SWINE DYSENTERY – AN EDIBLE CURE

Lowering the content of fibre and resistant starch offers a new approach to combating intestinal disease in pigs.

By Professor D J Hampson, D W Pethink, J R Pluske, B P Mullan, Z Durmic, D E McDonald and P M Siba

Pigs often suffer from infections of the intestinal tract. Many infectious agents exist, with some causing local tissue damage in the intestine, and others upsetting normal digestive and or absorptive physiology in more subtle ways. Some microorganisms can go on to cause disease in other parts of the body, reducing the efficiency of the intestine. The associated check in growth has particularly important consequences in terms of production.

The two bacterial pathogens discussed in this article differ in that they colonise different parts of the intestinal tract, and they have different mechanisms causing disturbed intestinal function. Both infections are common throughout the world, with resultant large economic losses.

Swine dysentery - severe damage to the gut

Swine dysentery is an infection of the large intestine (caecum and colon) with an anaerobic intestinal spirochaetal bacterium called Serpulina hyodysenteriae. The condition mainly affects weaners and growers/finishers. Figure 1 illustrates the severe bloody and mucoid inflammation of the colon wall which usually develops. The animals rapidly lose weight and become gaunt; their faeces are loose or watery and typically contain flecks of fresh blood and plugs of mucus. Toxins from the bacteria are thought to contribute to the severe erosions in the colon wall. In some untreated outbreaks up to 50% of pigs may die.

Colibacillosis - high mortality

Postweaning colibacillosis is an infection of the small intestine with certain strains of haemolytic Escherichia coli. By definition, the condition only occurs within the period 3 - 10 days after weaning, and is acute. Infected pigs develop a severe watery diarrhoea, and quickly become dehydrated. Toxins from the bacteria cause an outpouring of water and salts from the intestinal wall, although they do not cause bleeding or obvious physical damage to the wall. Without treatment, mortality rates of up to 100% may occur.

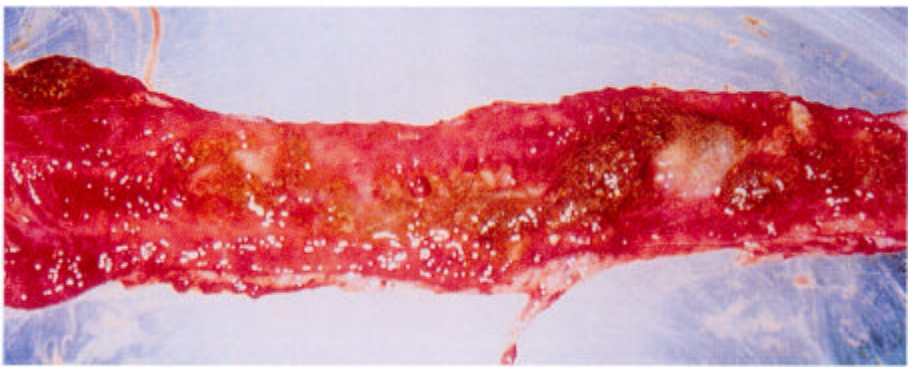

Figure 1 Colon of pig with swine dysentry showing extensive inflammation with fresh blood and mucous

Control through management

The best method to control infection from pathogenic organisms is to prevent their entry into a piggery. This is achievable for S hyodysenteriae, as the spirochaete is not present in all piggeries, but toxigenic E Coli strains are ubiquitous as part of the intestinal flora of the pig. If pathogenic organisms are present in a piggery, their impact may be reduced through management procedures; providing optimal environmental conditions, reducing stressors and minimising transmission or pathogens to susceptible groups of animals.

Segregated early weaning is an example of the latter strategy. In conjunction with this approach, antimicrobial drugs or vaccines are commonly used. But antimicrobials cannot be relied upon as a long term strategy, as pathogens are becoming increasingly resistant. And so far, vaccines developed for these diseases have not been particularly useful. Alternative means to control these two diseases and others are therefore urgently required. This need has stimulated our studies on the use of simple methods based on specific protective diets to control swine dysentery (SD) and postweaning colibacillosis (PWC).

Fibre benefits previously shown

A study carried out by Prohaszka and Lukacs in 1984 showed that swine dysentery did not occur on an infected farm after a highly fibrous (cellulose/hemicellulose) diet based on maize silage was fed. The fibre in the diet was not digested in the small

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a

b

Figure 2 *Caecum and colon of two pigs of the same age and body weight fed diets consisting of either a) cooked white rice and animal protein or b) hammer milled raw wheat and lupin. The commercial diet b contains more dietary fibre and has resulted in more fermentation and an increased colon length.*

intestine; it arrived in the proximal colon where it was broken down by microbial fermentation to produce volatile fatty acids. It was speculated that the diet was protective because of its low base content, which interacted with the volatile fatty acids to create an unfavourable environment for S hyodysenteriae. Previously, Bertschinger et al (1978) had reported that increasing dietary fibre and reducing protein levels in the diets of newly-weaned pigs reduced the occurrence and severity of postweaning colibacillosis.

New and unexpected results

In our initial studies we attempted to examine whether, as reported, a vigorous fermentation in the large intestine would inhibit the growth of S hyodysenteriae. We fed pigs either a commercial diet that was relatively high in fibre (a typical Australian diet comprising mainly wheat and lupins), or an experimental diet containing little fibre (cooked white rice and animal protein).

As expected, much more fermentable material (fibre) reached the large intestine in pigs fed the commercial diet. Consequently their caecal and colonic contents were more acidic, and contained more total volatile fatty acids than those of the pigs on the rice diet. The colon itself also contained more content and was clearly larger in the pigs fed the commercial diet, as illustrated in figure 2.

Pigs receiving the two diets were housed in adjacent pens to enhance the opportunities for transmission of infection, and then were experimentally challenged with cultures of S hyodysenteriae. Unexpectedly, all pigs fed the commercial diet developed swine dysentery while none of those on the experimental low-fibre cooked-rice control diet did - despite heavy exposure to the pathogen.

Absence of fibre found to be protective

This result was confirmed in subsequent trials with larger numbers of pigs fed the two diets. When animals were fed dietary combinations of cooked rice and lupin, and wheat and animal protein these also developed dysentery, showing that the protective effect was not a property of either the cooked rice or the animal protein alone (Siba et al, 1996). It appeared that the absence of fermentable fibre was protective.

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Further studies examined the role of dietary fibre in facilitating colonisation by S hyodysenteriae and development of disease. We added different sources of this material to the protective rice diet before experimental challenge (Pluske et al, 1996a). The fibre sources added included oaten chaff as a source of insoluble non-starch polysaccharides (NSP), guar gum as a source of soluble NSP, retrograde maize starch (novelose) as source of resistant starch, or both guar gum and novelose.

Pigs given oaten chaff were not colonised and did not develop dysentery, while those given guar gum with or without the resistant starch source were both colonised and developed dysentery. In this experiment the pigs given novelose alone were colonised by S hyodysenteriae but did not develop SD, although in a subsequent (unpublished) experiment some animals given different levels of this material also developed the disease.

Reduced fermentation may prevent colonisation

The mechanisms involved in this dietary protection from swine dysentery is not clear. It is known that the presence of components of the large intestinal microflora are important in facilitating colonisation by S hyodysenteriae, hence we speculated that overall reduced fermentation in the large intestine in pigs fed cooked rice and animal protein led to changes in the microflora, which in turn did not permit colonisation.

Pigs fed the protective diet also had dry colonic contents and faeces due to the reduced fermentation, and it is possible that these physical changes inhibited the spirochaete.

Refining the diet

Whatever the precise mechanism involved, we have identified a diet based on cooked white rice and animal protein that gives complete protection against SD even under intense experimental challenge. This diet has the potential to be used for treatments in outbreaks of SD, or as part of eradication or other control programmes. Unfortunately the protective diet is both labour-intensive to produce and is expensive - thus limiting its usefulness. We therefore looked for alternative protective diets which are potentially cheaper and more convenient. We repeated the same basic experiments using five different cereal grains known to have different levels of NSP (barley, groats, maize, sorghum and wheat). Each of these cereals were fed either hammer milled or steam flaked, to reduce levels of resistant starch. The diets were supplemented with animal protein to avoid complications of addition of sources of plant proteins.

Low NSP, low resistant starch required

In this study we found that fewer pigs fed diets based on steam flaked maize and steam-flaked sorghum developed disease than did those fed on the other diets (11-33% compared to 75-100%; Pluske et al, 1996b). These two cereals therefore appear to be useful starting points for the construction of other protective diets. Most significantly, analysis of resistant starch and NSP levels in the 10 diets led to the identification of interactions between the source of fibre, and to some predictive values.

Firstly, the incidence of disease was found to be reduced with lower levels of soluble NSP in the diet. Secondly, in diets with less than 1.5% soluble NSP there was also a positive linear correlation between increased levels of resistant starch, as presented in figure 3. Therefore, protective diets should contain both low soluble NSP (less than 1.5%) and low resistant starch.

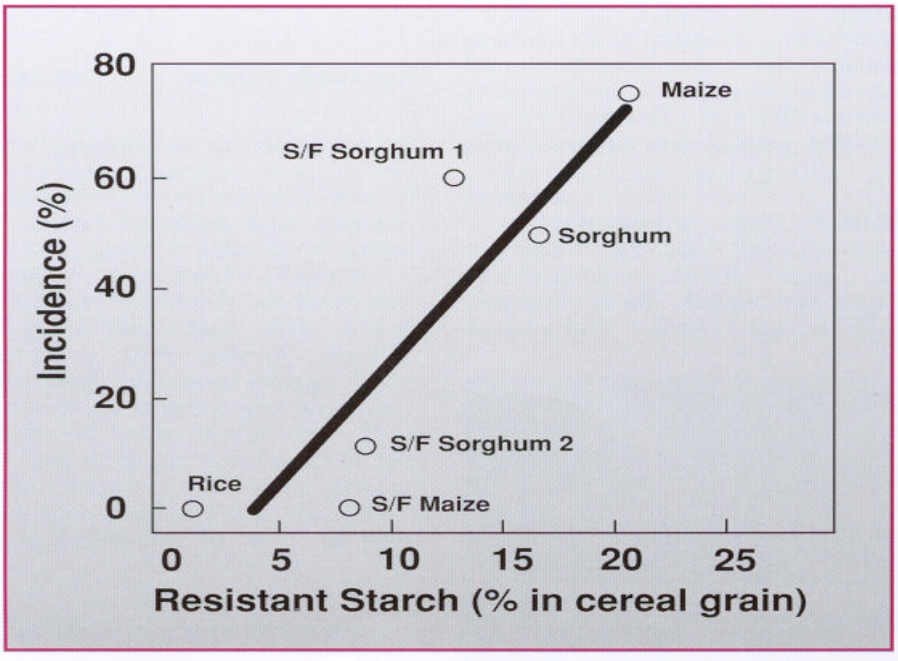

Figure 3 Relationship between level of resistant starch in diets containing less than 1.5g/100g soluble NSP and incidence of swine dysentery in groups of infected pigs (from Pluske et al, 1996b)

Extrusion and enzymes to enhance protection

In practical terms, besides careful selection of ingredients, physical treatment such as extrusion can be used to reduce resistant starch levels. It is well known that the addition of exogenous enzymes can reduce NSP concentrations in the diet, and we are currently pursuing the possibility of using such enzymes, for example xylanases, amylases and proteases, with or without physical treatments, to reduce NSP and resistant starch concentrations in pig diets based on common cereal grains.

Whether or not such treatments will be sufficient to produce diets that offer complete protection against swine dysentery is not known, but it is an approach that has the potential to be a cost-effective means of applying the principle.

Experiments with PWC

As a continuation of our studies with swine dysentery, which is an infection of the large intestine, we have examined the role of dietary fibre in susceptibility to post-weaning colibacillosis - an infection of the small intestine. Following weaning, the size and content of the entire intestinal tract of the piglet rapidly increases as a result of consumption of solid food (figure 4). The amount of fermentation in the large intestine increases as undigested fibre from the weaner diet arrives there from the small intestine.

In order to investigate the role of dietary fibre, we weaned animals onto diets consisting of the highly digestible cooked rice-animal protein, with or without the addition of 10% guar gum as a source of soluble NSP (McDonald et al, 1996). Other pigs were fed a commercial weaner diet

Figure 4

Relative size of the digestive tract in two litter-mates at four weeks old.

Top: tract from an unweaned piglet

Bottom: tract from a piglet weaned for five days

based on wheat, barley and lupins. After two days, half the pigs in all three groups were challenged with toxigenic strains of E.Coli, then killed three days later.

Positive results with rice diet

Interestingly, the pigs fed the cooked rice-animal protein diet alone had significantly greater empty body weight (intestinal tracts removed) than those receiving the other two diets (rice-animal protein diet with guar gum, or commercial wheat/barley/lupins diet). In the latter two groups it appeared that considerable post-weaning growth was put into the large intestine, at the expense of the rest of the carcase.

Furthermore, pigs fed the cooked rice-animal protein diet supplemented with fibre in the form of guar gum had much higher E. Coli counts throughout the small and large intestine than did those fed this diet without guar gum. The addition of a soluble NSP (guar gum) to this diet was therefore detrimental to the pigs' health.

Protective effect of low-NSP

The mechanism by which the NSP stimulated proliferation of the pathogenic bacteria is not known. It may have increased the viscosity of the intestinal contents - in turn prolonging intestinal transit times, and increasing the availability of substrate for growth of the pathogenic bacteria in the small intestine. In contrast, the pigs fed the cooked rice-animal protein diet without guar gum maintained low levels of E. Coli throughout the intestinal tract, despite a heavy experimental challenge.

Clearly, much further work is required to examine the effects of different forms of fibre in the weaner diet in relation to both body growth and proliferation of pathogenic E. Coli. This preliminary work suggests that weaner diets containing little soluble dietary fibre have potential benefits in both areas, and that hydrolysed rice may be a particularly suitable form of cereal grain for young weaner pigs.

A new paradigm

The current work seems to have created a new paradigm - diets that are low in soluble fibre protect from intestinal infections. In our work we have shown that two very different intestinal infections of pigs can be ameliorated, or even completely prevented, by the use of appropriate protective diets. In both cases diets low in fibre, particularly NSP, but also resistant starch (in the case of swine dysentery) reduced colonisation by the two pathogens. This new direction is particularly appealing - it is uncomplicated and effective.

We have not identified exactly how the protection works, and the mechanisms involved are likely to be different in the two infections. Clearly, further investigation is required. Since diets either selected for or treated to reduce soluble fibre can be relatively expensive, a better understanding of the protective mechanisms involved may identify alternative and cheaper ways of activating protection.

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