

### **Piglets infected with ETEC F4 and F18: effect of *MUC4* and *FUT1* genotypes.**

Massacci F.R.<sup>1,2,3</sup>, Tofani S.<sup>3</sup>, Tentellini M.<sup>3</sup>, Orsini S.<sup>3</sup>, Lovito C.<sup>3</sup>, Forte C.<sup>3</sup>, Luise D.<sup>1</sup>, Bevilacqua C.<sup>2</sup>, Marchi L.<sup>3</sup>, Bertocchi M.<sup>1</sup>, Rogel-Gaillard C.<sup>2</sup>, Pezzotti G.<sup>3</sup>, Estellé J.<sup>2</sup>, Trevisi P.<sup>1</sup>, Magistrali C.F.<sup>3</sup>

<sup>1</sup> Department of Agricultural and Food Science, University of Bologna, Bologna, Italy.

<sup>2</sup> GABI, INRA, AgroParisTech, Université Paris-Saclay, Jouy-en-Josas, France.

<sup>3</sup> Istituto Zooprofilattico Sperimentale dell'Umbria e delle Marche 'Togo Rosati', Perugia, Italy.

Enterotoxigenic *Escherichia coli* (ETEC) is the etiological agent of the post-weaning diarrhea (PWD) in piglets. The *MUC4* and the *FUT1* genes have been associated with the susceptibility to ETEC F4 and F18, respectively. The aim of this study was to investigate the effects of the genotype of *MUC4* and *FUT1* in piglets naturally infected with ETEC F4 and F18. A total of 71 piglets was divided into 3 groups based on two antimicrobial administration routes: A) parenteral antibiotic, B) oral antibiotic and C) control group without antibiotic. Animals arrived in the facility on weaning day (T0). For groups A and B, at T0 amoxicillin was administered during 5 days either parentally or orally. Animals were evaluated at the end of the amoxicillin administration (T1) and 7 days after (T2). At each time point, faecal scores and body weight were recorded, and presence of ETEC F4 and F18 in faecal samples was assessed by PCR. Results revealed that 50/71 piglets were naturally infected by ETEC F4 and 21/71 by ETEC F18 at T0; 7 piglets were positive for both the pathogens and 7 resulted negative. Only F18 was detected at T1. Both ETEC F4 and F18 strains were resistant to amoxicillin. At T0, Fisher tests showed that *MUC4* genotype was significantly associated with the presence of ETEC F4 and the faecal scores ( $p < 0.05$ ). Intriguingly, the *MUC4* resistant genotype was associated with ETEC F4 absence but also with a higher diarrhoea score. At T1, *FUT1* was associated with the presence of ETEC F18 ( $p < 0.05$ ) but not with the diarrhoea scores. Antibiotic administration was significantly associated with the presence/absence of F18 and the diarrhoea score at T1 and T2 ( $p < 0.05$ ). Our results confirm that *MUC4* and *FUT1* genotypes are associated with the susceptibility to ETEC F4 and F18 infection. Production of the the gut microbiota data is ongoing and the results will be correlated with the piglets' genotypes. Next step will thus be to study how the gut microbiota evolves in relation to ETEC infection, *MUC4* and *FUT1* genotypes and antibiotic administration.