Efficient application of high energy diets in combination with an oral live non-pathogenic Escherichia coli vaccination against post-weaning diarrhea

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Background & Objectives: Post-weaning *Escherichia coli* diarrhea (PWD), also called post-weaning enteric colibacillosis, in pigs remains a major cause of economic losses for the pig industry, due to either piglet death, or poor weight gain in surviving piglets [1,2]. PWD typically causes mild to severe watery diarrhea between 5 and 10 days after weaning and is primarily caused by enterotoxigenic *Escherichia coli* (ETEC). The most common adhesins found on ETEC from PWD in pigs are associated with fimbriae F4 (previously called K88) and F18, while the predominant enterotoxins are heat-labile toxin (LT), heat-stable toxin a (STa), and heat-stable toxin b (STb) [3,4,5,6]. Therapy to control PWD typically consists of antibiotic treatment, addition of therapeutic doses of zinc oxide (ZnO; >2500 ppm, 14 days) and changes in feed composition (safe feed) of the post-weaning diets. The objective of the present study was to compare vaccination with an oral live non-pathogenic *E. coli* F4 vaccine (Coliprotec® F4; PrevTec Microbia) in piglets fed a high energy diet with/without a safety concept (Nuscience) with two standard therapeutic approaches, namely ZnO (2500 ppm) and a safe feed formulation with addition of acid. The active vaccine strain in Coliprotec® F4 is oe of the components of the bivalent Coliprotec® F4F18 (PrevTec Microbia) vaccine.

Materials and methods: In a 600-sow farm with PWD caused by F4-ETEC, piglets (n=128 per treatment group) were vaccinated at 18 days of age with Coliprotec[®] F4. At weaning, piglets were randomly distributed into 5 groups with different treatments (Table 1). During the 7-week post-weaning period several technical production parameters (weight, average daily weight gain, feed intake, feed conversion rate, antibiotic treatment, mortality) were recorded. Statistical differences between groups were calculated using JMP[®] program.

Results: A summary of the obtained results is given in Table 1.

Table 1. Summary of trial outline and obtained performance results. Statistically different numbers are given with different superscripts.

Group	Α	В	С	D	E
Vaccination			+++	+++	+++
Adapted diet	Normal formula	Normal formula + acid	Safe start / high energy grow	High energy formula	High energy formula + acid
ZnO (2500 ppm, 14 days)	+++				
Weight at weaning (kg)	6.21 a	6.21 a	6.20 a	6.17 a	6.20 a
Weight at 21 dpw * (kg)	9.39 a	8.89 b	9.13 °	9.19 °	9.31 °
Weight at 50 dpw (kg)	22.74 a	21.84 b	23.95 °	24.37 c	24.44 c
ADWG° (0-21 dpw) (g/d)	151 a	124 °	140 °	144 ^c	148 ^c
ADWG (0-50 dpw) (g/d)	331 a	313 a	355 b	364 b	365 b
FCR [®] (feed/growth)	1.54 a	1.57 a	1.46 b	1.41 ^b	1.43 ^b
Mortality (%)	1.6 a	1.6 a	1.6 a	0.0 b	0.8 c
% Piglets with diarrhea (0-21 d)	1.82 a	5.65 b	1.53 a	3.68 ab	2.53 ab
AUC ** clinical fecal score (0-21 d)	58.2 a	115.6 °	40.4 b	66.2 a	61.6 a
Treatment incidence †	1.12 a	2.20 b	0.68 c	0.74 ^c	0.62 °

^{*} dpw - days post-weaning; ° ADWG - average daily weight gain; ® FCR - feed conversion rate; "AUC - Area under the curve of all fecal scores collected from 8 pens and 5 droppings/pen per day over the first 21 days of the study; † treatment incidence - number of treated piglets per 100 piglets in trial

Discussion & Conclusions: This comparative study clearly shows that vaccination against PWD with Coliprotec® F4 has several advantages on technical performance parameters. The use of a high energy diet in combination with Coliprotec® F4 combined a reduction of weight loss with lower antibiotic use. The vaccinated groups performed as compared to ZnO (group A) and safe feed formulation (group B). In conclusion, control of PWD through oral vaccination is a good option in order to protect piglets from the negative clinical effects of F4-ETEC infection in the post-weaning period.

References: [1] van Beers-Schreurs et al., 1992. Veterinary Quarterly 14, 29-34. [2] Fairbrother et al., 2005. Animal Health Research Reviews 6, 17-39. [3] Kwon et al., 2002. The Veterinary Record 150, 35-37. [4] Frydendahl, 2002. Veterinary Microbiology 85, 169-182. [5] Chen et al., 2004. Veterinary Microbiology 103, 13-20. [6] Vu Khac et al., 2006. BMC Veterinary Research 2, 10.