HYPERGASTRINEMIA AND GASTRIC ACID SECRETION IN RATS INFECTED WITH *TAENIA TAENIAEFORMIS*

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Hypergastrinemia is known to develope in ruminants infected with abomasal nematodes belonging to the Trichostrongylidae (McLeay *et al.*, 1973). The cause of this serum gastrin elevation has been suggested to be gastric pathological changes caused by the nematode. Hypergastrinemia and gastric hyperplasia were also reported in rats heavily infected with larval *Taenia taeniaeformis*, a cestode parasite in the liver (Bullock and Curtis, 1930; Cook *et al.*, 1981).

In this study, changes in serum gastrin levels in rats experimentally infected with *T. taeniaeformis*, their relationship to the number of eggs inoculated and gastric acid secretion were examined.

Elevations of serum gastrin levels were first observed in rats inoculated with 5,000, 2,000, and 500 eggs at 28, 56 and 70 days post infection (PI). Thus, it was observed in rats that the greater the number of eggs inoculated, the earlier the hypergastrinemia could be observed.

At 11 weeks PI, notable pathological changes in rats inoculated with 2,000 eggs included increased stomach weight (control: 1.2g, infected: 5.6g), gastric mucosal hyperplasia, hypersecretion of mucus and elevated gastric luminal pH (control: pH 3.9, infected: pH 8.4). Light microscopic examination revealed that mucosal hyperplasia was restricted to the glandular mucosa. Parietal and chief cells were rare, and PAS-positive mucous cells were the major cell type in the hyperplastic stomach. Edema of the lamina propria, with fibroblastic proliferation, infiltration by eosinophils and an increased number of globule leukocytes were also observed.

No difference in gastric acid secretion at the basal acid output between control and infected groups (control: 1.7, infected: 1.9 μEq.H^+/15min.) was observed. However, the infected group failed to respond to stimulation with histamin, with the maximum acid output being 12.9 and 2.8 (μEq.H^+/15min.) in the control and infected groups, respectively.

The above results suggested that heavy infection with larval *T. taeniaeformis* can result in suppression of gastric acid secretion, gastric mucous cell hyperplasia, hypersecretion of mucus, and elevation of gastric luminal pH. All these events might lead to the resulting hypergastrinemia. Nevertheless, further investigations on the mechanism and the primary stimuli of these pathological changes are needed for better understanding of the pathophysiology of *T. taeniaeformis* infection.