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ROLE OF SIALIC ACID IN *BRACHYSPIRA HYODYSENTERIAE* ADHESION TO PORCINE COLONIC MUCUS

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Introduction

Infection with *Brachyspira hyodysenteriae* results in mucoid hemorrhagic diarrhea. This pathogen is associated with the colonic mucus layer, mainly composed of mucins. Infection increases mucin secretion in the colon, increases *B. hyodysenteriae* binding sites on mucins, and regulates mucin glycosylation. Here, we analyzed potential mucin epitopes for *B. hyodysenteriae* adhesion in the colon, as well the effect of mucins on bacterial growth.

Materials & Methods

Bacterial adhesion assays were performed to Sialyl-Lewis x, Lewis b, Lacto-N-tetraose, Core-2 and LacdiNAc glycoconjugates providing a range of epitopes for binding. Associations between mucin glycan data and *B. hyodysenteriae* binding to porcine colon mucins data were determined with Pearson correlation coefficient. The role of sialic acid and galactose in *B. hyodysenteriae* adhesion were analyzed after sialidase A and β-galactosidase treatment of the insoluble mucins. Additionally, the effect of porcine colon mucins on *B. hyodysenteriae* growth was determined in defined media without glucose and growth was measured every 2 h for 24 h.

Results

A high statistical correlation was identified between *B. hyodysenteriae* adhesion to insoluble porcine mucins and the presence of sialylated structures. Furthermore, *B. hyodysenteriae* binding to insoluble mucins decreased after sialidase A treatment compared to the non-treated mucin control, while adhesion to β-galactosidase treated mucins remained unchanged. Binding of *B. hyodysenteriae* to synthetic glycoconjugates was not statistically different from background. *B. hyodysenteriae* growth increased in the presence of mucins from two out of five infected pigs,

suggesting utilization of mucins as a carbon source for growth.

Discussion & Conclusion

The results highlight a role of sialic acid as an adhesion epitope for *B. hyodysenteriae* interaction with colonic mucins. Furthermore, the mucin response and glycosylation changes exerted in the colon during *B. hyodysenteriae* infection results in a potentially favorable environment for pathogen growth in the intestinal mucus layer.