Mucin secretion enhancement to condition the piglet's digestive tract

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Piglets used to be weaned at eight weeks, having been fed exclusively with milk for more weeks than they are today. The feed was not as rich in protein when presented to the piglets and very often the piglets also had access to earth. All these natural aspects disappeared from our way of production for a variety of economical reasons.

Today, we are playing brinkmanship with what can be done by:

- Undertaking the earliest weaning possible, very often at 21 days of age, but at even younger ages for more than 20% of piglets.
- Giving the richest feed possible to ensure a strong meat deposition from the beginning.
- Providing the minimum space to reduce heating costs at the beginning and to reduce the space cost per pig.

Over the first six months of a pig's life their digestive tract will encounter a lot of different challenges. First, the digestive system has to develop with villus growth and the establishment of the gut microflora.

This development is very fragile and equilibrium is hard to find. For any kind of equilibrium, disturbances are not good but unfortunately weaning is a disruption that we can not avoid!

Naturally solid feed supply to piglets starts as soon as possible after weaning to minimise the period of time without any feed in their digestive tracts. This short 'starvation time' has several undesirable effects – villus height decreases, the gut microflora is disturbed, and the space between epithelial cells increases, which allows translocation of bacteria across the gut wall.

As we start early solid feeding by creep feeding we also start disruption of the digestive tract. We are asking this part of the body to simultaneously make use of milk, while it is also preparing to utilise a different kind of feed, with a variable source of proteins and some non-starch polysaccharides.

A lot of techniques now aim to stabilise the gut microflora. This approach is very interesting and works quite well in laboratories but unfortunately it is recognised in literature that it has a very variable effect under conventional on-farm conditions.

On this basis we have to admit that acidification gives some quite good results and that some use of antibiotics or a high level of zinc is also very powerful in some cases.

This being said, we also know that antibiotic growth promoters are not going to return and some discussion at the recent 'Responsible Use of Antibiotics' symposium reinforced this. Because of the environmental impact and green pressures, we can also expect that the use of 3000ppm zinc has a limited life.

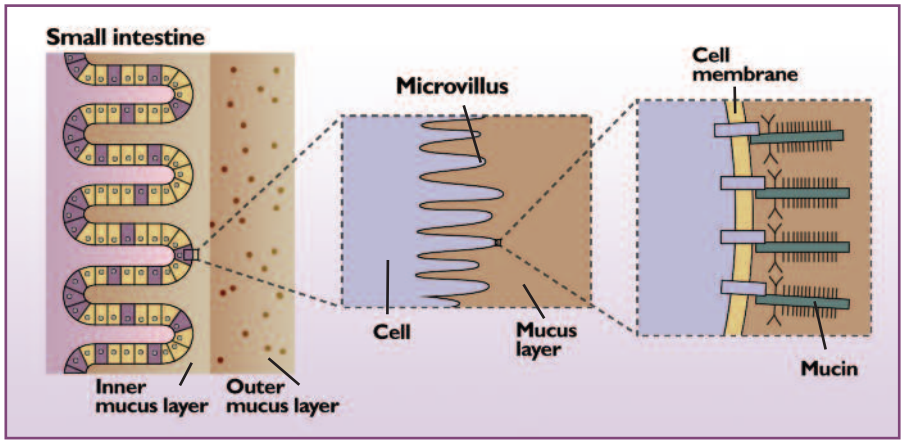
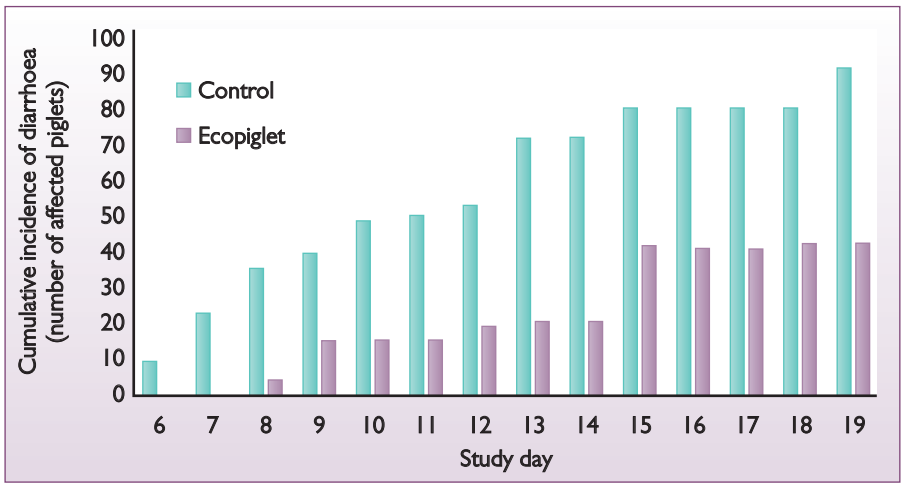


Fig. 2. The digestive tract.

Fig. 1. Cumulative incidence of diarrhoea (number of affected piglets).



New approach

One approach, which has not been explored, is at what level would mucin production enhancement by nutrigenomics be worthwhile?

The main component of mucus, responsible for its viscosity and elasticity and its gel aspect, are the mucins. The different mucins encountered in mammals have certain common specifications – they are all big polymers, they are made of glycoproteins with high glycosylation and each monomer is made of a central peptide nucleus surrounded by a series of oligosaccharides.

Mucins have a key role to play in the protection of the intestinal wall. They represent

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a physical barrier against the bacteria which are present in the intestinal lumen and they also play a role in the inhibition of micro-organism adhesion to the intestinal epithelial cells.

It was shown for instance that pure mucus or highly purified mucins will inhibit the adhesion of Yersinia enterocolitica and E. coli to intestinal villi.

Another study showed that mucin can bind to rotaviruses, thereby reducing their effects on target cells.

Various bacteria can produce several adhesins (nine for E. coli). Adhesins are adhesive proteins that can be fimbriated or not (haemagglutinin, external membrane proteins or toxins) that recognise carbohydrates on target cells and attach to them.

The capacity of mucins to inhibit an organism's adhesion to epithelial cells can be due to similarities between natural sites of adhesion and the mucins' carbohydrates.

Mucin secretion increases in response to numerous enteric infections, mainly those due to toxigenic or invasive as E. coli or Y. enterocolitica.

Mucins are not completely understood and the host response they trigger is probably aimed at repelling micro-organisms and toxins away from mucosa and to ultimately expel them from the intestinal tract. We know that this kind of 'in vitro' knowledge is sometimes difficult to transfer to the field, but if we have got the material it is worth trying.

Table 1. Type of diarrhoea.

	Control	Ecopiglet	P-value
Number of affected piglets	94	45	
Faecal consistency			
Without form, pasty (1)	26 (28.9%)	22 (48.9%)	0.012
Liquid faeces (2)	68 (73.1%)	23 (51.1%)	0.012
Abdominal fill			
Normal/distended (1)	41 (44.1%)	36 (80.0%)	<0.001
Abdomen more retracted than usual (2)	52 (55.9%)	9 (20.0%)	<0.001

	Control	Ecopiglet	P-value
Number of piglets	400	393	
Number of treated piglets	199 (49.8%)	87 (22.1%)	<0.001

Table 2. Concomitant treatments.

Ulvans, polysaccharides from green algae (ulva lactuca) have been shown to have the capacity to enhance the production of mucins (Barcelo et al, 2000). Using this fact, a product was designed based on dried ulva lactuca, with the aim of preparing the intestinal tract of piglets for the challenges they will face.

A study was conducted by an external organisation (Tests and Trials SL, Spain) on a 1200 sow unit with 833 piglets from 72 sows and the results are presented in Fig 1.

Piglets given this commercial mineral complementary feed (Ecopiglet) from day five to weaning were 50% less likely to show clinical signs of diarrhoea. Diarrhoea classification in the affected piglets showed that those receiving Ecopiglet had less severe diarrhoea than the ones included in the control group ($p<0.05$; Table 1).

Faecal samples of the first five affected piglets with diarrhoea type 2 (liquid faeces) were taken and analysed in the laboratory and E. coli and Clostridium perfringens were detected.

Some piglets needed concomitant treatment by injection while suckling, mainly to treat diarrhoea (Table 2). When more than one piglet suffered diarrhoea in a litter, all piglets of this litter were treated.

The treatments were amoxicillin trihydrate (0.15g per 10kg BW) plus colistine sulphate (0.25 MUI per 10kg BW) for three consecutive days or enrofloxacin (50mg per 10kg) for three consecutive days.

In the Ecopiglet group, less treatment was necessary than in the control group ($p<0.0001$) and a second treatment was not needed in any piglet from the Ecopiglet group, while 8% needed a second treatment in the control group.

Conclusion

In conclusion, any kind of feed management that can help reduce the need of medication has to be seriously considered today.

Encouraging the secretion of mucins by selecting specific vegetal extracts from the earth or sea is very promising and must be one of the good alternatives to medication.

The full potential is not yet completely known, but the recent progress of nutrigenomics will help us to quickly identify the right extracts, the right way of extraction and the right combinations.

By looking at mucin gene expression in reaction to different kinds of algae extract, it should now be possible to design new feed additives that allow less medication to be used. ■