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

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Parasitic infection results in various physiologic alterations in the host. Marked hypergastrinemia is reported in larval *Taenia taeniaeformis* infection of rats, accompanied by gastric hyperplasia and inhibition of acid secretory activity.

In this study, changes in serum gastrin levels were examined from 84 to 210 days post-infection (DPI) in F344/Jcl female rats, orally infected with 50, 500, 2,000 or 5,000 *T. taeniaeformis* eggs at 4 weeks of age. In heavily infected rats, hypergastrinemia occurred by 84 DPI and a high level of serum gastrin was maintained until 210 DPI. In contrast, rats with mild infection did not exhibit hypergastrinemia at 84 DPI and the serum gastrin level did not rise during the observation period. This suggests that the occurrence of hypergastrinemia depends on the number of parasites in the liver.

Rats of the same sex and strain were orally infected with 2,000 *T. taeniaeformis* eggs at 6 weeks of age to observe histopathologic changes, acid secretory activity, intragastic pH, and the serum gastrin level from 14 DPI to 84 DPI. However, larval development in these rats was unexpectedly suppressed. There was a considerable decrease in numbers of metacestodes, with a mean of 83 ± 50 larvae per rat at 84 DPI. Neither gastric hyperplasia nor hypergastrinemia was observed during the observation period. This suggested that the number and/or stage of development of the larvae in the liver might be important for the occurrence of hyperplasia and hypergastrinemia. Biochemical analysis of sera from these infected rats showed decreases in levels of albumin, cholinesterase, total cholesterol and free fatty acid, and increases of bilirubin and triglycerides. An increase in gastric mucus secretion was observed from 49 DPI, and elevation of intragastric pH was distinct at 84 DPI (infected: 6.23; control: 2.27). It was suggested that the rise in intragastric pH was independent of gastric hyperplasia. Acid secretory activity was then investigated. Although there was no significant difference in basal acid output levels between uninfected control rats (1.73 mEq.H^+/15 min) and infected rats (1.74 mEq.H^+/15 min), there was a tendency for the response to histamine (10 mg/kg b.w.) stimulation to be inhibited. The mean acid output in infected rats following histamine stimulation was 11.31 mEq.H^+/15 min in comparison with 19.58 mEq.H^+/15 min in uninfected

control rats. This showed that a change in acid secretory activity might occur without gastric hyperplasia characterized by an increase of mucus cells with a decrease of parietal and chief cells.

Immunohistochemical study of tissue sections taken from stomachs with and without hyperplasia revealed the following pathologic changes in hyperplastic stomachs : i) a considerable widening and increase cell division of the glandula gastrica, ii) no distinct increase in G cell numbers in the pyloric antrum, and iii) a decrease in enterochromaffin-like cell numbers of the glandula gastrica, which may be important in gastric acid secretion.