



HOKKAIDO UNIVERSITY

Title	Taenia taeniaeformis : Mechanisms of hepatic larvae-induced gastric hyperplasia and hypergastrinemia in rats
Author(s)	Abella Jose Alexander Cabiling
Citation	Japanese Journal of Veterinary Research, 45(3), 178-179
Issue Date	1997-11-28
Doc URL	https://hdl.handle.net/2115/2608
Type	departmental bulletin paper
File Information	KJ00002398568.pdf



INFORMATION

Hokkaido University conferred the degree of Doctor of Veterinary Medical Science (equivalent to Ph. D.) on June 30, 1997 to 6 Recipients.

The titles of their theses and other information are as follows :

Taenia taeniaeformis : Mechanisms of hepatic larvae-induced gastric hyperplasia and hypergastrinemia in rats

Abella Jose Alexander Cabiling

Laboratory of Parasitology,
Department of Disease Control,
Graduate School of Veterinary Medicine,
Hokkaido University, Sapporo 060, Japan

Various pathological changes have been observed in parasitic infections and normally these are restricted to the site of infection or adjacent tissues or organs. Interestingly, rats infected with larval *Taenia taeniaeformis*, exhibit hyperplastic change of the stomach and hypergastrinemia, even though the site of infection, which is the liver, is remotely located from the stomach. The underlying pathogenetic mechanism(s) how these changes are induced is unknown. This study was therefore undertaken to clarify the possible mechanism(s) of induction of gastric hyperplasia and hypergastrinemia of rats infected with *Taenia taeniaeformis*.

Firstly, to know the sequence of occurrence of the principal pathological changes, namely, hypergastrinemia, gastric hyperplasia and intragastric alkalinity, associated with this condition, rats were necropsied every 2 weeks after infection with *Taenia taeniaeformis* until 112 days postinfection (DPI) and the gross and microscopic change of the stomachs, intragastric pH and levels of serum gastrin were examined. Changes of these parameters were not recognized until 42 DPI, however, hyperplasia of the gastric glandular mucosa, increase of intragastric

pH and hypergastrinemia were simultaneously observed at 56 DPI.

The histopathological changes of the stomachs include : increase in the number of undifferentiated cells and mucus secreting cells, hypersecretion of mucus and decrease in the number of parietal cells and chief cells. These observations indicate that there is preferential differentiation of undifferentiated cells to mucus secreting cells, while differentiation to parietal cells and chief cells was suppressed. These changes, recognized at 56 DPI were observed in infected rats examined thereafter.

When examined for basal and maximal acid secretion after histamine stimulation ($\mu\text{Eq.H}^+$ /15 min.), no difference was observed in basal acid output between control (1.7) and infected (1.9) rats, however infected rats did not respond to histamine stimulation (control 12.9; infected 2.8).

To clarify the mechanism of induction of hypergastrinemia, the number of gastrin secreting antral G cells were examined from 28 to 98 DPI. Notable changes in numbers of G cells per unit area of gastric antrum were not recognized between infected and control rats during the

period of study. However, considering the total area of the antrum in hyperplastic stomachs, the total number of G cells could have increased after 56 DPI.

It is well known that in rats, induction of hypergastrinemia results to an increase in the number of enterochromaffin-like (ECL) cells, particularly with the administration of drugs which inhibit gastric acid secretion. In this study, change in the number of ECL cells was assessed after staining with Grimelius and chromogranin A+B. It was evident that the number of ECL cells considerably decreased after 56 DPI. The mechanism behind this reduction is not clear, however, the observed change is considered a characteristic of *Taenia taeniaeformis* infection.

Some cytokines, such as IL-1 and TNF- α has been shown to cause proliferation of gastric mucus cells. However, in this study, hypergastrinemia and gastric hyperplasia were observed in nude rats infected with *Taenia taeniaeformis* and the histopathological changes exhibited by these rats is similar to those in infected euthymic rats. Therefore, the influence of T cells or T cell derived factors are not required for the generation of hyperplastic change in the stomach. It is suggested that the changes in the stomach is not

only dependent on the number of larvae but also on its degree of development (growth). *Taenia taeniaeformis* larvae are well developed in nude rats and gastric hyperplasia and hypergastrinemia were observed in nude rats infected with fewer numbers of eggs compared to euthymic rats. This could be due to the absence of antibody production against certain larval derivatives in nude rats which may contribute to the pathological change, however, gastric hyperplasia and hypergastrinemia were not observed in infected euthymic rats in which the larvae were comparatively not well developed. Moreover, the histopathological changes of the spleen and associated antibody changes at 56 DPI indicate immunosuppression.

These observations led us to hypothesize that, i) certain factors are released from the *Taenia taeniaeformis* larvae developing in the liver, directly or indirectly promoting the differentiation of mucus secreting cells from undifferentiated cells but suppressing the ECL and parietal cell differentiation. This suppresses gastric acid production, raising the pH of the stomach and thus inducing hypergastrinemia. Moreover, partial immunosuppression could facilitate the development of hypergastrinemia and hyperplasia in rats.

Original papers of this thesis appeared in "Parasitology International", Vol. 46, 97–104 (1997) and "The Journal of Veterinary Medical Science", Vol. 59, 1039–1043 (1997).

Oocyte collection and production of IVF embryos using
ultrasound-guided transvaginal follicular aspiration in cattle

Masato Konishi

*Embryo Transfer Laboratory,
Central Research Institute For Feed and Livestock,
ZEN-NOH, Tsukuba, Ibaraki 300-33, Japan*

Original papers of this thesis appeared in "Theriogenology", vol. 45, 573–581 (1996), "Theriogenology", vol. 46, 33–43 (1996), and "J. Vet. Med. Sci.", vol. 58, 893–896 (1996).