# Role of *Butea Frondosa* in Ameliorating Gastric Markers in Induced Gastric Lesions of Rats

D. BANJI*, OTILIA J. F. BANJI, MEENU SINGH[1] AND A. R. ANNAMALAI[2]
Department of Pharmacology, Nalanda College of Pharmacy, Cherlapally-508 001, [1]Balaji Institute of Pharmaceutical Sciences, Ananthpur-515 001, [2]Rajah Muthiah Medical College, Annamalai University, Annamalai Nagar-608 002, India



**The study evaluated the ability of the alcohol extract of *Butea frondosa* to protect the gastro-duodenal lining from injury inflicted by acetic acid and pyloric ligation in rats. The induced gastric lesions lead to the generation of alkaline phosphatase and pepsin, which serve as important markers of gastric damage. Alcohol extract of *Butea frondosa* was administered in doses of 10, 100, 300 and 500 mg/kg as a single schedule and for the time dependent studies in a dose of 100 mg/kg for 7, 14, 21 and 28 days, respectively. Our studies reveal a decline in the formation of alkaline phosphatase and pepsin with 300 and 500 mg/kg of the extract and following treatment for 21 and 28 days, respectively. Extract of *Butea frondosa* produces significant diminution in the formation of gastric markers implying possible gastro-protective action.**

**Key words: Alkaline phosphatase, *Butea frondosa*, markers, pepsin**

Peptic ulcer disease is a health hazard both in terms of morbidity and mortality. The development of gastric ulcers occurs due to acid pepsin mixture and the breakdown of mucosal defense. Mucus like alkaline secretions, mucosal hydrophilicity, rapid epithelial cell renewal, rich mucosal blood flow, mucosal sulphydryls and increased resistance of gland cells in deep mucosa to acid and peptic activity are the mechanisms involved in mucosal defense[1]. Pepsin is a proteolytic enzyme capable of causing mucosal erosion and ulcerations. Alkaline phosphatase (ALP) catalyses the hydrolysis of various phosphate esters at alkaline pH and can be considered to be a biomarker for gastric damage[2,3].

*Butea frondosa* Koen. Ex Roxb. is used traditionally against ulcers, skin diseases, herpes, acne, while internally it is used against gas colics, worms and piles. Phytochemically, it is rich in flavanoids, terpenoids and lipid constituents[4]. The present study was designed to investigate the impact of

***Address for correspondence**
E-mail: davidbanji@gmail.com

aqueous extract of the stem of *Butea frondosa* (AESBF) on peptic and alkaline phosphatase activity.

The fresh stem of *Butea frondosa* was collected locally, identified by a Botanist and a voucher specimen is maintained in our herbarium bearing number BF/2. The stem of the plant was dried in the shade and coarsely powdered. 50 g of stem powder was exhaustively extracted with 200 ml of water under reflux by maintaining the temperature between 40-50° for 32 h. The extract was evaporated under vacuum (4.3%, w/v) and stored in a refrigerator. The required quantity of extract was suspended in 1.0% aqueous solution of tragacanth and used.

Albino rats weighing 180-200 g of either sex were used in this study. They were divided into 6 groups for the dose dependent and time dependent studies with each group containing 6 animals. Clearance to carry out the work was obtained from the Institutional animal ethical committee bearing no. Ref: IAEC/Clear/27/2004–05 dated 03/02/2005. At a dose of 1.6 g/kg the aqueous extract of the stem of *Butea frondosa* did not exhibit any toxic effects as shown by previous workers[5]. Hence doses of 10, 100, 300 and 500 mg/kg were selected for our study.

Albino rats were fasted for 24 h and provided with water *ad libitum*. Ulceration induced by the administration of 15% v/v acetic acid (0.05 ml/rat) plus pyloric ligation. Group-1 animals received 1ml of 1% aqueous tragacanth orally, Group-2 received famotidine (3 mg/kg) orally. Groups-3, 4, 5 and 6 received 10, 100, 300, 500 mg/kg of aqueous extract of stem of *Butea frondosa* (AESBF) 30 min before administration of acetic acid. Administration of extract was done as a single dose and for 7, 14, 21 and 28 day. Half an hour after the last dose a midline incision was made under the influence of enflurane anesthesia, pylorus was ligated and abdominal wall was closed in two layers and sutured. Three hours after pyloric ligation the animals were sacrificed. Stomach was dissected and opened along the greater curvature[6], contents drained into a graduated centrifuge tube and peptic activity of gastric juice was determined[7].

The excised wound tissue was immersed in 4 ml buffer solutions and ground in a mortar. It was centrifuged for 10 min and the supernatant was used for the determination of alkaline phosphatase using the method of Kind and King[8]. The results are expressed as mean±SEM and analyzed using one way ANOVA followed by Dunnet's multiple comparison tests. $P<0.05$ was considered statistically significant.

Extensive elevation in peptic and ALP activity was observed in the control group. The reference standard famotidine, produced lowering in the peptic activity to 43.51±1.06 IU and ALP activity to 29.27±0.4 IU compared with the control ($P<0.05$). A reduction in peptic activity was observed with 10 and 100 mg/kg body weight of AESBF, however 300 and 500 mg/kg of AESBF produced a comparable effect to the standard and a significant lowering of peptic activity compared with the control ($P<0.01$). A significant decrease in ALP activity was observed with 300 and 500 mg/kg body weight of AESBF (Table 1).

Treatment of 100 mg/kg of AESBF for 7, 14, 21 and 28 days significantly decreased the concentration of pepsin. However, peptic activity was significantly diminished following 28 days treatment. Attenuation in the levels of ALP was observed following the same duration of treatment (Table 2).

**TABLE 1: EFFECT OF AQUEOUS EXTRACT OF STEM OF *BUTEA FRONDOSA* ON PEPTIC AND ALKALINE PHOSPHATASE ACTIVITIES IN INDUCED GASTRIC LESIONS IN RATS**

| Treatment | Dose (mg/kg) | Peptic activity (IU) | Alkaline phosphatase (IU) |
|---|---|---|---|
| Control | -- | 69.85±0.75 | 40.58±0.47 |
| Famotidine | 3 | 43.51±1.06* | 29.27±0.48* |
| AESBF | 10 | 54.33±0.95 | 38.05±0.50 |
| AESBF | 100 | 45.85±1.17* | 35.01±0.34 |
| AESBF | 300 | 41.19±1.20* | 31.23±0.46* |
| AESBF | 500 | 40.30±0.98* | 28.58±0.45* |

Values seen are mean±SEM, $n=6$, *$P<0.05$ compared with control.

**TABLE 2: EFFECT OF AQUEOUS EXTRACT OF STEM OF *BUTEA FRONDOSA* ON PEPTIC AND ALKALINE PHOSPHATASE ACTIVITIES FOLLOWING DIFFERENT DURATIONS OF TREATMENT IN RATS**

| Treatment | Dose (mg/kg) | Treatment duration (Days) | Peptic activity (IU) | Alkaline phosphatase (IU) |
|---|---|---|---|---|
| Control | -- | 14 | 70.05±0.98 | 43.58±0.87 |
| Famotidine | 3 | 14 | 43.51±0.70** | 29.27±0.96* |
| AESBF | 10 | 07 | 57.68±0.66 | 33.89±0.66 |
| AESBF | 100 | 14 | 52.36±0.63* | 30.48±0.60 |
| AESBF | 300 | 21 | 42.44±0.60* | 29.66±0.55* |
| AESBF | 500 | 28 | 38.07±0.84* | 27.28±0.70* |

Values seen are mean±SEM, $n$=6, aqueous extract of stem of *Butea frondosa*, *$P$<0.05 compared with control.

The biological design of the gastroduodenal lining is such that a harmonious balance is maintained between the offensive and defensive factors. The gastric mucosa is constantly exposed to exogenous and endogenous irritants, which impinge the integrity of the gastro duodenal lining. In contrast to the conventional opinion that only gastric acid was a culprit for ulceration, contemporary views revolve around the fact that pepsin also facilitates the disruption of the mucosal barrier. Mucosal erosions are facilitated by proteolytic enzymes such as pepsin, which is capable of hydrolyzing mucosal proteins[2]. Mutilation of mucosal integrity can be facilitated by ulcerogens. Release of ALP during injury would result in tissue necrosis[2]. ALP is an important marker enzyme for polymorph neutrophil infiltration, which is generated during injury.

Acetic acid used as an ulcerogenic agent in this study is capable of producing severe gastric hemorrhagic erosions. Acetic acid induced lesions resemble clinical ulcers in terms of location, chronicity and severity[9]. Exposure to acetic acid can produce a significant decrease in mucus and prostaglandins secretion. Acetic acid is also a histamine liberator. It has been reported that back diffusion of HCl and increased capillary permeability induced by acetic acid is mainly due to release of histamine[9]. Pyloric ligation, which is done following the administration of acetic acid, is a model, which responds to antiulcer drugs encompassing different mechanisms. In our study, treatment with famotidine, an $H_2$ receptor antagonist was found to significantly reduce peptic activity and the generation of ALP. The vulnerability of gastric mucosa to histamine liberators like acetic acid is minimized by famotidine.

AESBF significantly reduced peptic activity at all dose levels. ALP activity was also altered with a dose of 300 and 500 mg/kg of AESBF as a single schedule. Following treatment with 100 mg/kg AESBF for 21 and 28 days an elaborate decline in peptic activity and ALP was observed. We propose that mucosal integrity might be maintained by AESBF due to decreased activation of pepsinogen to pepsin. As *Butea frondosa* possesses flavonoids, they might be responsible for free radical scavenging thereby circumventing injury to macromolecules of the cell. Due to decreased injury, the generation of ALP may be minimized by AESBF thereby reducing the chances of re-exacerbation. In conclusion, AESBF is effective in preventing the formation of markers, which significantly reflect protection to the gastric mucosa.

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