for 20 days (62.386 ± 5.92) produced healing comparable to famotidine at the dose rate of 40 mg/kg body weight for 10 days and 20 days (50.49 ± 5.6 and 67.44 ± 6.03) respectively.

Musa (AAB group 'Nendran') powder at the dose rate of 1000 mg/kg body weight for 10 days (40.842 ± 5.94) and 2000 mg/kg body weight for 20 days (44.283 ± 3.82) produced healing comparable to famotidine at the dose rate of 40 mg/kg body weight for 10 days (50.493 ± 5.6).

Musa alcoholic extract at the dose rate of 500 mg/kg body weight for 10 days (37.583 ± 6.69), powder at the dose rate of 2000 mg/kg body weight for 10 days (31.967 ± 5.69) and 1000 mg/kg body weight for 20 days (32.567 ± 6.15) produced healing comparable with control groups for 10 days and 20 days (28.587 ± 4.47 and 32.767 ± 5.41) respectively.

Summary of ulcer index and healing index produced by *Musa* (AAB group 'Nendran') unripe fruit in comparison with famotidine and control groups are presented in Table 31 and 33 and represented graphically in Fig.2 and 5 respectively.

4.3 *Withania somnifera* root

Healing obtained with *W. somnifera* alcoholic extract at the dose rate of 250 mg/kg body weight for 20 days (48.473 ± 7.67) and 500 mg/kg body weight for 20 days (54.586 ± 5.74) and powder at the dose rate of 1000 mg/kg body weight for 20 days (41.96 ± 6.36) are comparable with famotidine at the dose rate of 40 mg/kg body weight for 10 days and 20 days (50.493 ± 5.6 and 67.443 ± 6.03) respectively.

All other experimental group under study with this plant produced healing comparable to famotidine at the dose rate of 40 mg/kg body weight for 10 days (50.49 ± 5.6).

Summary of observations of ulcer index and healing index produced by *W. somnifera* root are presented in Table 31 and 34 and represented graphically in Fig.3 and 6 respectively.

4.4 Haematological study

The study of haematological parameters of all the group revealed no significant changes at 1 per cent level and all values fall within the normal range of blood values for the species under study. The details of the observations are given in Table 35 and that of statistical analysis in Table 36.

4.5 Histopathological study

Grossly, the lesion observed were petechial haemorrhages, shallow erosions and deep ulcers. More than one type of lesion was observed in the stomach, but in a large number of cases the lesions were petechial haemorrhages and shallow erosions. The haemorrhagic ulcers were linear, mostly occurring in the glandular corpus (Fig.8).

Microscopically, examination of the mucosal ulcers revealed on cross section a roughly wedge shaped zone of

coagulative necrosis characterized further by capillary haemorrhages in the lamina propria, desquamation of the epithelial layer and development of a superficial area of ulceration. The lesions whether shallow erosion or deep ulcers were covered by an exudate consisting of mucous, fibrin and necrotic debris (Fig.9). The edges of the lesions were raised above the surrounding tissue and there was submucosal odema in majority of the cases (Fig.10).

The ulcers which were largely limited to the glandular corpus resolved by granulation tissue formation and subsequently by reepithelialization.

The granulation tissue consisted of proliferating capillaries along with the proliferation of fibroblats (Fig.10). Besides, there was infiltration by a mixed inflammatory cell population and the healing lesion was overlaid by a usually thin layer of necrotic debris (Figs. 12 and 13). The phase of reepithelialization was characterized by mucous-secreting goblet cell metaplasia and glandular hyperplasia (Fig.14). The re-epithelialization was almost complete and the functional morphology was almost restored in the Famotidine treated group (Fig.15). This was followed to a lesser degree by the *Musa* treated group and next in order by the Withania treated group. Of the four treatment groups, the response was found to be minimal with Ocimum.

Table 2. Control group (A), aspirin alone treated for 7 days, ulcer index (control) (200 mg/kg body weight)

	1	2	3	4	5	6	7	8
No. of ulcers	4	6	4	4	5	5	5	6
Ulcer score	50	40	40	40	40	40	40	40
% of incidence	100	100	100	100	100	100	100	100
Ulcer index (drug)	66.5	58.5	56.5	56.5	57.5	57.5	57.5	58.5

Mean ulcer index = 58.63 ± 1.15

Table 3. Control group (B), natural healing for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	2	3	3	1	2	0	3	2
Ulcer score	30	40	40	30	30	10	40	30
% of incidence	87.5	87.5	87.5	87.5	87.5	87.5	87.5	87.5
Ulcer index (drug)	42.94	53.94	53.94	41.94	42.94	20.94	53.94	42.94
Healing index (drug)	26.72	7.95	7.95	28.43	26.72	65.20	7.95	26.75

Mean ulcer index = 44.19 ± 3.86
Mean healing index = 28.59 ± 4.47

Table 4. Control group (C), natural healing for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	2	4	3	4	0	2	2
Ulcer score	10	30	40	40	40	10	30	30
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	19.38	41.38	53.38	52.38	53.38	19.38	41.38	41.38
Healing index (drug)	66.9	29.4	8.91	10.61	8.91	66.90	29.40	29.40

Mean ulcer index = 40.26 ± 4.94
Mean healing index = 32.77 ± 5.41

Table 5. F (10) - Famotidine, 40 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	2	1	2	0	0	0	2
Ulcer score	0	30	30	30	10	10	0	30
% of incidence	50	50	50	50	50	50	50	50
Ulcer index (drug)	6.25	38.25	37.25	38.25	16.25	16.25	6.25	38.25
Healing index (drug)	89.33	34.72	36.43	34.72	72.26	72.26	89.33	34.72

Mean ulcer index = 24.63 ± 5.22
Mean healing index = 50.49 ± 5.60

Table 6. F (20) - Famotidine, 40 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	0	2	0	0	1	0	0
Ulcer score	0	0	30	0	0	30	0	0
% of incidence	25	25	25	25	25	25	25	25
Ulcer index (drug)	3.13	3.13	35.13	3.13	3.13	34.13	3.13	3.13
Healing index (drug)	94.65	94.65	40.05	94.65	94.65	41.75	94.65	94.65

Mean ulcer index = 11.01 ± 5.15
Mean healing index = 67.44 ± 6.03

Table 7. G (1) - *Ocimum sanctum*, leaf alcoholic extract, 250 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	2	5	0	6	0	5	0	2
Ulcer score	30	40	10	40	10	40	10	30
% of incidence	62.5	62.5	62.5	62.5	62.5	62.5	62.5	62.5
Ulcer index (drug)	39.81	52.81	17.81	53.81	17.81	52.81	17.81	39.81
Healing index (drug)	32.06	9.88	69.6	8.17	69.6	9.88	69.6	32.06

Mean ulcer index = 36.56 ± 5.82
Mean healing index = 36.59 ± 6.37

Table 8. G (2) - *Ocimum sanctum*, leaf alcoholic extract, 500 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	6	0	6	2	6	0	6	2
Ulcer score	40	10	40	30	40	10	40	30
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	55.38	19.38	55.38	41.38	55.38	19.38	55.38	41.38
Healing index (drug)	5.49	66.92	5.49	29.38	5.49	66.92	5.49	29.38

Mean ulcer index = 42.88 ± 5.56
Mean healing index = 28.72 ± 6.44

Table 9. G (3) - *Ocimum sanctum*, leaf alcoholic extract, 250 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	2	4	3	3	2	0	0	3
Ulcer score	30	40	40	40	30	10	10	40
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	41.38	53.38	52.38	52.38	41.38	19.38	19.38	52.38
Healing index (drug)	29.38	8.90	10.61	10.61	29.38	66.92	66.92	10.61

Mean ulcer index = 41.51 ± 5.13
Mean healing index = 31.24 ± 5.61

Table 10. G (4) - *Ocimum sanctum*, leaf alcoholic extract, 500 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	4	4	4	0	4	2	0	0
Ulcer score	40	40	40	10	40	30	10	10
% of incidence	62.5	62.5	62.5	62.5	62.5	62.5	62.5	62.5
Ulcer index (drug)	51.81	51.81	51.81	17.81	51.81	39.81	17.81	17.81
Healing index (drug)	11.58	11.58	11.58	69.60	11.58	32.06	69.60	69.60

Mean ulcer index = 37.56 ± 5.95
Mean healing index = 35.48 ± 6.41

Table 11. G (5) - *Ocimum sanctum*, leaf powder, 500 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	3	2	0	2	1	3	0	2
Ulcer score	40	30	10	30	30	40	10	30
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	52.38	41.38	19.38	41.38	40.38	52.37	19.38	41.38
Healing index (drug)	10.61	29.39	66.92	29.39	31.10	10.61	66.92	29.39

Mean ulcer index = 38.51 ± 4.52
Mean healing index = 35.04 ± 4.85

Table 12. G (6) - *Ocimum sanctum*, leaf powder, 1000 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	2	1	2	1	3	0	3
Ulcer score	10	30	20	30	10	40	20	40
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	19.38	41.38	30.38	41.38	20.38	52.38	29.38	52.38
Healing index (drug)	66.92	29.38	48.15	29.38	65.22	10.61	49.86	10.61

Mean ulcer index = 35.88 ± 4.60
Mean healing index = 37.68 ± 4.99

Table 13. G (7) - *Ocimum sanctum*, leaf powder, 500 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	3	0	2	0	3	2	2
Ulcer score	10	40	20	30	20	40	30	30
% of incidence	62.5	62.5	62.5	62.5	62.5	62.5	62.5	62.5
Ulcer index (drug)	17.81	50.81	27.81	39.81	27.81	50.81	39.81	39.81
Healing index (drug)	69.60	13.29	52.54	32.06	52.54	13.29	32.06	32.06

Mean ulcer index = 36.81 ± 4.09
Mean healing index = 36.98 ± 4.37

Table 14. G (8) - *Ocimum sanctum*, leaf powder, 1000 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	2	2	4	0	2	2	0	0
Ulcer score	30	30	40	10	30	30	20	10
% of incidence	62.5	62.5	62.5	62.5	62.5	62.5	62.5	62.5
Ulcer index (drug)	39.81	39.81	51.81	17.81	39.81	39.81	27.81	17.81
Healing index (drug)	32.06	32.06	11.58	69.60	32.06	32.06	52.54	69.60

Mean ulcer index = 34.31 ± 4.25
Mean healing index = 39.69 ± 4.46

Table 15. G (9) - *Musa* (AAB group, "Nendran"), mature and unripe fruit alcoholic extract, 500 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	2	2	3	4	2	2	0
Ulcer score	0	30	30	40	40	30	30	0
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	9.38	41.38	41.38	52.38	53.38	41.38	41.38	9.38
Healing index (drug)	83.99	29.39	29.39	10.61	8.90	29.39	29.39	83.99

Mean ulcer index = 36.26 ± 6.12
Mean healing index = 37.58 ± 6.69

Table 16. G (10) - *Musa* (AAB group, "Nendran"), mature and unripe fruit alcoholic extract, 1000 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	2	2	0	3	0	0	0
Ulcer score	10	30	30	0	40	10	0	0
% of incidence	37.5	37.5	37.5	37.5	37.5	37.5	37.5	37.5
Ulcer index (drug)	14.69	36.69	36.69	4.69	47.69	14.69	4.69	4.69
Healing index (drug)	74.93	37.39	37.39	91.99	18.61	74.93	91.99	91.99

Mean ulcer index = 20.57 ± 6.09
Mean healing index = 55.22 ± 6.75

Table 17. G (11) - *Musa* (AAB group, "Nendran"), mature and unripe fruit alcoholic extract, 500 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	2	0	0	1	0	0	2	2
Ulcer score	30	10	10	30	10	10	30	30
% of incidence	50	50	50	50	50	50	50	50
Ulcer index (drug)	38.25	16.25	16.85	37.25	16.25	16.25	38.25	38.25
Healing index (drug)	34.72	72.26	72.26	36.43	72.26	72.26	34.72	34.72

Mean ulcer index = 27.13 ± 4.10
Mean healing index = 47.31 ± 4.13

Table 18. G (12) - *Musa* (AAB group, "Nendran"), mature and unripe fruit alcoholic extract, 1000 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	1	2	0	0	0	0	0	0
Ulcer score	30	30	0	20	0	0	10	0
% of incidence	25	25	25	25	25	25	25	25
Ulcer index (drug)	34.13	35.13	3.13	23.13	3.13	3.13	13.13	3.13
Healing index (drug)	41.75	40.05	94.65	60.52	94.65	94.65	77.59	94.65

Mean ulcer index = 14.76 ± 5.00
Mean healing index = 62.39 ± 5.92

Table 19. G (13) - *Musa* (AAB group, "Nendran"), mature and unripe fruit powder 1000 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	4	2	3	4	0	0	0
Ulcer score	10	40	30	40	40	20	10	10
% of incidence	50	50	50	50	50	50	50	50
Ulcer index (drug)	16.25	50.25	38.25	49.25	50.25	26.25	16.25	16.25
Healing index (drug)	72.22	14.25	34.73	15.95	14.25	55.20	72.22	72.22

Mean ulcer index = 32.88 ± 5.62
Mean healing index = 40.84 ± 5.94

Table 20. G (14) - *Musa* (AAB group, "Nendran"), mature and unripe fruit powder, 2000 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	3	2	4	4	1	0	1	2
Ulcer score	40	30	40	40	10	20	10	30
% of incidence	87.5	87.5	87.5	87.5	87.5	87.5	87.5	87.5
Ulcer index (drug)	53.94	42.94	54.94	54.94	51.94	30.94	21.94	42.94
Healing index (drug)	7.95	26.72	6.24	6.24	62.50	47.50	62.50	26.72

Mean ulcer index = 40.57 ± 4.97
Mean healing index = 31.97 ± 5.69

Table 21. G (15) - *Musa* (AAB group, "Nendran"), mature and unripe fruit powder, 1000 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	4	3	0	3	5	0	4
Ulcer score	20	40	40	10	40	40	10	40
% of incidence	62.5	62.5	62.5	62.5	62.5	62.5	62.5	62.5
Ulcer index (drug)	27.81	51.81	50.81	17.81	50.81	52.81	17.81	51.81
Healing index (drug)	52.54	11.58	13.29	69.60	13.29	9.88	69.60	11.58

Mean ulcer index = 40.19 ± 5.68
Mean healing index = 32.57 ± 6.15

Table 22. G (16) - *Musa* (AAB group, "Nendran"), mature and unripe fruit powder, 2000 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	1	1	2	2	0	2	0
Ulcer score	10	30	20	30	30	10	30	10
% of incidence	62.5	62.5	62.5	62.5	62.5	62.5	62.5	62.5
Ulcer index (drug)	17.81	38.81	28.81	39.81	39.81	17.81	39.81	17.81
Healing index (drug)	69.60	33.77	50.83	32.06	32.06	69.60	32.06	69.60

Mean ulcer index = 30.06 ± 3.80
Mean healing index = 44.28 ± 3.82

Table 23. G (17) - *Withania somnifera*, root alcoholic extract, 250 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	2	4	0	2	4	0	2	4
Ulcer score	30	40	10	30	40	10	30	40
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	41.38	53.38	19.38	41.38	53.38	19.38	41.38	53.38
Healing index (drug)	29.38	8.90	66.92	29.38	8.90	66.92	29.38	8.90

Mean ulcer index = 40.38 ± 4.98
Mean healing index = 32.56 ± 5.49

Table 24. G (18) - *Withania somnifera*, root alcoholic extract, 500 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	1	4	2	2	4	0	2
Ulcer score	10	10	40	30	30	40	0	30
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	19.38	20.38	53.38	41.38	41.38	53.38	9.38	41.38
Healing index (drug)	66.92	65.22	8.90	29.38	29.38	8.90	83.99	29.38

Mean ulcer index = 35.01 ± 5.84
Mean healing index = 38.56 ± 6.37

Table 25. G (19) - *Withania somnifera*, root alcoholic extract, 250 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	2	0	4	0	4	0	2
Ulcer score	10	30	0	40	0	40	0	30
% of incidence	50	50	50	50	50	50	50	50
Ulcer index (drug)	16.25	38.25	6.25	50.25	6.25	50.25	6.25	38.25
Healing index (drug)	72.26	34.72	89.33	14.25	49.33	14.25	89.33	34.72

Mean ulcer index = 26.50 ± 6.98
Mean healing index = 48.47 ± 7.67

Table 26. G (20) - *Withania somnifera*, root alcoholic extract, 500 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	1	0	2	2	0	2	0
Ulcer score	0	10	10	30	30	0	30	0
% of incidence	50	50	50	50	50	50	50	50
Ulcer index (drug)	6.25	17.25	16.25	38.25	38.25	6.25	38.25	6.25
Healing index (drug)	89.33	70.56	72.26	34.72	34.72	89.33	34.72	89.33

Mean ulcer index = 20.88 ± 5.31
Mean healing index = 54.59 ± 5.74

Table 27. G (21) - *Withania somnifera*, root powder, 500 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	3	5	0	0	6	3	5	3
Ulcer score	40	40	10	10	40	40	40	40
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	52.38	54.38	19.38	19.38	55.38	52.38	54.38	52.38
Healing index (drug)	10.61	7.20	66.92	5.40	10.61	10.61	7.20	10.61

Mean ulcer index = 45.01 ± 5.60
Mean healing index = 26.44 ± 6.25

Table 28. G (22) - *Withania somnifera*, root powder, 1000 mg/kg, b.wt. for 10 days

	1	2	3	4	5	6	7	8
No. of ulcers	4	0	5	4	2	0	2	4
Ulcer score	40	10	40	40	30	10	30	40
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	53.38	19.38	54.38	53.38	41.38	19.38	41.38	53.38
Healing index (drug)	8.90	67.06	7.20	8.90	29.38	67.06	29.38	8.90

Mean ulcer index = 42.01 ± 5.29
Mean healing index = 30.42 ± 5.90

Table 29. G (23) - *Withania somnifera*, root powder, 500 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	4	4	0	2	2	4	0	0
Ulcer score	40	40	10	30	30	40	10	10
% of incidence	75	75	75	75	75	75	75	75
Ulcer index (drug)	53.38	53.38	19.38	41.38	41.38	53.38	19.38	19.38
Healing index (drug)	8.90	8.90	66.90	29.30	29.30	8.90	66.90	66.90

Mean ulcer index = 37.63 ± 5.62
Mean healing index = 35.30 ± 6.17

Table 30. G (24) - *Withania somnifera*, root powder, 1000 mg/kg, b.wt. for 20 days

	1	2	3	4	5	6	7	8
No. of ulcers	0	4	4	2	0	4	0	0
Ulcer score	10	40	40	30	10	40	10	10
% of incidence	50	50	50	50	50	50	50	50
Ulcer index (drug)	16.25	50.25	50.25	38.25	16.25	50.25	16.25	16.25
Healing index (drug)	72.26	14.24	14.24	34.72	72.26	14.24	72.26	72.26

Mean ulcer index = 31.75 ± 6.01
Mean healing index = 41.96 ± 6.36

Table 31. Summary of observations of ulcer index obtained with *O. sanctum*, *Musa* (AAB group, "Nendran"), and *W. somnifera* in comparison with famotidine and control groups

No. of animals 8

Groups	Mean ulcer index
O. sanctum	
G(1)	36.560 ± 5.82
G(2)	42.880 ± 5.56
G(3)	41.505 ± 5.13
G(4)	37.560 ± 5.95
G(5)	38.505 ± 4.52
G(6)	35.880 ± 4.60
G(7)	36.880 ± 4.60
G(8)	36.810 ± 4.09
	34.310 ± 4.25
Musa (AAB group, "Nendran")	
G(9)	36.225 ± 6.12
G(10)	20.565 ± 6.09
G(11)	27.125 ± 4.10
G(12)	14.755 ± 5.00
G(13)	32.875 ± 5.62
G(14)	40.565 ± 4.97
G(15)	40.185 ± 5.68
G(16)	30.060 ± 3.80
W. somnifera	
G(17)	40.380 ± 4.98
G(18)	35.005 ± 5.84
G(19)	26.500 ± 6.98
G(20)	20.875 ± 5.31
G(21)	45.005 ± 5.60
G(22)	42.005 ± 5.29
G(23)	37.630 ± 5.62
G(24)	31.750 ± 6.01
Control groups	
CG(A)	
CG(B)	44.190 ± 3.86
CG(C)	40.225 ± 4.94
Famotidine groups	
F(10)	24.625 ± 5.22
F(20)	11.005 ± 5.15

ANALYSIS OF VARIANCE TABLE

	Degrees of freedom	Sum of squares	Mean square	F value	Prob.
Treatment	28	21726.273	775.938	3.599**	0.0000
Error	203	43760.878	215.571		

CD = 13.21

** Significant at 1 per cent level

Table 32. Summary of observations of healing index produced by *O. sanctum* in comparison with Famotidine and control groups

No. of animals 8

Groups	Mean healing index
G(1)	36.499 ± 6.37
G(2)	28.718 ± 6.44
G(3)	31.242 ± 5.61
G(4)	35.479 ± 6.41
G(5)	35.040 ± 4.85
G(6)	37.678 ± 4.99
G(7)	36.978 ± 4.37
G(8)	39.692 ± 4.46
CG(B)	28.587 ± 4.47
CG(C)	32.767 ± 5.41
F(10)	50.493 ± 5.60
F(20)	67.443 ± 6.03

ANALYSIS OF VARIANCE TABLE

	Degrees of freedom	Sum of squares	Mean square	F value	Prob.
Treatment	11	10323.138	938.467	3.916**	0.0001
Error	84	20128.881	239.630		

CD = 6.42

** Significant at 1 per cent level

Table 33. Summary of observations of healing index produced by *Musa* (AAB group, "Nendran") in comparison with Famotidine and control groups

No. of animals 8

Groups	Mean healing index
G(9)	37.583 ± 6.69
G(10)	55.220 ± 6.75
G(11)	77.312 ± 4.13
G(12)	62.386 ± 5.92
G(13)	40.842 ± 5.94
G(14)	31.967 ± 5.69
G(15)	32.567 ± 6.15
G(16)	44.283 ± 3.82
CG(B)	28.587 ± 4.47
CG(C)	32.767 ± 5.47
F(10)	50.793 ± 5.60
F(20)	67.443 ± 6.03

ANALYSIS OF VARIANCE TABLE

	Degrees of freedom	Sum of squares	Mean square	F value	Prob.
Treatment	11	14049.118	1277.193	5.004**	0.000
Error	84	21270.384	253.219		

CD = 21.005

** Significant at 1 per cent level

Table 34. Summary of observations of healing index produced by *W. somnifera* in comparison with Famotidine and control groups

No. of animals 8

Groups	Mean healing index
G(17)	32.556 ± 5.49
G(18)	38.563 ± 6.37
G(19)	48.473 ± 7.67
G(20)	54.583 ± 5.74
G(21)	26.435 ± 6.25
G(22)	30.419 ± 5.90
G(23)	35.299 ± 6.17
G(24)	41.957 ± 6.36
CG(B)	28.587 ± 4.47
CG(C)	32.767 ± 5.41
F(10)	50.493 ± 5.60
F(20)	67.443 ± 6.03

ANALYSIS OF VARIANCE TABLE

	Degrees of freedom	Sum of squares	Mean square	F value	Prob.
Treatment	11	13478.051	1225.277	4.249**	0.0001
Error	84	24222.471	288.363		

CD = 21.005

** Significant at 1 per cent level

Table 35. Haematology

Summary of observations of haematological parameters of *O. sanctum*, *Musa* and *W. somnifera* in comparison with Famotidine and control groups

	RBC 10^6/m.m^3	WBC 10^3/m.m^3	Hb gm %	DC%				
				N	L	M	B	E
Normal values	(9-10.0)	(8.8-15.0)	(10.8-17.5)	(15-40)	(50-80)	(0-3)	(0-1.5)	(0-5)
	8.14 ± 1.11	12.04 ± 1.92	14.13 ± 0.48	21.75 ± 0.49	76.88 ± 0.29	0.25 ± 0.46	0.375± 0.52	0.625± 0.33
Control group A	8.431± 0.34	11.244± 0.73	15.188± 0.62	22.875± 0.86	76.000± 0.84	0.250± 0.16	0.125± 0.12	0.625± 0.26
Control group B	7.275± 0.55	12.60 ± 1.02	13.875± 0.26	21.25 ± 0.69	77.375± 0.84	0.54 ± 0.18	0.375± 0.18	0.50 ± 0.31
Control group C	7.594± 0.36	13.10 ± 1.08	14.65 ± 0.44	22.25 ± 0.84	76.625± 0.82	0.125± 0.12	0.25 ± 0.16	0.75 ± 0.31
Famotidine F (10)	7.979± 0.12	11.515± 1.17	15.025± 0.66	21.25 ± 0.45	78.00 ± 0.33	0.25 ± 0.16	0.00 ±	0.50 ± 0.27
Famotidine F (20)	8.750± 0.24	12.438± 1.084	14.013± 0.35	22.625± 0.53	76.25 ± 0.62	0.25 ± 0.16	0.125± 0.12	0.625± 0.33
OS/AE/250 mg/kg b.wt. for 10 days	7.978± 0.59	14.225± 1.10	13.40 ± 0.31	21.125± 0.40	77.625± 0.26	0.375± 0.18	0.25 ± 0.16	0.50 ± 0.27
OS/AE/500 mg/kg b.wt. for 10 days	7.192± 0.58	14.013± 0.60	14.025± 0.44	21.375± 0.46	77.625± 0.37	0.125± 0.12	0.125± 0.12	0.625± 0.26
OS/AE/250 mg/kg b.wt. for 20 days	8.263± 0.37	12.875± 0.36	13.35 ± 0.39	21.375± 0.33	78.00 ± 0.27	0.125± 0.12	0.00 ±	0.50 ± 0.19

Table 35. (Contd.)

OS/AE/500 mg/kg b.wt. for 20 days	8.187± 0.44	14.54 ± 0.79	13.70 ± 0.59	21.50 ± 0.38	78.00 ± 0.38	0.125± 0.12	0.125± 0.12	0.250± 0.16
OS/PW/500 mg/kg b.wt. for 10 days	7.95 ± 0.46	13.175± 1.10	14.35 ± 0.35	20.375± 0.50	78.50 ± 0.46	0.25 ± 0.16	0.125± 0.12	0.75 ± 0.31
OS/PW/1000 mg/kg b.wt. for 10 days	8.65 ± 0.32	11.45 ± 0.86	14.975± 0.16	21.75 ± 0.53	77.125± 0.58	0.50 ± 0.18	0.125± 0.12	0.50 ± 0.33
OS/PW/500 mg/kg b.wt. for 20 days	8.083± 0.59	12.25 ± 1.02	14.95 ± 0.23	21.25 ± 0.65	77.375± 0.37	0.25 ± 0.16	0.375± 0.18	0.75 ± 0.31
OS/PW/1000 mg/kg b.wt. for 20 days	8.069± 0.42	11.575± 1.08	14.725± 0.23	21.50 ± 0.42	77.50 ± 0.60	0.125± 0.12	0.375± 0.18	0.50 ± 0.27
MU/AE/500 mg/kg b.wt. for 10 days	8.844± 0.19	12.835± 0.44	13.70 ± 0.51	21.125± 0.35	78.125± 0.23	0.125± 0.12	0.125± 0.12	0.50 ± 0.27
MU/AE/1000 mg/kg b.wt. for 10 days	8.517± 0.36	13.77 ± 0.45	13.875± 0.35	21.125± 0.40	78.50 ± 0.33	0.125± 0.12	0.000±	0.375± 0.26
MU/AE/500 mg/kg b.wt. for 20 days	8.30 ± 0.48	12.625± 0.89	13.875± 0.39	21.375± 0.33	77.25 ± 0.79	0.25 ± 0.16	0.125± 0.12	0.50 ± 0.19
MU/AE/1000 mg/kg b.wt. for 10 days	7.963± 0.49	12.61 ± 0.31	14.050± 0.39	21.125± 0.40	78.125± 0.40	0.250± 0.16	0.00 ±	0.50 ± 0.33
MU/PW/1000 mg/kg b.wt. for 10 days	8.988± 0.27	11.138± 1.01	13.487± 0.39	22.00 ± 0.46	76.75 ± 0.53	0.625± 0.26	0.250± 0.16	0.375± 0.26
MU/PW/2000 mg/kg b.wt. for 10 days	8.463± 0.24	11.919± 0.66	14.125± 0.41	21.50 ± 0.50	77.375± 0.53	0.375± 0.18	0.250± 0.16	0.50 ± 0.27
MU/PW/1000 mg/kg b.wt. for 20 days	8.688± 0.24	12.444± 0.52	13.625± 0.31	22.00 ± 0.50	76.75 ± 0.49	0.375± 0.18	0.25 ± 0.16	0.625± 0.33

Table 35. (Contd.)

MU/PW/2000 mg/kg b.wt. for 10 days	8.438± 0.35	11.506± 0.49	13.975± 0.25	21.625± 0.50	77.375± 0.63	0.25 ± 0.16	0.375± 0.18	0.375± 0.26
WS/AE/250 mg/kg b.wt. for 10 days	8.281± 0.46	12.759± 0.71	14.705± 0.25	21.25 ± 0.37	77.625± 0.42	0.50 ± 0.18	0.375± 0.18	0.25 ± 0.25
WS/AE/500 mg/kg b.wt. for 10 days	8.649± 0.34	13.824± 0.48	14.70 ± 0.21	21.50 ± 0.33	77.875± 0.35	0.375± 0.18	0.25 ± 0.16	0.00 ±
WS/AE/250 mg/kg b.wt. for 20 days	0.069± 0.61	14.238± 0.49	14.175± 0.38	21.50 ± 0.33	77.50 ± 0.33	0.375± 0.18	0.375± 0.18	0.25 ± 0.25
WS/AE/500 mg/kg b.wt. for 20 days	7.95 ± 0.53	12.069± 0.91	14.00 ± 0.40	20.875± 0.29	78.125± 0.52	0.375± 0.18	0.125± 0.12	0.50 ± 0.33
WS/PW/500 mg/kg b.wt. for 10 days	7.90 ± 0.52	11.781± 0.51	14.258± 0.46	20.875± 0.29	78.00 ± 0.50	0.375± 0.18	0.25 ± 0.16	0.50 ± 0.27
WS/PW/1000 mg/kg b.wt. for 10 days	8.198± 0.51	12.887± 0.57	14.35 ± 0.32	21.125± 0.29	77.875± 0.29	0.25 ± 0.16	0.125± 0.12	0.625± 0.26
WS/PW/500 mg/kg b.wt. for 20 days	8.081± 0.46	13.125± 1.03	13.85 ± 0.36	21.25 ± 0.37	77.875± 0.52	0.25 ± 0.16	0.125± 0.12	0.50 ± 0.27
WS/PW/1000 mg/kg b.wt. for 20 days	7.944± 0.51	13.775± 0.89	14.20 ± 0.40	21.125± 0.40	77.875± 0.35	0.125± 0.12	0.375± 0.18	0.50 ± 0.27

O.S - *Ocimum sanctum*
MU - *Musa* AAB group "Nendran"
W.S - *Withania somnifera*
PW - Powder
AE - Alcoholic extract
bwt - body weight

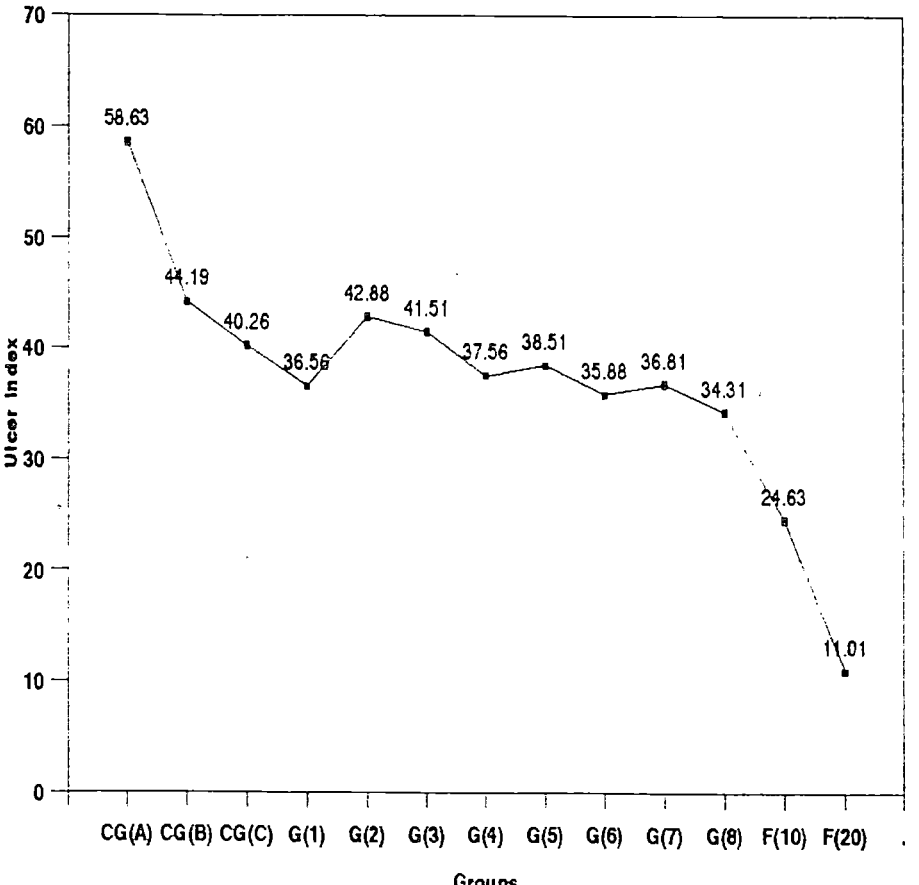
Table 36. ANALYSIS OF VARIANCE TABLE (Heamatological parameters)

	Degrees of freedom	Sum of squares	Mean square	F value	Prob.
RBC					
Treatment	29	40.131	1.384	0.928 NS	-
Error	210	313.176	1.491		
WBC					
Treatment	29	214.954	7.412	1.404 NS	0.0915
Error	210	1108.441	5.278		
Hb					
Treatment	29	57.532	1.984	1.593 NS	0.034
Error	210	261.600	1.246		
N					
Treatment	29	60.371	2.084	1.129 NS	0.305
Error	210	387.125	1.843		
L					
Treatment	29	88.921	3.066	1.523 NS	0.0497
Error	210	422.875	2.014		
M					
Treatment	29	4.233	0.146	0.659 NS	-
Error	210	46.500	0.221		
B					
Treatment	29	3.871	0.133	0.798 NS	-
Error	210	35.125	0.167		
E					
Treatment	29	6.121	0.211	0.358 NS	-
Error	210	123.875	0.590		

NS - Not significant at 1% level

RBC = Red blood count
WBC = White blood count
Hb = Haemoglobin
N = Neutrophil
L = Lymphocyte
M = Monocyte
B = Basophil
E = Eosinophil

Fig.1 COMPARATIVE ULCER INDEX OF OCIMUM SANCTUM WITH FAMOTIDINE AND CONTROL GROUPS



CG(A) = Control group, Ulcer index (control) (Aspirin alone treated for 7 days)

CG(B) = Control group for 10 days (Natural healing)

CG(C) = Control group for 20 days (Natural healing)

G(1) = A.E, 250mg/Kg. B.Wt for 10 days

G(2) = A.E, 500mg/Kg. B.Wt for 10 days

G(3) = A.E, 250mg/Kg. B.Wt for 20 days

G(4) = A.E, 500mg/Kg. B.Wt for 20 days

G(5) = P.W, 500mg/Kg. B.Wt for 10 days

G(6) = P.W, 1000mg/Kg. B.Wt for 10 days

G(7) = P.W, 500mg/Kg. B.Wt for 20 days

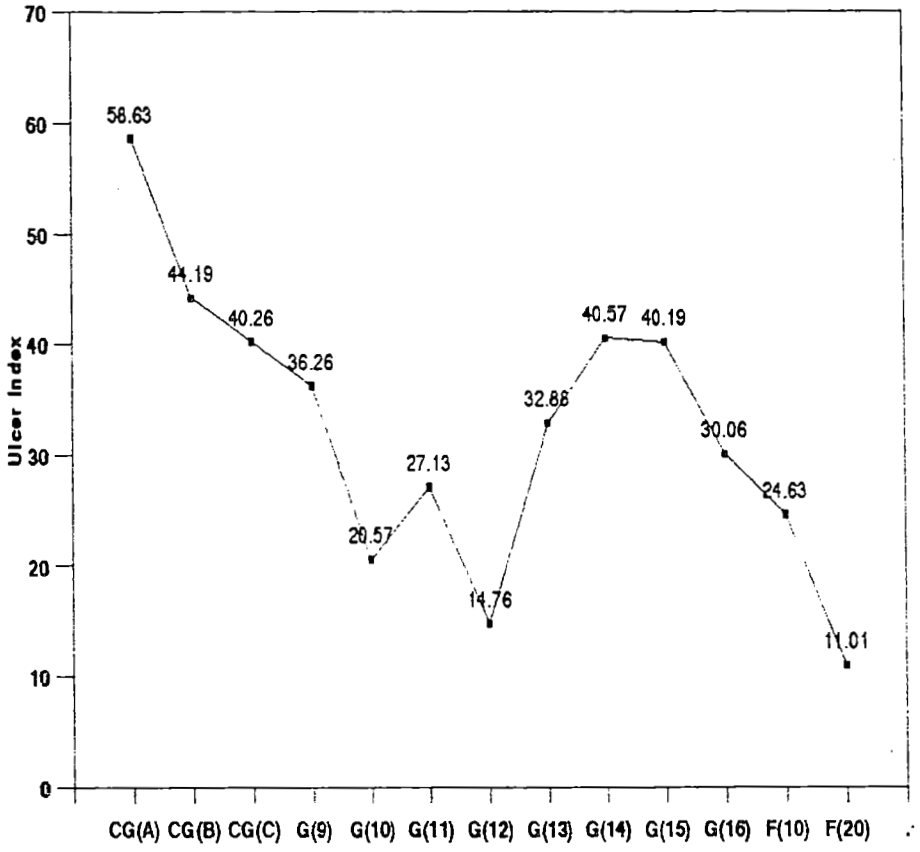
G(8) = P.W, 1000mg/Kg. B.Wt for 20 days

F(10) = Famotidine 40mg/Kg. B.Wt for 10 days

F(20) = Famotidine 40mg/Kg. B.Wt. for 20 days

G - Group, P.W - Powder, CG - Control group
B.Wt - Body weight, A.E - Alcoholic extract
F - Famotidine

Fig.2 COMPARATIVE ULCER INDEX OF MUSA(AAB GROUP,'NENDRAN') WITH FAMOTIDINE AND CONTROL GROUPS



CG(A) = Control group, Ulcer index (control) (Aspirin alone treated for 7 days)

CG(B) = Control group for 10 days (Natural healing)

CG(C) = Control group for 20 days (Natural healing)

G(9) = A.E, 500 mg/Kg. B.Wt for 10 days

G(10) = A.E, 1000 mg/Kg. B.Wt for 10 days

G(11) = A.E, 500 mg/Kg. B.Wt for 20 days

G(12) = A.E, 1000 mg/Kg. B.Wt for 20 days

G(13) = P.W, 1000 mg/Kg. B.Wt for 10 days

G(14) = P.W, 2000 mg/Kg. B.Wt for 10 days

G(15) = P.W, 1000 mg/Kg. B.Wt for 20 days

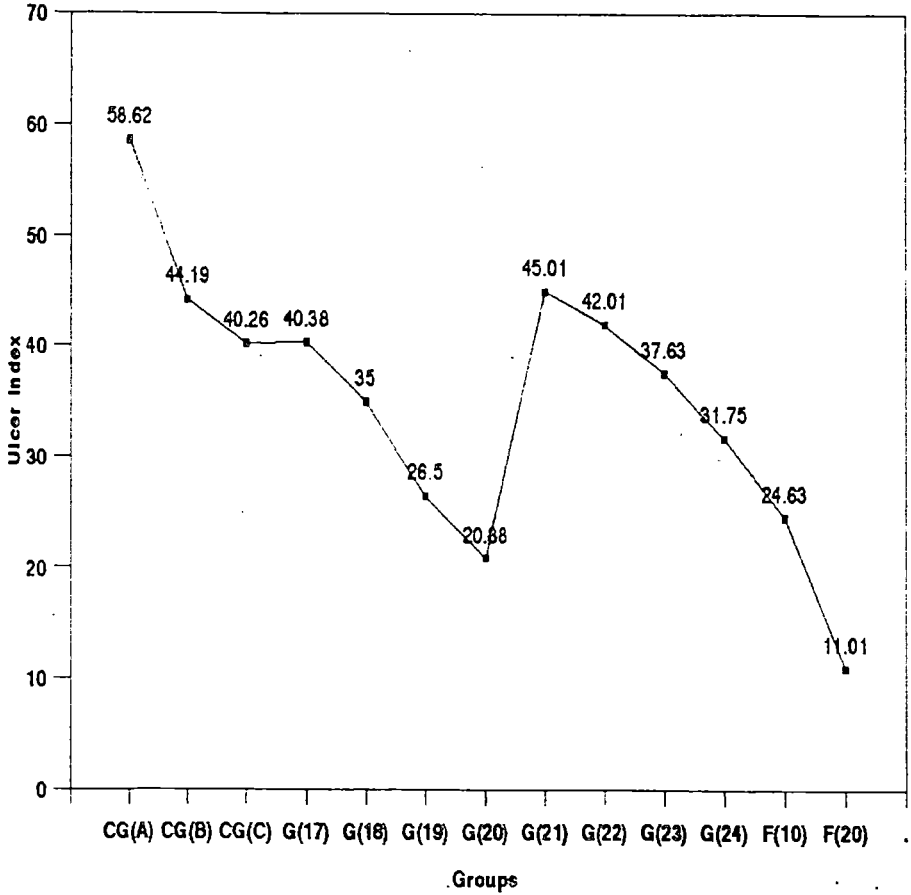
G(16) = P.W, 2000 mg/Kg. B.Wt for 20 days

F(10) = Famotidine, 40 mg/Kg. B.Wt for 10 days

F(20) = Famotidine, 40 mg/Kg. B.Wt for 20 days

G - Group, P.W - Powder, CG - Control group,
B.Wt - Body weight, A.E - Alcoholic extract, F - Famotidine

Fig.3 COMPARATIVE ULCER INDEX OF WITHANIA SOMNIFERA WITH FAMOTIDINE AND CONTROL GROUPS



CG(A) = Control group, Ulcer index (control) (Aspirin alone treated for 7 days)

CG(B) = Control groups for 10 days (Natural healing)

CG(C) = Control group for 20 days (Natural healing)

G(17) = A.E, 250 mg/Kg. B.Wt for 10 days

G(18) = A.E, 500 mg/Kg. B.Wt for 10 days

G(19) = A.E, 250 mg/Kg. B.Wt for 20 days

G(20) = A.E, 500 mg/Kg. B.Wt for 20 days

G(21) = P.W, 500 mg/Kg. B.Wt for 10 days

G(22) = P.W, 1000 mg/Kg. B.Wt for 10 days

G(23) = P.W, 500 mg/Kg. B.Wt for 20 days

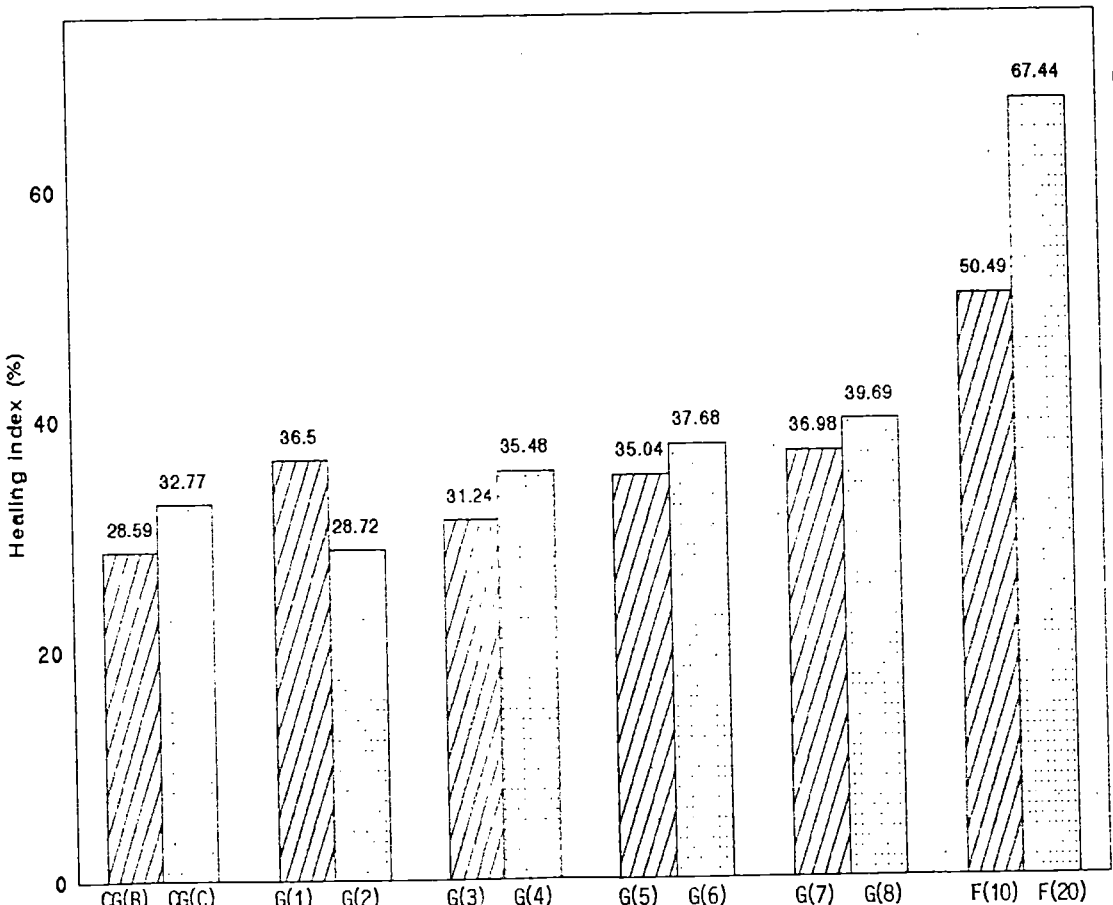
G(24) = P.W, 1000 mg/Kg. B.Wt for 20 days

F(10) = Famotidine 40mg/kg B.wt for 10 days

F(20) = Famotidine 40mg/kg B.wt for 20 days

G - Group, P.W - Powder, CG - Control Group,
B.Wt - Body weight, A.E - Alcohol extract, F - Famotidine

Fig.4 COMPARATIVE ANTI-ULCER EFFECT OF OCIMUM SANCTUM WITH FAMOTIDINE AND CONTROL GROUPS



CG(B) - Control group for 10 days (Natural healing)

CG(C) - Control group for 20 days (Natural healing)

G(1) - A.E, 250 mg/Kg. B.Wt for 10 days

G(2) - A.E, 500 mg/Kg. B.Wt for 10 days

G(3) - A.E, 250 mg/Kg. B.Wt for 20 days

G(4) - A.E, 500 mg/Kg. B.Wt for 20 days

G(5) - P.W, 500 mg/Kg. B.Wt for 10 days

G(6) - P.W, 1000 mg/Kg. B.Wt for 10 days

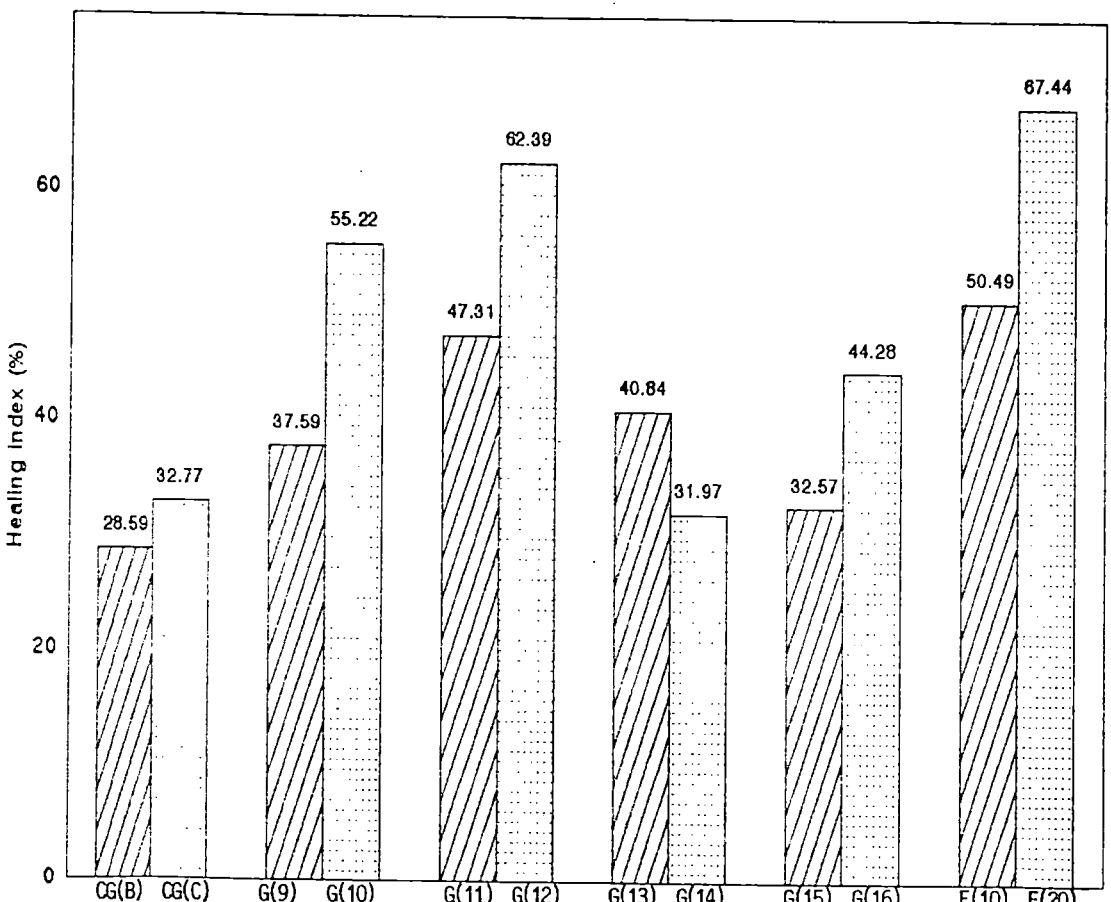
G(7) - P.W, 500 mg/Kg. B.Wt for 20 days

G(8) - P.W, 1000 mg/Kg. B.Wt for 20 days

F(10) - Famotidine 40 mg/Kg. B.Wt for 10 days

F(20) - Famotidine 40 mg/Kg. B.Wt for 20 days

Fig.5 COMPARATIVE ANTI-ULCER EFFECT OF MUSA (AAB GROUP, NENDRAN) WITH FAMOTIDINE AND CONTROL GROUPS



G - Group, P.W - Powder, CG - Control group, B.Wt - Body weight
A.E - Alcoholic extract, F - Famotidine

CG(B) = Control group for 10 days (Natural healing)

CG(C) = Control group for 20 days (Natural healing)

G(9) = A.E, 500 mg/Kg, B.Wt for 10 days

G(10) = A.E, 1000 mg/Kg. B.Wt for 10 days

G(11) = A.E, 500 mg/Kg. B.Wt for 20 days

G(12) = A.E, 1000 mg/Kg. B.Wt for 20 days

G(13) = P.W, 1000 mg/Kg. B.Wt for 10 days

G(14) = P.W, 2000 mg/Kg. B.Wt for 10 days

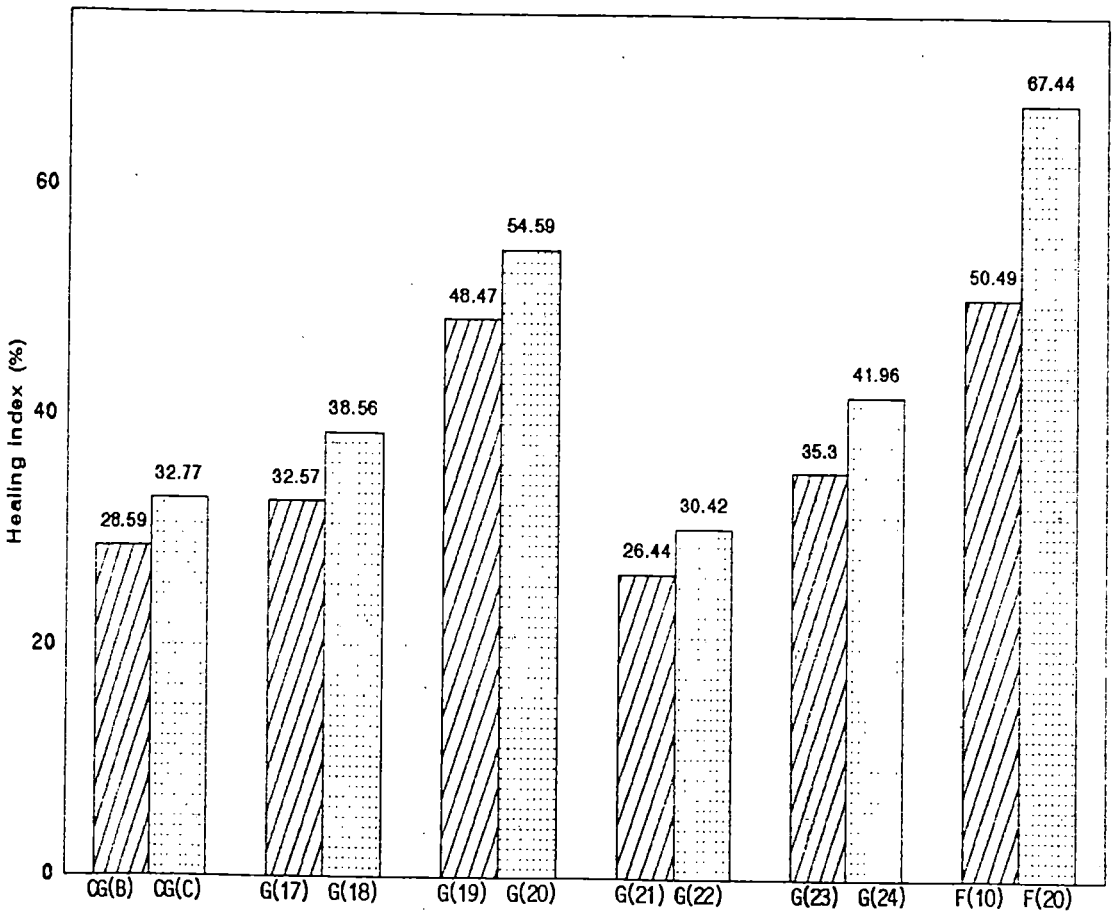
G(15) = P.W, 1000 mg/Kg. B.Wt for 20 days

G(16) = P.W, 2000 mg/Kg. B.Wt for 20 days

F(10) = Famotidine, 40 mg/Kg. B.Wt for 10 days

F(20) = Famotidine, 40 mg/Kg. B.Wt for 20 days

Fig.6 COMPARATIVE ANTI-ULCER EFFECT OF WITHANIA SOMNIFERA WITH FAMOTIDINE AND CONTROL GROUPS



G - Group, P.W - Powder, CG - Control group,
A.E - Alcoholic extract, F - Famotidine

CG(B) = Control group for 10 days (Natural healing)

CG(C) = Control group for 20 days (Natural healing)

G(17) = A.E, 250 mg/Kg. B.Wt for 10 days

G(18) = A.E, 500 mg/Kg. B.Wt for 10 days

G(19) = A.E, 250 mg/Kg. B.Wt for 20 days

G(20) = A.E, 500 mg/Kg. B.Wt for 20 days

G(21) = P.W, 500 mg/Kg. B.Wt for 10 days

G(22) = P.W, 1000 mg/Kg. B.Wt for 10 days

G(23) = P.W, 500 mg/Kg. B.Wt for 20 days

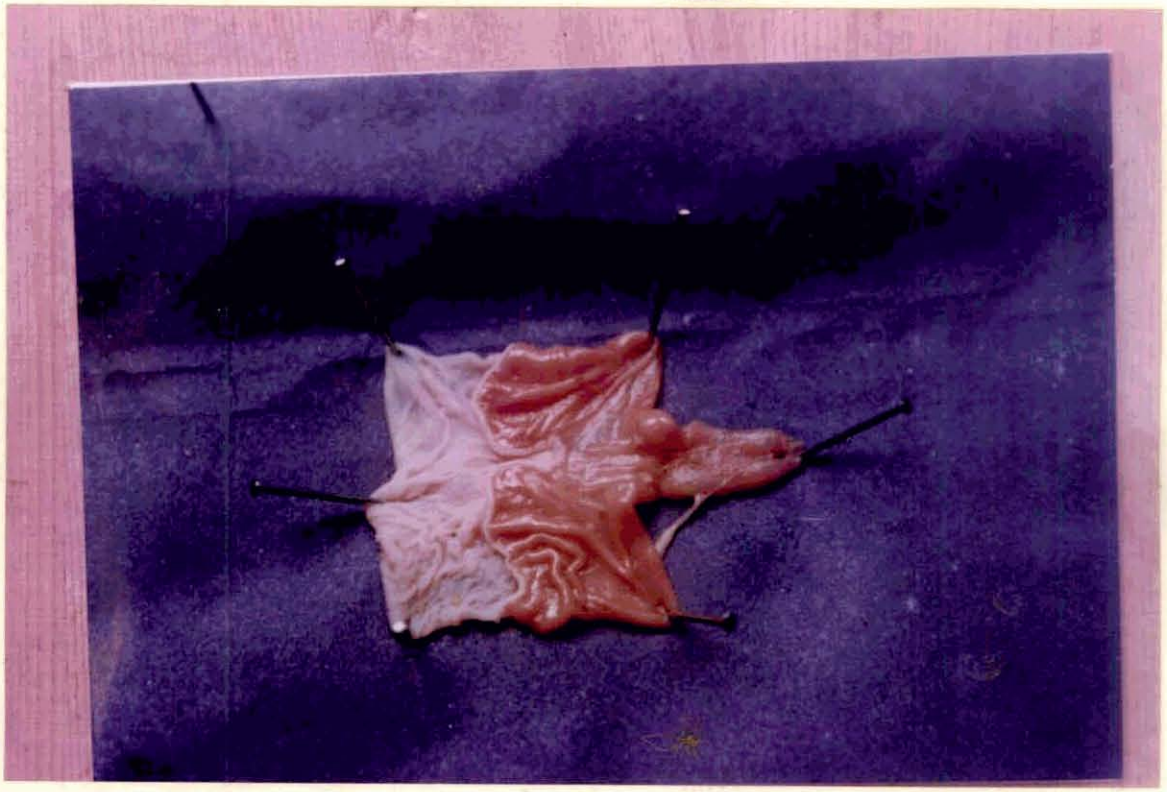
G(24) = P.W, 1000 mg/Kg. B.Wt for 20 days

F(10) = Famotidine 40mg/kg B.Wt for 10days.

F(20) = Famotidine 40mg/kg B.Wt for 20days.

Fig.7. Normal stomach

Fig.8. Stomach showing linear haemorrhages, shallow erosions and deep ulcers



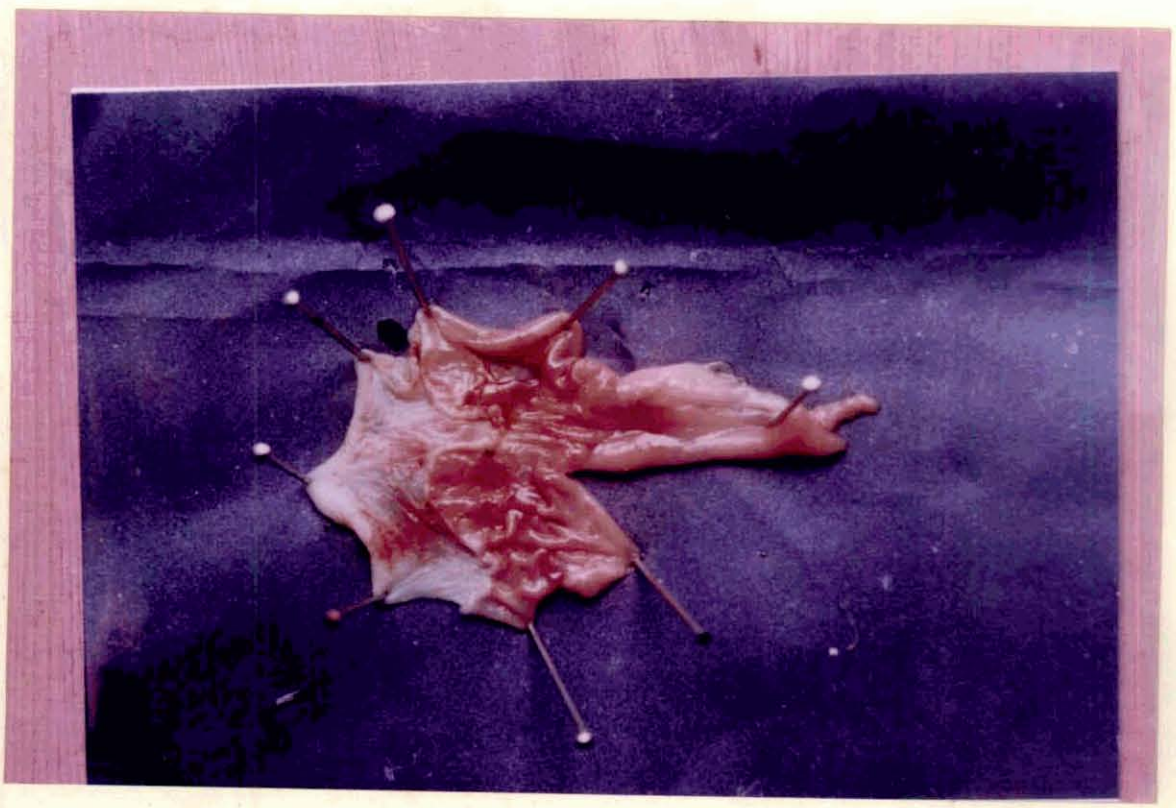
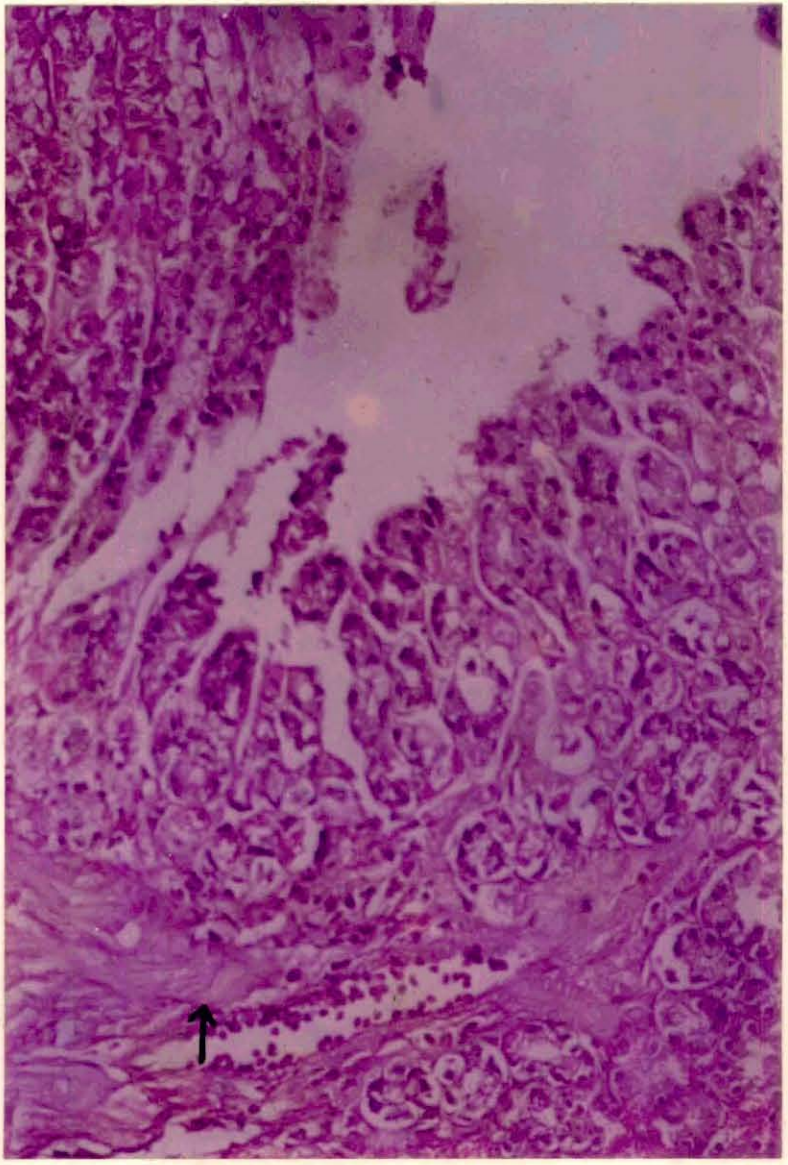


Fig.9. Stomach - Cross section of ulcer covered by an exudate consisting of mucous, fibrin and necrotic debris (H&Ex125)

Fig.10. Stomach - Cross section of mucosal ulcer outlined by a wedge shaped zone of coagulative necrosis (H&Ex125)



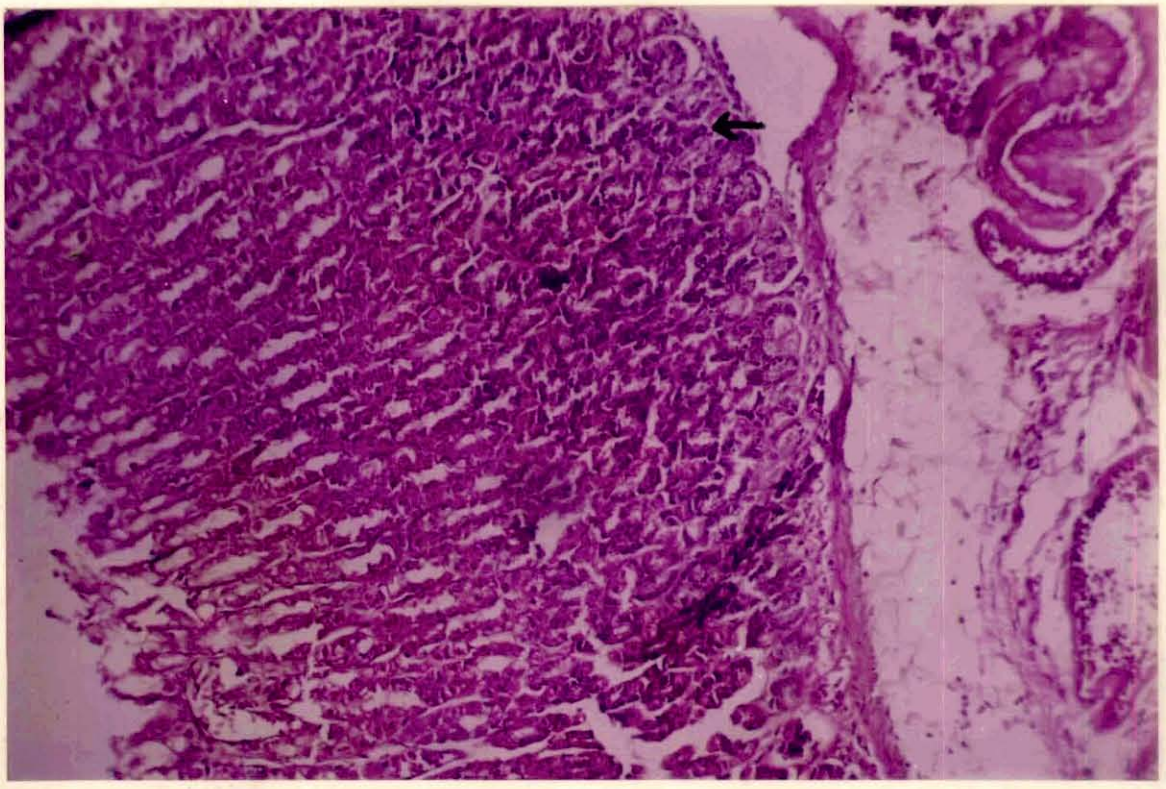
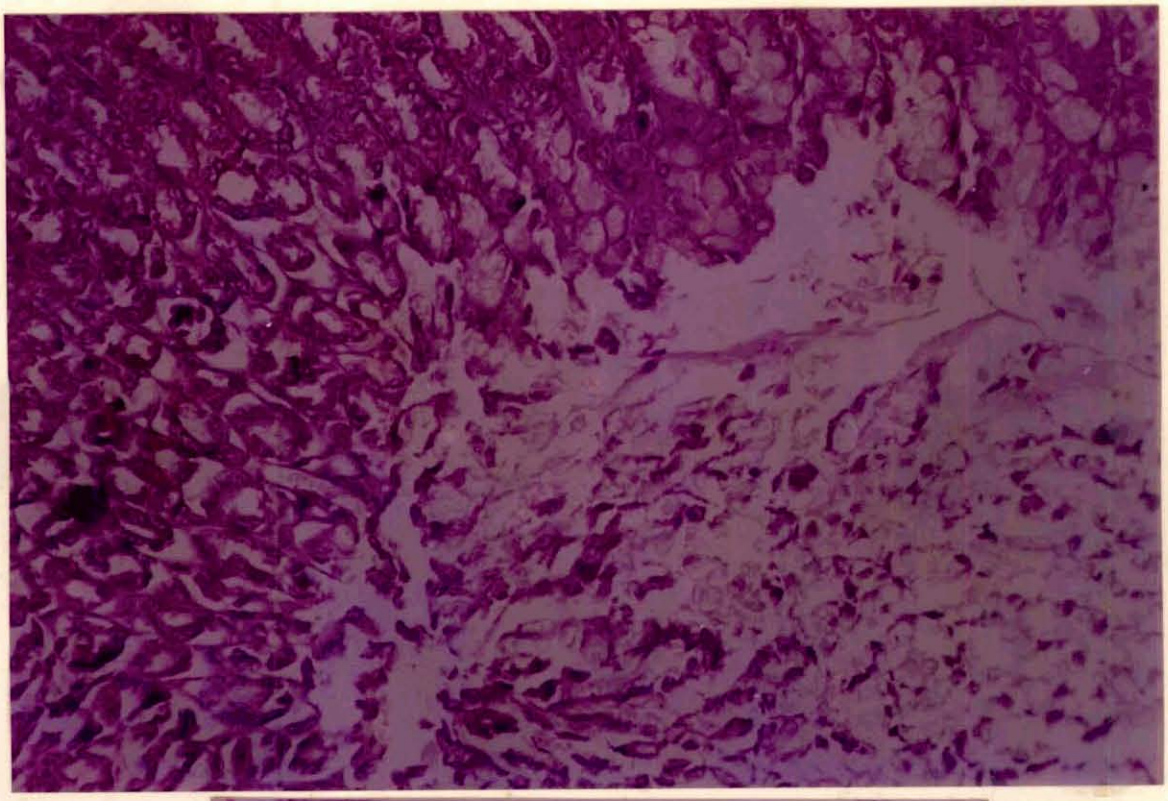


Fig.11. Stomach - Cross section of ulcer showing healing by granulation tissue proliferation (H&Ex125)

Fig.12. Stomach - Cross section of a healing ulcer showing infiltration by inflammatory cell population and formation of granulation tissue (H&Ex125)



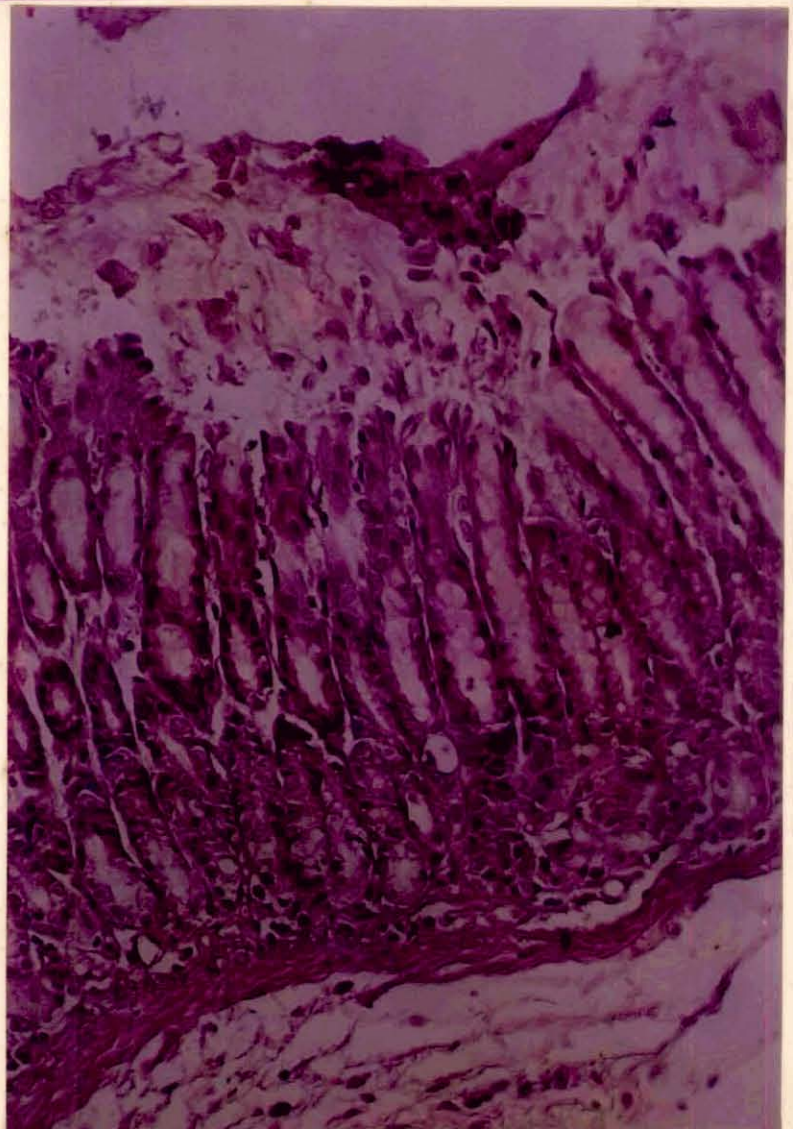
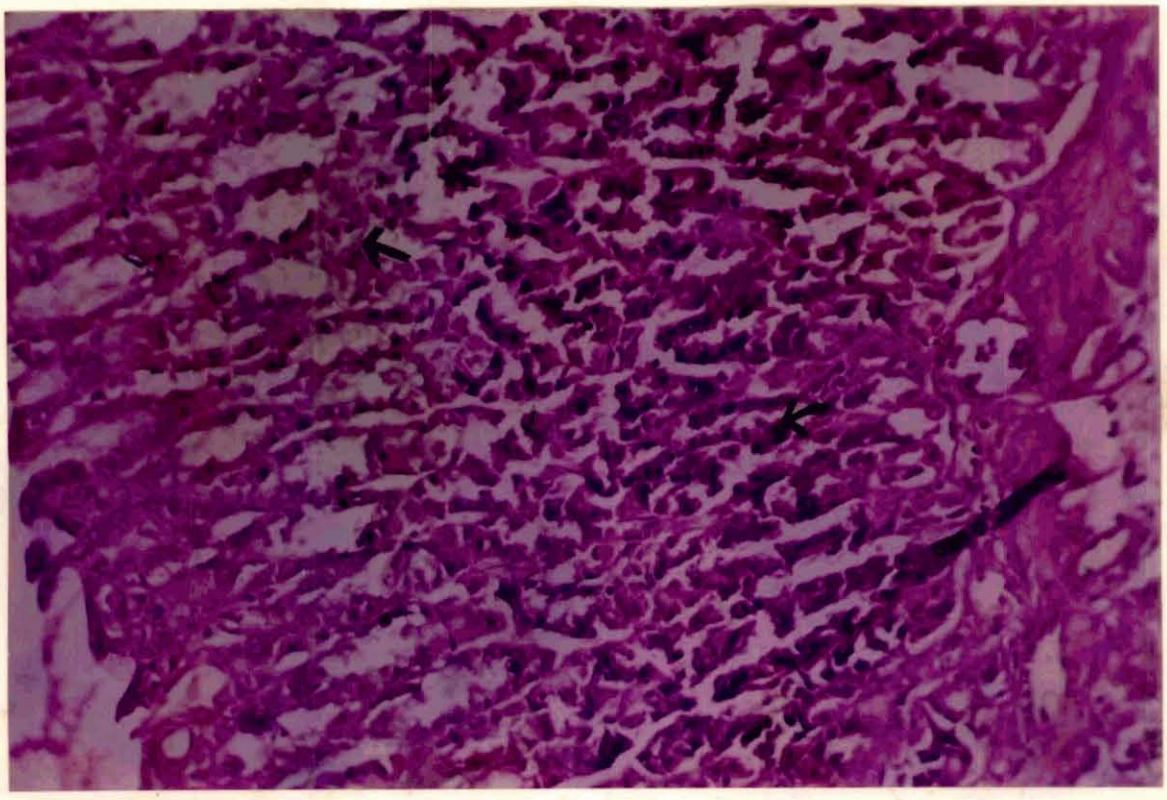


Fig.13. Stomach - Cross section of healing ulcer showing inflammatory cell infiltration and glandular hyper plasia (H&Ex125)

Fig.14. Stomach - Cross section showing re-epithelialization marked by goblet cell metaplasia and glandular hyperplasia (H&Ex125)



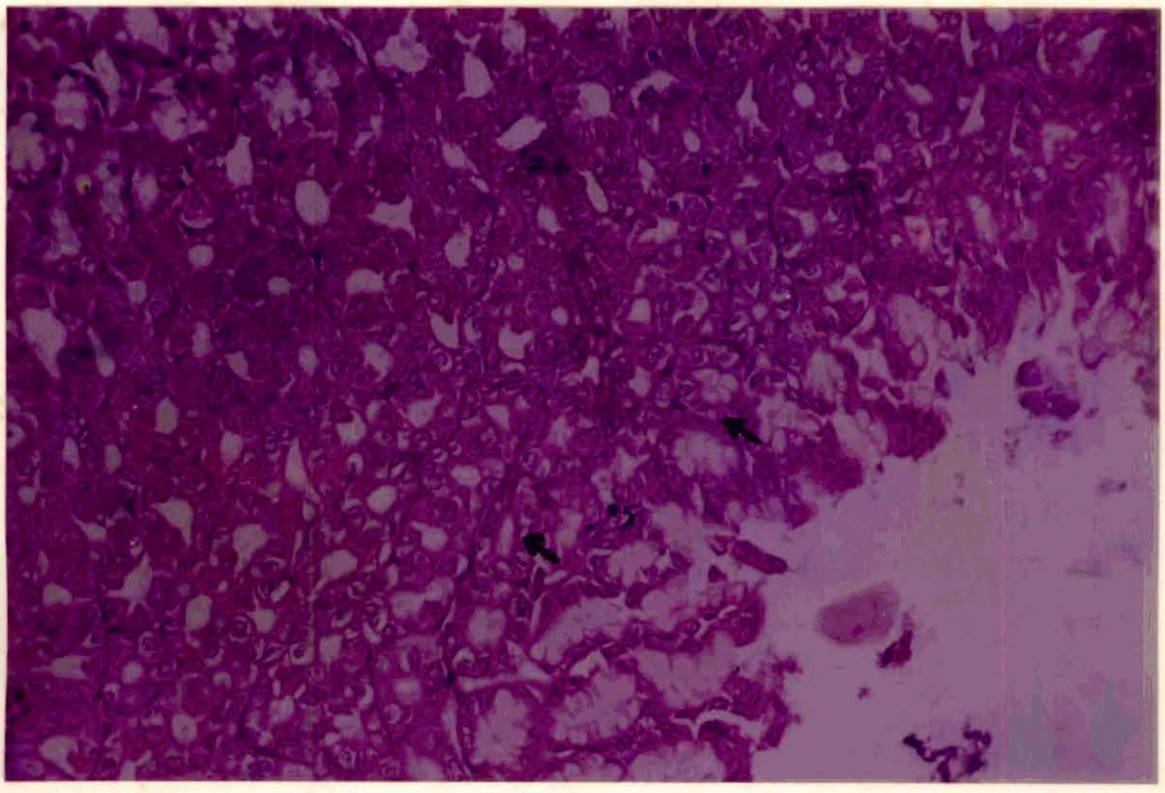
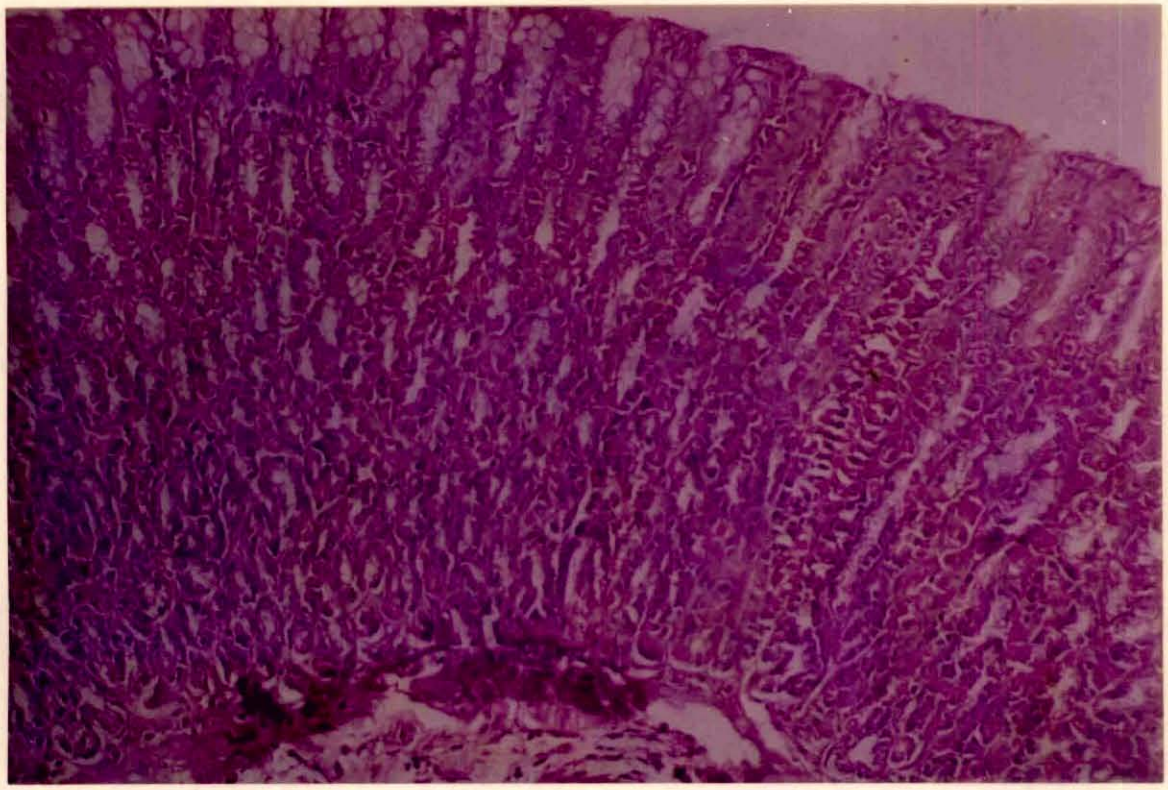


Fig.15. Stomach - Cross section showing almost complete re-epithelialization of the gastric mucosa (H&Ex125)



Discussion

DISCUSSION

5.1 *Ocimum sanctum* leaves

Very few studies appear to have been done to evaluate the anti-ulcerogenic property of *O. sanctum* in drug induced peptic ulcers. In the present study it was found that no groups with this plant produced healing comparable to famotidine at the dose rate of 40 mg/kg body weight for 10 days and 20 days, but in all groups the ulcer index was significantly reduced when compared with that of aspirin treated controls.

Among the various groups under this plant, *O. sanctum* powder at the dose rate of 1000 mg/kg body weight for 20 days produced maximum healing (39.692 ± 4.46). But the healing produced were not dose-dependent and not comparable with famotidine at the dose rate of 40 mg/kg body weight for 10 days and 20 days which indicates that *O. sanctum* has no significant anti ulcerogenic effect.

According to Mandal *et al.* (1993) steam distilled product of *O. sanctum* leaves at the dose rate of 0.1 ml per 100 gm body weight orally in rats reduced the ulcer index, free and total acidity when administered proplylactically. But did not study the curative property (healing) of *O. sanctum* leaves.

Mechanism by which *O. sanctum* leaves reduced ulcerindex could not be ascertained from the present investigation.

Bhargava and Singh (1981) found that pretreated alcoholic extract of *O. sanctum* whole plant at the dose rate of 100 mg/kg and 200 mg/kg body weight administered orally significantly reduced the stress induced and aspirin induced gastric ulcers in rats. He suggested that the *O. sanctum* whole plant induced a state of non-specific, increased resistance to a variety of stress induced biological changes in animals.

Medirátta *et al.* (1988) observed that *O. sanctum* significantly inhibited antigen induced histamine release from the peritoneal mast cells of sensitized rats in vitro. This may be the probable mechanism of anti-ulcer action of *O. sanctum* leaves.

Vanisree *et al.* (1995) studied the effect of pretreatment with a suspension of *O. sanctum* leaf powder at 200 mg/kg body weight orally for 8 days on Hcl-ethanol induced gastric lesion in rats and found an increase in volume and acidity of gastric juice and a decrease in peptic activity occurred in Hcl-ethanol exposed mucosa, similarly the increase in lipid peroxidation, decrease in activity of antioxidant and decrease in protein and glycoprotein content which occurred in the

ulcerated mucosa was not seen in the *O. sanctum* treated mucosa.

5.2 *Musa* (AAB group, "Nendran") mature and unripe fruit

The alcoholic extract of *Musa* (AAB group "Nendran") mature and unripe fruit produced a dose-dependent increase in anti-ulcer activity while healing effect produced by banana powder are not-dose-dependent but the effects are comparable. All the groups produced a significant decrease in ulcer index when compared to aspirin treated control group (CG(A)) and healing better than control group for 10 days.

Waalkes *et al.* (1958) suggested the anti-ulcer activity of *Musa* is due to its high serotonin content.

Sanyal *et al.* (1961) observed that pretreatment of banana emulsion reduced gastric secretion and thus prevent chronic ulceration and perforation induced by repeated injection of histamine. He suggested the anti-ulcer activity of banana due to its high serotonin content.

Based on the study by Waalkes *et al* (1958), Sanyal *et al.* (1963) compared the anti-ulcer activity of banana powder at 1 gm/kg body weight with aluminium hydroxide (antacid) at 1 gm/kg body weight. The result clearly indicate that banana powder helps in prevention and healing of phenyl-butazone

induce ulcers by its demulcent action and are comparable with aluminium hydroxide.

In the present study, comparison was done with more powerful anti-ulcer drug, Famotidine and instead of pretreatment with banana, powder and alcoholic extract was given after induction of ulcers with aspirin for 7 days. Still banana powder at 1 gm/kg body weight for 10 days and 20 days significantly reduced the ulcerindex but the healing produced are not comparable with famotidine at the dose rate of 40 mg/kg for 10 days and 20 days.

Study conducted by Best *et al.* (1984) suggest that unripe banana powder and alcoholic extract had both preventive and curative effect on aspirin induced gastric ulcers. According to him the mode of action of the banana appear to be due to stimulation of mucosal cell growth, promotion of mucus secretion.

Ghosal and Saini (1984) isolated two steryl-acyl glucoside active against peptic and duodenal ulcers from the fruits of *Musa paradisiaca*.

Goel *et al.* (1986) found that *Musa* powder at 1 gm/kg body weight increased mucosal thickness in experimentally induced gastric ulcers in rats.

Mukhopadhyay *et al.* (1987) suggested that plantain fruit powder at the dose rate of 500 mg/kg body weight BID appear to increase gastric mucosal resistance possibly by enhancing the production of cellular mucus.

Goel *et al.* (1989) found that banana tend to increase the eicosanoid accumulation in human colonic mucosa by increasing the availability of arachidonate which is responsible for the anti-ulcer activity of *Musa paradisiace* against NSAID induced ulcers.

The present study advocate the use of banana powder and alcoholic extract as an anti-ulcer agent.

5.3 *W. somnifera* root

All groups significantly reduced the ulcer index when compared with aspirin treated control. *W. somnifera* alcoholic extract and powder produced a dose-dependent increase in healing. *W. somnifera* alcoholic extract at the dose rate of 250 mg/kg body weight for 20 days (48.473 ± 7.67), alcoholic extract at the dose rate of 500 mg/kg body weight for 20 days (54.586 ± 5.74) and *W. somnifera* powder at the dose rate of 1000 mg/kg body weight for 20 days (41.957 ± 6.36) produced healing comparable to famotidine at the dose rate of 40 mg/kg for 10 days and 20 days.

Work of Bhattacharya *et al.* (1987) also indicated the anti-ulcerogenic effect of *W. somnifera* on restrained stress induced gastric ulcers in rats where it produced significant reduction in number of ulcers per stomach from 10.6 ± 1.9 to 1.2 ± 0.9 and ulcerindex from 46.0 to 11.6.

The exact mechanism of the anti-ulcerogenic effect of *W. somnifera* was not studied but Sahni (1993) proposed that the anti-ulcerogenic effect of *W. somnifera* is due to the inhibition of histamine and serotonin activity in CNS.

5.4 Haematological findings

None of the treatment groups produced any significant changes in the haematological parameters.

5.5 Histopathological findings

Grossly, the lesion observed were petechial haemorrhages, shallow erosions and deep ulcers. More than one type of lesion was observed in the stomach, but in a large number of cases the lesions were petechial haemorrhages and shallow erosions. The haemorrhagic ulcers were linear, mostly occurring in the glandular corpus (Fig.8).

Microscopically, examination of the mucosal ulcers revealed on cross section a roughly wedge shaped zone of

coagulative necrosis characterized further by capillary haemorrhages in the lamina propria, desquamation of the epithelial layer and development of a superficial area of ulceration. The lesions whether shallow erosion or deep ulcers were covered by an exudate consisting of mucous, fibrin and necrotic debris (Fig.9). The edges of the lesions were raised above the surrounding tissue and there was submucosal odema in majority of the cases (Fig.10).

The ulcers which were largely limited to the glandular corpus resolved by granulation tissue formation and subsequently by reepithelialization.

The granulation tissue consisted of proliferating capillaries along with the proliferation of fibroblats (Fig.11). Besides, there was infiltration by a mixed inflammatory cell population and the healing lesion was overlain by a usually thin layer of necrotic debris (Figs. 12 and 13). The phase of reepithelialization was characterized by mucous-secreting goblet cell metaplasia and glandular hyperplasia (Fig.14). The re-epithelialization was almost complete and the functional morphology was almost restored in the Famotidine treated group (Fig.15). This was followed to a lesser degree by the *Musa* treated group and next in order by the Withania treated group. Of the four treatment groups, the response was found to be minimal with Ocimum.

Similar lesions were observed by George and Somvanshi (1987) in rabbits with gastric ulcers.

Therefore the present observations stress on the need for further studies to evaluate the mechanism of action of anti-ulcer activity and more studies to be directed to evaluate the curative effect by administering the plant extract for longer period rather than evaluating the prophylatic activity which was done in most of the studies under these plants. Also studies on toxic effects of the plants has to be done to evaluate the potential damage it can cause to the various system/organs when administered for longer duration.

Summary

SUMMARY

The present study was undertaken in albino rats to evaluate the anti-ulcer activity of powder and alcoholic extract of *Ocimum sanctum* (Thulasi) leaf, *Musa* (AAB group "Nendran") mature, unripe fruit and *Withania somnifera* (Amukkiram) root in comparison with Famotidine, a standard anti-ulcer drug.

The gastric ulcer was induced by using aspirin at the dose rate of 200 mg/kg body weight orally for 7 days with restricted feeding and water *adlib*. Two hundred and thirty rats weighing 150-200 gm body weight of either sex were used for the study with eight rats in each group. The rats were treated with powder/alcoholic extract of the plants under study for 10 days and 20 days after 7 days of aspirin administration. The doses of different plant preparations were shown as in Table 1. All the rats were sacrificed on 18th and 28th day after 10 days and 20 days of treatment with powder/alcoholic extract of plant preparations respectively. The ulcer index and healing index was calculated by applying the formulae

$$\text{Ulcer index} = \text{Number of ulcers} + \text{Ulcer score} + \frac{\text{\% incidence}}{\text{Number of animals}}$$

$$\text{Healing index} = \frac{\text{Ulcer index (control) CG(A)} - \text{Ulcer index (drug)}}{\text{Ulcer index (control) [CG(A)]}} \times 100$$

Number of ulcers on the gastric mucosa were counted with the help of magnifying lense. Ulcer score was determined based on arbitrary scoring system

The control group A [CG(A)] was destroyed on the 8th day after 7 days of aspirin administration alone and ulcer index was determined.

Famotidine, a standard anti-ulcer drug was given orally at the dose rate of 40 mg/kg body weight for 10 days and 20 days respectively. The anti-ulcer activity of plants under study was compared with Famotidine.

Control group B [CG(B)] and control group C [CG(C)] were given aspirin at the dose rate of 200 mg/kg body weight for 7 days, and from 8th day onwards they were maintained by normal feeding and watering for 10 days and 20 days respectively to assess the natural healing.

Haematological study of both the experimental and control groups were done after sacrificing the rats to assess any change in the erythrocyte count, total leucocyte count, differential count and haemoglobin count.

Histopathological study was also done with regard to the various ulcer lesions and healing process of ulcers of both control and experimental groups.

Result of the present study with *O. sanctum* leaf powder and alcoholic extract showed that none of the experimental groups produced statistically significant healing effects comparable to Famotidine groups.

Healing obtained with *O. sanctum* alcoholic extract at the dose rates of 500 mg/kg body weight for 10 days, and 250 mg/kg body weight for 20 days are comparable only with the control groups for 10 days and 20 days respectively. Rest of the groups under this plant produced significantly higher healing efficiency than control groups for 10 days and 20 days.

Similarly results obtained with *Musa* (AAB group "Nendran") mature, unripe fruit showed that all experimental group under study with this plant have significantly higher healing effect than control groups for 10 days.

The alcoholic extracts of *Musa* (AAB group "Nendran") unripe fruit at the dose rate of 1000 mg/kg body weight for 10 days, 500 mg/kg body weight for 20 days and 1000 mg/kg body weight for 20 days produced healing comparable to Famotidine groups treated for 10 days and 20 days respectively.

Musa (AAB group "Nendran") unripe fruit powder at the dose rate of 1000 mg/kg body weight for 10 days and 2000 mg/kg body weight for 20 days produced healing comparable to Famotidine administered for 10 days.

Musa (AAB group "Nendran") alcoholic extract at the dose rate of 500 mg/kg body weight for 10 days, powder at the dose rate of 2000 mg/kg body weight for 10 days and 1000 mg/kg body weight for 20 days produced healing comparable with control group for 10 days and 20 days respectively.

The ulcer healing effect obtained with *W. somnifera* root indicated that alcoholic extracts at the dose rate of 250 mg/kg body weight for 20 days and 500 mg/kg body weight for 20 days and powder at the dose rate of 1000 mg/kg body weight for 20 days are comparable with Famotidine administered for 10 days and 20 days respectively.

All other experimental groups under study with his plant produced healing comparable to Famotidine administered for 10 days.

The study of haematological parameters of all the groups revealed no significant changes and all values fall within the normal ranges of blood values for the species under study.

The histopathological study of ulcer revealed lesions with roughly wedge shaped zone of coagulative necrosis characterised further by capillary haemorrhages in the lamina propria, desquamation of the epithelial layer and development of a superficial areas of ulceration. The ulcers were largely limited to the glandular corpus.

Healing of ulcers were by granulation tissue formation and subsequently by re-epithelialization. The granulation tissue consisted of proliferating capillaries along with the proliferation of flibroblasts. Besides there was infiltration by a mixed inflammatory cell population and healed lesions were overlain by a usually thin layer of necrotic debris.

O. sanctum powder at the doses rate of 1000 mg/kg body weight for 20 days produced maximum healing. But the healing produced were not dose dependent and not comparable with Famotidine groups administered for 10 days and 20 days which indicate that *O. sanctum* has no significant anti-ulcerogenic effect.

The alcoholic extract of *Musa* (AAB group, "Nendran") unripe fruit produced a dose-dependent increase in anti-ulcer activity while healing effect produced by *Musa* powder are not dose-dependent but the effects are comparable with Famotidine groups administered for 10 day and 20 days respectively. All the groups produced a significant decrease in ulcer index when compared.with aspirin treated control group [CG(A)] and healing better than control group [CG(B)] for 10 days. The present study advocates the use of *Musa* powder and alcoholic extract as an effective anti-ulcer agent.

All experimental groups under *W. somnifera* root significantly reduced the ulcer index when compared with aspirin treated control [CG(A)]. Alcoholic extracts of *Withania*

somnifera produced a dose-dependent increase in anti-ulcer activity while healing effect produced *W. somnifera* root powder are not dose-dependent.

The mechanism of anti-ulcerogenic effect and toxicity of these plants need further detailed study.

Table 1. Doses of *Ocimum sanctum* leaves, *Musa* (AAB group "Nendran") mature and unripe fruit, *Withania somnifera* root and Famotidine (in mg/kg orally) were fixed as below

Plants	Alcoholic extracts				Powder			
	10 days treatment		20 days treatment		10 days treatment		20 days treatment	
Ocimum sanctum	G(1) 250	G(2) 500	G(3) 250	G(4) 500	G(5) 500	G(6) 1000	G(7) 500	G(8) 1000
Musa (AAB group Nendran)	G(9) 500	G(10) 1000	G(11) 500	G(12) 1000	G(13) 1000	G(14) 2000	G(15) 1000	G(16) 2000
Withania somnifera	G(17) 250	G(18) 500	G(19) 250	G(20) 500	G(21) 500	G(22) 1000	G(23) 500	G(24) 1000
Famotidine	10 days treatment F(10) 40 mg/kg p.o				20 days treatment F(20) 40 mg/kg p.o			

Control group A [CG(A)] - Aspirin treated controls (200 mg/kg b.wt)

Control group B [CG(B)] - Natural healing for 10 days

Control group C [CG(C)] - Natural healing for 20 days

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ANTI - ULCER ACTIVITY OF *Ocimum sanctum* (Thulasi) *Musa* (AAB Group, "Nendran") AND *Withania somnifera* (Amukkiram) IN RATS

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ABSTRACT OF A THESIS

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ABSTRACT

The study was conducted in adult albino rats of either sex to assess the comparative anti-ulcer effect of powder and alcoholic extract of *Ocimum sanctum* (leaves), *Musa* (AAB group, "Nendran", mature, unripe fruit) and *Withania somnifera* (root) with Famotidine, at different dose levels given orally for 10 days and 20 days after induction of gastric ulcers with aspririn at the dose rate of 200 mg/kg body weight for 7 days orally. The doses of different plant preparation were fixed arbitarily (Table 1).

Table 1. Doses of *Ocimum sanctum* leaves, *Musa* (AAB group "Nendran") mature and unripe fruit, *Withania somnifera* root and Famotidine (in mg/kg orally) were fixed as below

Plants	Alcoholic extracts				Powder			
	10 days treatment		20 days treatment		10 days treatment		20 days treatment	
Ocimum sanctum	G(1) 250	G(2) 500	G(3) 250	G(4) 500	G(5) 500	G(6) 1000	G(7) 500	G(8) 1000
Musa (AAB group Nendran)	G(9) 500	G(10) 1000	G(11) 500	G(12) 1000	G(13) 1000	G(14) 2000	G(15) 1000	G(16) 2000
Withania somnifera	G(17) 250	G(18) 500	G(19) 250	G(20) 500	G(21) 500	G(22) 1000	G(23) 500	G(24) 1000
Famotidine	10 days treatment F(10) 40 mg/kg p.o				20 days treatment F(20) 40 mg/kg p.o			

Control group A [CG(A)] - Aspirin treated controls (200 mg/kg b.wt)

Control group B [CG(B)] - Natural healing for 10 days

Control group C [CG(C)] - Natural healing for 20 days

Control group A [CG(A)] was given aspirin at the dose rate of 200 mg/kg body weight for 7 days and on the 8th day, the rats were sacrificed and number of ulcers and severity (ulcer score)

were determined with the help of magnifying lens. The ulcer index and healing index were calculated based on the formulae:

$$\text{Ulcer index} = \text{Number of ulcers} + \text{Ulcer score} + \frac{\text{\% incidence}}{\text{Number of animal}}$$

$$\text{Healing index} = \frac{\text{Ulcer index (control) CG(A)} - \text{Ulcer index (drug)}}{\text{Ulcer index (control) CG(A)}} \times 100$$

The control group B [CG(B)] and control group C [CG(C)] were administered aspirin orally for 7 days and they were maintained by normal feeding and watering without any treatment for 10 days and 20 days respectively to assess natural healing.

Famotidine was taken as standard drug and given orally at the dose rate of 40 mg/kg body weight for 10 days and 20 days respectively. The anti-ulcer activity of plants under study was compared with Famotidine.

Haematological parameters such a erythrocyte count, total leukocyte count, differential count and haemoglobin count were determined to assess any changes in the haemogram.

Histopathological study was also conducted to evaluate the severity of ulceration and healing process.

The result of the present study with *O. sanctum* leaf powder and alcoholic extract showed that none of the experimental

groups produced statistically significant healing effects comparable to Famotidine groups.

Observation with *Musa* (AAB group, "Nendran") unripe fruit powder and alcoholic extract have indicated that all experimental group under study with the plant have better healing effect than control group for 10 days.

The alcoholic extract of *Musa* (AAB group, "Nendran") at the dose rate of 1000 mg/kg body weight for 10 days, 500 mg/kg body weight for 20 days and 1000 mg/kg body weight for 20 days produced healing comparable to Famotidine at the dose rate of 40 mg/kg body weight for 10 days and 20 days respectively.

The result of healing obtained with *W. somnifera* root indicate that alcoholic extracts at the dose rate of 250 mg/kg body weight for 20 days and 500 mg/kg body weight for 20 days and powder at the dose rate of 1000 mg/kg body weight for 20 days are comparable with Famotidine groups administered for 10 days and 20 days respectively. All other experimental group under study with this plant produced healing comparable to Famotidine administered for 10 days.

Haematological study revealed no significant change and all values fall within the normal range of blood value for the species under study.

Histopathological study revealed various ulcer lesions and healing process.