

## A proposed role for PGE<sub>2</sub> in the genesis of stress-induced gastric ulcers

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Prostaglandins are potent inhibitors of gastric secretion in rats (Robert, Nezamis & Phillips, 1968), dogs (Robert, Phillips & Nezamis, 1968) and humans and they are able to prevent ulcer formation in rats after pylorus ligation, administration of steroids or infusion of secretagogues (Robert, Nezamis & Phillips, 1968).

In our experiments the effect of PGE<sub>2</sub> administration has been evaluated on stress induced gastric ulcers in rats. PGE<sub>2</sub> (50-200-800 µg/kg s.c.) and PGF<sub>2α</sub> (100-400-1600 µg/kg s.c.) inhibit ulcer formation in

dose-dependent fashion, PGE<sub>2</sub> being 10 times more potent than PGF<sub>2α</sub>. Indomethacin, a drug known to inhibit the synthesis of prostaglandins (Vane, 1971), when administered at doses scarcely ulcerogenic at room temperature (1.25, 2.5, 50 mg/kg orally) increases the severity of gastric ulcers induced by stress, but this effect is completely antagonized by PGE<sub>2</sub> administration.

A possible role for prostaglandins on gastric homeostasis was discussed.

### References

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## The effect of mucosal damage on gastric glycoprotein synthesis in the rat

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Recently great interest has been shown in the role that gastric mucus plays in the protection of the

mucosa against acid-pepsin digestion (Waldron-Edward, 1970). As glycoproteins are important constituents of gastric mucus, the effects of ulcerogenic procedures on gastric mucosal glycoprotein synthesis were investigated by measurement of the rate of incorporation of a radioactively labelled sugar.

Female Wistar rats, Biorex strain, 140-150 g, were used. Phenylbutazone (200 mg/kg), suspended in 1 ml of 0.1% aqueous Tween 80, was given to rats as a single oral dose. Stress erosions

**Table 1** The effect of various experimental procedures on the rate of incorporation of *N*-acetyl-0[1-<sup>3</sup>H]-glucosamine into gastric mucosal glycoproteins

Treatment	No. of animals (n)	Percentage of rats with erosions	Rate of incorporation of [ <sup>3</sup> H]-GlcNAc (d/min/mg protein)	Percentage reduction in rate of incorporation
Control	17	0	5430 ± 720	—
Starved 24 h	6	0	2500 ± 610 ( <i>P</i> < 0.01)	54
Starved + cold 6 h	6	0	4050 ± 790 ( <i>P</i> > 0.2)	25
Restrained + cold 6 h	11	55	2940 ± 510 ( <i>P</i> < 0.02)	46
* Phenylbutazone (200 mg/kg orally)	6	33	3150 ± 560 ( <i>P</i> < 0.025)	42

\* Rats were killed 6 h after single administration.

Portions of fundal tissue of rats were incubated with 1.0 µCi [<sup>3</sup>H]-*N*-acetylglucosamine for 2.5 hours. The values shown are means for the numbers of rats (*n*) ± s.e. mean. Probability values (*P*) for the differences from controls (Student's *t* test) are also given.