SUMMARY

SUMMARY

Role of Nirmali (*Strychnos potatorum* Linn.) in drug induced experimental ulcers was described.

1. Experimental gastric ulcers were induced in **albino rats** by the drugs like

Salicylic acid	- 100 mg/kg, intraperitoneally	(8)
Aspirin	- 100 mg/kg, intraperitoneally	(8)
Paracetamol	- 100 mg/kg, intraperitoneally	(164)
Indomethacin	- 25 mg/kg, intraperitoneally	(9)
Hydrocortisone	– 50 mg/kg, intraperitoneally	(165)
Prednisolone	– 30 mg/kg, intraperitoneally	(165)
in **guinea pigs** by		
Phenylbutazone	– 100 mg/kg, orally	(137)
and in **mice** by		
Histamine	– 33 microgram/mouse, intraperitoneally	(166)

Drugs were given once daily for three consecutive days.

2. Glandular part of the stomach of all animals (100%) developed massive ulcers by the drugs. In most of the cases (80 –100%) ulcers were accompanied by haemorrhage. Adhesion and acute dilatation were seen.

3. **Powdered seed of Nirmali (*Strychnos potatorum* Linn.)** in a dose of 1g/kg/day, orally (exerted maximum activity as revealed from the pilot experiment) was given to the animals for three consecutive days along with the ulcerogenic drug.

4. **Nirmali (*Strychnos potatorum* Linn.) was found "antiulcerogenic"** in all the ulcer models studied since it reduced the rate of incidence and severity of ulcers (60 – 90%) induced by ulcerogenic drugs.

5. Gastric juice was collected from the animals and the rate of gastric secretion, gastric acidity and peptic activity were measured during ulceration as well as after treatment with Nirmali (*Strychnos potatorum* Linn.).

6. Rate of gastric secretion, gastric acidity and peptic activity were not significantly affected during ulceration and after treatment with Nirmali (*Strychnos potatorum* Linn.).

7. Dissolved gastric mucins of the animals were analysed during ulceration and effect of Nirmali (*Strychnos potatorum* Linn.) on it was studied.

8. Level of dissolved gastric mucin in terms of its constituent carbohydrate components viz. total hexoses, hexosamine, methyl pentose and sialic acid was found decreased significantly ($p < 0.025$ to $p < 0.001$) during ulceration. Nirmali (*Strychnos potatorum* Linn.) treatment, on the other hand, increased significantly ($p < 0.025$ to $p < 0.001$) the level of dissolved gastric mucin.

9. Gastric mucosal mucus was collected from the ulcerated stomach and the amount was estimated. Effect of Nirmali (*Strychnos potatorum* Linn.) on the said parameter was studied.

10. Level of gastric mucosal mucus of the animals was found decreased

significantly ($p < 0.001$) during ulceration . Nirmali (*Strychnos potatorum* Linn.) treatment could significantly ($p < 0.001$) increase the level of gastric mucosal mucus.

11. **Anti-ulcer effect of Nirmali (*Strychnos potatorum* Linn.) was, thus, not related with offensive factors like "acid-pepsin" but had relation with defensive parameters like "mucosubstances of gastric juice and gastric mucosa".**

12. Lipid peroxidation was studied in ulcerated stomach. Effect of Nirmali (*Strychnos potatorum* Linn.) on the said parameter was studied.

13. Level of lipid peroxides in stomach was found increased ($p < 0.001$) during ulceration. Nirmali (*Strychnos potatorum* Linn.) treatment could significantly ($p < 0.001$) decrease the level of lipid peroxides.

14. DNA content of the gastric mucosa was estimated in ulcerated stomach as well as in stomach after Nirmali (*Strychnos potatorum* Linn.) treatment.

15. DNA content of the gastric mucosa, decreased during ulceration, was found significantly increased ($p < 0.001$) by the treatment with Nirmali *(Strychnos potatorum* Linn.).

16. **Ulcerogenic effect of drugs was thus explained in terms of lipid peroxidation which was protected by Nirmali (*Strychnos potatorum* Linn.).**

In conclusion it can be suggested that,

Ulcerogenic drugs could increase gastric lipid peroxidation thereby generate reactive oxygen metabolites. This could damage gastric cells as observed by various workers (118 - 123). This was reflected by decreased amount of DNA in gastric mucosa which,

in turn, was responsible for decreased synthesis of gastric mucosubstances. In absence of proper protective layer of mucosubstances, ulcer developed in the stomach.

Nirmali (Strychnos potatorum Linn.), on the other hand, could inhibit gastric lipid peroxidation thereby inhibit generation of reactive oxygen metabolites. This could protect the gastric cell from damage. DNA of gastric mucosa was, thus, found increased with a concomitant increase in the level of gastric mucosubstances. These mucosubstances gave a proper protection in the stomach for which ulcers could not develop.

Anti-ulcer property of Nirmali (Strychnos potatorum Linn.) was thus explained by its anti-oxidative activity. ##