Effect of *Helicobacter* sp. infection on the number of antral gastric endocrine cells in swine

R. Sapierzyński, M. Fabisiak, M. Kizerwetter-Świda¹, A. Cywińska¹

Department of Clinical Sciences
¹ Department of Preclinical Sciences, Faculty of Veterinary Medicine, Warsaw Agricultural University (SGGW), Nowoursynowska 159c, 02-766 Warsaw, Poland

Abstract

The aim of the present study was to evaluate the effect of *Candidatus Helicobacter suis* (CHS) and other *Helicobacter* sp. different from *Candidatus Helicobacter suis* (non-*Candidatus Helicobacter suis*, non-CHS) infection on the number of endocrine G and D cells and G/D cells ratio in antral gastric mucosa in swine. Twenty nine stomachs were obtained from clinical healthy pigs about 6 months old and weighing approximately 100-120 kg after slaughter at abattoir located in central Poland. From each stomach samples of the antral gastric mucosa were taken for histopathology, and PCR examination for presence of *Helicobacter* genus and *Candidatus Helicobacter suis*. Samples for histopathology and immunohistochemistry were fixed in 10% buffered formalin. To reveal the expression of gastrin- and somatostatin-producing cells specific antibodies were used. Selected endocrine cells were counted in the midzone of pyloric glands, the results were expressed as a mean of the number of immunoreactive cells in one microscopic field, and as the ratio of gastrin to somatostatin cells (G/D). It can be concluded that some species of swine *Helicobacter* can alter the number of endocrine cells in gastric antral mucosa. Some of these alterations, for example increase the number of G cells, decrease of the D cells and especially increase of ratio G to D cells can be responsible for development of gastroesophageal ulcers in swine.

Key words: swine, *Helicobacter* sp., *Candidatus Helicobacter suis*, stomach, endocrine cells.

Introduction

The relationship between *Helicobacter* sp. infection observed in the stomach of swine and gastric disease is unclear. Infection with these organisms is considered as a possible factor connected to the etiopathogenesis of gastric ulcer of the pars oesophagea, dysplastic lesions and gastritis (Barbosa et al. 1995, Park et al. 2000, Park et al. 2003, Krakowka et al. 2005b). In human, it seems that *Helicobacter pylori* can cause gastric and duodenal ulcers by increase in gastrin secretion by antral G cells. Infection stimulates increased release of gastrin, which in turn induces acid secretion that leads to excessive duodenal acid load and cause ulceration.

Like duodenal mucosa, the mucosa of the *pars oesophagea* is susceptible to destructive effect of gastric acid. Normally, there is a balance between action of acid and defensive mechanisms of mucosa, but when secretion is increasing these mechanisms can be insufficient. Secretion in gastrointestinal tract is under control of hormones, which are produced in reaction