



## INCREASED INTESTINAL VISCOSITY DEPRESSES CARCASS GROWTH AND ENCOURAGES INTESTINAL PROLIFERATION OF *ESCHERICHIA COLI* IN WEANER PIGS

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### Introduction

Although it is recognised that high levels of dietary insoluble non-starch polysaccharides (NSP) depress growth in pigs (1), trials investigating the effect of dietary soluble NSPs (sNSP) on performance have produced inconsistent results, depending on the type of grain fed, age of pig and other interacting factors. Diets with high levels of sNSP depress growth and have anti-nutritive properties when fed to poultry, and part of this effect is attributed to the ability of sNSP to increase the viscosity of intestinal digesta (2). A previous study into the effects of guar gum, a viscous water-soluble NSP, on the performance and health of weaner pigs showed a detrimental effect on growth and an exacerbation of experimental post-weaning colibacillosis caused by enterotoxigenic *Escherichia coli* when this ingredient was fed (3).

The aim of this study was to further investigate the potential detrimental effects of increased intestinal viscosity in weaner pigs by feeding them an experimental diet supplemented with carboxymethylcellulose (CMC), a water-soluble preparation resistant to fermentation. The effect of increased viscosity on gastrointestinal development, growth performance and incidence of diarrhoea caused by haemolytic *E. coli* was monitored.

### Materials and Methods

Twenty-four pigs were weaned abruptly at 21 days of age and allocated according to liveweight into one of three low-fibre dietary treatments: 1. Diet R – a diet comprised mainly of cooked white rice, balanced for weaner nutritional requirements with an animal protein supplement (bloodmeal, meat and bonemeal, fishmeal); 2. Diet RLV, made of cooked white rice plus an animal protein supplement with the addition of low viscosity CMC (50-200 mPa•s when 4% solution at 25°C) comprising 4% of the total weight of the air-dry diet; or 3. Diet RHV, cooked white rice plus an animal protein supplement with the addition of a high viscosity CMC (400-800 mPa•s when 2% solution at 25°C) added at 4% of the total weight of the air-dry diet. Minor adjustments were made in amounts of ingredients in diets RLV and RHV to ensure energy and protein content were as close as possible to diet R.

Pigs were housed in pairs, and fed *ad libitum* for 13 days after weaning. Daily individual liveweight and voluntary food intake records were kept, and faecal swabs were cultured for presence of endogenous haemolytic *E. coli* on days 7, 8 and 9 post-weaning. One pig was excluded as it refused to eat for the duration of the trial. At the end of the period, the pigs were killed 1.5 hours after feeding, and intestinal measurements taken. Viscosity of the supernatant of digesta samples was recorded at 12 rpm on a cone-plate viscometer at 25°C.

### Results

**Table 1.** The effect of diet on intestinal viscosity, growth rates and intestinal weights.

	R n=8	RLV n=8	RHV n=7	sem	P-value
Duodenal viscosity (mPa•s)	1.42 <sup>a</sup>	6.14 <sup>b</sup>	8.93 <sup>b</sup>	2.63	0.0007
Ileal viscosity (mPa•s)	1.411 <sup>a</sup>	6.03 <sup>b</sup>	7.91 <sup>b</sup>	2.20	0.0006
Caecal viscosity (mPa•s)	1.67 <sup>a</sup>	4.09 <sup>a</sup>	6.18 <sup>b</sup>	2.43	0.0277
Full small plus large intestine (g)	647 <sup>a</sup>	882 <sup>b</sup>	883 <sup>b</sup>	90	0.0005
Liveweight gain (g/day)	291 <sup>a</sup>	393 <sup>b</sup>	325 <sup>ab</sup>	63	0.046
Carcass daily gain <sup>c</sup> (g/day)	105 <sup>a</sup>	149 <sup>a</sup>	29 <sup>b</sup>	60	0.017

<sup>a,b</sup> Mean values with different superscripts differ significantly <sup>c</sup>carcass gain = liveweight minus weight of full gut.

Dietary CMC increased the luminal viscosity in a viscosity-dependent manner along the full length of the small intestine and in the caecum.

Higher viscosity was consistent with heavier weight of the full gut which resulted in greater liveweight gain values in pigs fed RLV and RHV. Once the intestinal weights were subtracted from liveweight, the relative carcass gain of pigs fed RLV and RHV decreased.

**Table 2.** Number of pigs in each group with diarrhoea.

Diet	Day 7 <sup>c</sup>	Day 8	Day 9	Day 10
R (n=8)	0/8 <sup>a</sup>	1/8 <sup>a</sup>	0/8 <sup>a</sup>	0/8 <sup>a</sup>
RLV (n=8)	5/8 <sup>b</sup>	3/8 <sup>b</sup>	4/8 <sup>b</sup>	4/8 <sup>b</sup>
RHV (n=7)	7/7 <sup>b</sup>	7/7 <sup>b</sup>	7/7 <sup>b</sup>	5/7 <sup>b</sup>
P-value	<0.05	<0.05	<0.05	<0.05

<sup>a,b</sup>Mean values with different superscripts differ significantly <sup>c</sup> no. days after weaning

Pigs fed diet R remained healthy and did not display any diarrhoea for the duration of the trial. Pigs fed the viscous diets began to display loose faeces around 4-5 days post-weaning. By the 7<sup>th</sup> day after weaning, five out of eight of the pigs fed RLV and all of the pigs fed RHV had diarrhoea (Table 2). Both groups RLV and RHV were shedding significantly more haemolytic *E. coli* serotype O141;K88 (P=0.011, Table 3) daily than pigs fed diet R.

**Table 3.** Mean % of bacteria, cultured from faeces, that were haemolytic *E. coli*.

Diet	Day 7	Day 8	Day 9	Day 10
R (n=8)	13.6	14.3	25.0	0
RLV (n=8)	61.4	62.5	58.8	15.0
RHV (n=7)	84.7	61.4	70.0	29.3

### Discussion

Addition of CMC to the diet increased viscosity of the intestinal contents and induced significant changes in intestinal size and luminal environment. High viscosity CMC depressed daily carcass gain, and both viscous diets predisposed the pigs to intestinal proliferation of haemolytic *E. coli*. In chickens, CMC interferes with nutrient absorption within the small intestine and allows pooling of digesta (2). In pigs, soluble dietary viscous polysaccharides increase oro-caecal transit time but also stimulate intestinal myoelectric activity (4). The combination of altered motility and greater transit time apparently favoured the proliferation of enterotoxigenic *E. coli* in this study. This finding has implications for use of feed ingredients which may increase viscosity of the intestinal contents, such as those containing high levels of sNSP. The inclusion of CMC in weaner diets is a novel method of inducing experimental post-weaning colibacillosis without requiring experimental inoculation of *E. coli*.

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