Manipulating the Environment in the Porcine Large Intestine Using Fermentable Carbohydrates to Control Swine Dysentery

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Swine dysentery (SD) is a contagious mucohaemorrhagic diarrhoeal disease with severe impacts on production efficiency in grower/finisher pigs. The causative agent of SD is the intestinal spirochaete *Brachyspira hyodysenteriae* that induces inflammation and necrosis of the caecum and colon (Hampson *et al.*, 2006). Recently, Thomsen *et al.* (2007) found that an organic diet containing sweet lupins and dried chicory root completely prevented SD following experimental challenge with *B. hyodysenteriae*. However, based on the study by Thomsen *et al.* (2007) it wasn't possible to determine whether the dietary protection against SD was due to the galactans supplied by the sweet lupins or inulin from the dried chicory roots or if both carbohydrate sources are needed. It was hypothesised that diets (barley and triticale based) containing galactans (as lupins) and fructans (as inulin) could prevent the occurrence of swine dysentery (SD) after experimental infection with *B. hyodysenteriae*.

A 2x2 factorial experiment was undertaken with the main effects being protein source (185g/kg canola meal (low in galactans) versus 220g/kg lupins (high in galactans)) and inulin supplementation (+/- 80g/kg inclusion). Forty Large White x Landrace pigs, 10 pigs per diet, weighing 21±2.8 kg were allowed to adapt to the diets for two weeks and then each pig was challenged orally four times on consecutive days with 80 mL of broth containing ~108 viable cells (*B.hyodysenteriaie*). Pigs were euthanised when they showed clinical signs of SD or at the end of the experiment six weeks post-infection.

Table 1. *Number of positive pigs, relative risk (RR)[1] of a pig showing clinical signs of swine dysentery (SD) and average number of days until pigs developed clinical SD or were slaughtered. Pigs were euthanised when they developed clinical SD or at the end of the experiment 42 d post-infection.*

Inulin (g/kg)...	0		80				
Protein source, Lupin (g/kg)...	0	220	0	220	SEM	Inulin[2]	Lupin[3]
Pigs challenged	10	10	10	10		Inulin: 1	Lupin: 1
Pigs with clinical SD	7	3	0	3		No inulin: 8.3	Canola meal: 1.4
RR of clinical SD	12.3	1	0	1		(1.7 – 58.0)	(0.3 – 7.3)
No. of days to slaughter	18.3[a]	34.5[b]	37.1[b]	36.7[b]	1.50	P=0.017	P=0.687

[1]The relative risk for the medical outcome in the group of interest compared with the reference group. [2,3]Relative risk of pig showing clinical SD when fed (1) diets with or without inulin, or (2) diets containing lupin versus canola meal. Relative risk and 95% confidence intervals are given. [ab]Significant interaction between Inulin and Lupin. Means in the same row with different superscripts differ significantly ($P < 0.05$).

Pigs fed diets without inulin had a 8.3 times higher risk of developing clinical SD (Table 1) and were 16 times more likely (P=0.004) to have colon contents that were culture positive for *B hyodysenteriae*, compared with the pigs fed a diet with 80g/kg inulin. Diets containing lupins didn't significantly protect the pigs in this experiment from developing clinical SD, but lupin and/or inulin inclusion in the diets delayed the onset of disease compared with the diet based mainly on canola meal.

This experiment demonstrates that diets supplemented with highly fermentable carbohydrates from inulin and sweet lupins might protects pigs against developing SD by possibly modifying the microbiota in the gastrointestinal tract. Pigs fed inulin had reduced risk of developing SD and the onset of disease was delayed in pigs fed lupin.

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