FACULTY OF VETERINARY MEDICINE
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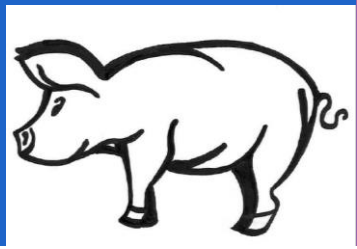
INVITATION

Public defense of the doctoral thesis of Chloë De Witte

Role of *Helicobacter suis* and *Fusobacterium gastrosuis* in the pathogenesis of gastric ulcer disease in pigs

28/02/2019



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Curriculum Vitae

Chloë De Witte was born on December 11, 1990 in Izegem, Belgium. In 2014, she obtained her Master's degree in Veterinary Medicine, option Research, with summa cum laude. She also received the price for the best master thesis, option Research, and was awarded the price of the Faculty of Veterinary Medicine for the best graduation results.

In October 2014, Chloë started her PhD research at the Department of Pathology, Bacteriology and Avian Diseases, Faculty of Veterinary Medicine, financed by the Special Research Fund of Ghent University. She also obtained the certificate of the Doctoral Training Programme of Life Sciences and Medicine.

Chloë De Witte is (co-)author of several papers in international peer-reviewed journals. She also gave presentations at international conferences, where she was awarded for the best poster and oral presentation. She actively participated in the practical sessions for the course 'Bacteriology and mycology', and she presented lectures for the course 'Bacterial and fungal infectious diseases and zoonoses'.

When and where?

The public defense will take place on Thursday the 28th of February, at 5 pm

Kliniekauditorium B – Entrance 12
Faculty of Veterinary Medicine, UGent
Salisburylaan 133, Merelbeke

Afterwards, there will be a reception in the Museum of Morfologie ☺

Register

If you wish to attend the reception, you are kindly asked to confirm your presence before 14 February by e-mail to chloe.dewitte@ugent.be

Summary

Gastric ulcera are highly prevalent in swine production and may lead to significant economic losses as well as animal welfare issues. Nevertheless, its pathogenesis is largely unknown. In order to develop effective control measures, it is important to identify the risk factors involved in gastric lesion development.

The **general aim** of this thesis was to investigate the role of pathogens in the development of porcine gastric ulceration, which may ultimately facilitate the development of effective control measures.

Several studies have attributed a role to *Helicobacter suis* in the development of ulceration of the upper, nonglandular *Pars oesophagea*, although *H. suis* does not colonize this stomach region. It is not completely clear how *H. suis* influences lesion development, but our studies indicate that alterations in gastric acid secretion may be involved.

During the acute phase of a *H. suis* infection, the gastric acid secretion is unaffected and so no irritation occurs of the *Pars oesophagea*. Later on, a decreased gastric acid secretion in the glandular part of the stomach affects the composition of the *Pars oesophageal* microbiota, which may affect development of lesions in this non-glandular part of the stomach. Indeed, higher numbers of *Fusobacterium gastrosuis* were detected in the *Pars oesophagea* of *H. suis*-infected 6-8 months old pigs than in non-infected pigs of the same age group. *In vitro* experiments showed that viable *F. gastrosuis* bacteria as well as bacterial lysate induced massive epithelial cell death and genome analysis showed presence of several, potential virulence genes. Finally, increased production of gastric acid during the chronic phase of infection might further aggravate severity of lesions in this stomach region, which is not protected by mucus.

Our results lead to the hypothesis that *H. suis* is involved in gastric pathology through its effects on gastric acid secretion and on the gastric microbiota composition. We hypothesize that, in a gastric environment altered by *H. suis*, colonization and invasion of the *Pars oesophagea* and production of epithelial cell death inducing metabolites by the novel pathogen *F. gastrosuis* cause lesions of this stomach region.

Experimental studies in pigs infected with *H. suis* and *F. gastrosuis* would be necessary to confirm these hypotheses. Inhibition of colonization by these bacteria and/or their induced pathologies can be considered as potential control measures against porcine gastric ulceration.

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