

INVITATION PUBLIC DEFENSE

Strain-specific enterotoxin secretion and impact of gut epithelial cells on porcine ETEC toxin secretion

Haxiu Wang

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PROMOTORS

Prof. dr. Bert Devriendt
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Curriculum Vitae

Haixiu Wang was born on in Shandong, China. She obtained her B.S. in Biotechnology, in 2012 from the Shandong Agricultural University (SDAU) and M.S. in Preventive Veterinary Medicine from the Graduate School of Chinese Academy of Agricultural Sciences (GSCAAS) in 2015. After graduating, she is currently a PhD candidate in Prof. Dr. Bert Devriendt's research group at Ghent University. Her research focused on the strain-specific regulation of enterotoxins varying secretion capacity between porcine ETEC strains in vitro. She has authored and co-authored several publications in international peer-reviewed journals.

Where?

The defense will take place on Monday, 19, 2022 at 17.00h

Auditorium 1 (Hoogbouw)

Faculty of Veterinary Medicine
Ghent University, Campus Merelbeke
Salisburylaan 133, Merelbeke

How to attend?

If you would like to attend, please register before 14 December 2022, by email to haixiu.wang@ugent.be

Members of the Jury

Prof. dr. Filip van Immerseel
Chairman of the Jury
Faculty of Veterinary Medicine, UGent

Prof. dr. Patrick Butaye
Faculty of Veterinary Medicine, UGent

Prof. dr. Marc Heyndrickx
Faculty of Veterinary Medicine, UGent

Prof. dr. Damien Thiry
Faculty of Veterinary Medicine, ULiège

Prof. dr. Stéphanie Blanquet-Diot
Microbiologie Environnement Digestif et Santé (MEDIS), UCA

Summary

Porcine ETEC is an important cause of bacterial diarrheal illness and is regarded as a global health threat for farm animals. Neonatal diarrhea (ND) and post weaning diarrhea (PWD) caused by ETEC result in severe economic losses for the farming industry worldwide due to increased morbidity and mortality and reduced growth rates. Neonatal piglets can be protected by the transfer of maternal antibodies upon vaccination of the sow. However, upon weaning piglets become highly susceptible to ETEC as the passive immunity wanes at weaning. To protect newly weaned piglets against ETEC infections, an oral live bivalent vaccine, Coliprotec® F4/F18 (Elanco GmbH), has been marketed in the EU and other countries since 2017. However, it could not provide complete protection to piglets. Furthermore, the live vaccine has some limitations and associated risks. Therefore, further research into the development of vaccines or alternative strategies is needed. This requires a deeper understanding of the pathogenesis of ETEC and its interaction with its hosts at the molecular level.

Given the importance of the heat labile (LT) and the heat stable enterotoxins (pSTa and STb) in ETEC pathogenesis, in this thesis, we investigated the intrinsic variation in enterotoxin secretion levels between strains and assessed the role of the type I and II secretion system herein. The first study of the thesis describes the variation in production and secretion of LT and STs between porcine ETEC strains. Interestingly, in some strains, the secretion capacity did not correlate with their ability to produce enterotoxins. A further analysis of these strains revealed that varying levels of the type II secretion system components GspD and YghG, were involved in regulating the secretion of LT. Using isogenic overexpression strains, the correlation between YghG expression and LT secretion levels was confirmed. Likewise, differences in ST secretion levels between ETEC strains could in part be attributed to TolC, a critical type I secretion system component. In addition, to address the influence of the host on enterotoxin secretion by ETEC, we further investigated the role of host-derived factors secreted by the gut epithelium in the controlling secretion of LT and STs by different ETEC strains. We found that epithelial factors secreted by gut epithelial cells affected the transcriptional landscape in ETEC, resulting in variation in enterotoxin secretion between ETEC strains. Strains with high LT secretion levels decreased their LT secretion in response to molecules secreted by the gut epithelium, while low LT secretor strains increased their LT secretion. In contrast, molecules secreted by the gut epithelium further induced pSTa and STb secretion in strains with a high ST secretion capacity, but downregulated ST secretion in low secretor strains. In addition, molecules secreted by the gut small intestinal epithelium affected the transcript levels of transcription factors and secretion system components involved in LT and ST transcription and secretion, respectively.