

Nutritional management of the gastrointestinal tract to reduce enteric diseases in pigs

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Summary

This paper discusses the general hypothesis that certain enteric diseases in pigs can be controlled, or at least ameliorated, by nutritional management of the gastrointestinal (GI) tract. The impetus for such an approach is the mounting concerns amongst retailers, the public, feed manufacturers, producers and regulatory bodies regarding the 'safety' of the production chain supplying pig meat. The prime concern is the use of antibiotics, recent events in Europe sending a clear signal that alternatives to the use of viable and cost-effective growth-promoting antibiotics will be required sooner rather than later. Results of research summarized in this paper suggest that associations, currently largely undefined, exist between bacterial pathogens, the host, and dietary substrates to cause diseases in the GI tract. Available data implicate the non-starch polysaccharide (NSP) and amylase-resistant starch in the clinical expression of a number of economically important bacterial diseases. The use of highly-digestible cereal sources such as cooked white rice limits the severity of enteric diseases such as post-weaning diarrhoea, swine dysentery and porcine intestinal spirochaetosis. Interactions with other dietary components in the aetiology of enteric diseases also are possible, including the level and type of protein fed to pigs.

Keywords: pig, post-weaning, diarrhoea, spirochaetes, microflora, nutrition

Introduction

The activity of pathogenic bacteria in the gastrointestinal (GI) tract of pigs causes considerable economic losses to the Australian pig industry. A number of distinct bacterial species are involved, and each of these pathogens tends to colonise and cause disease in different regions of the GI tract. Furthermore, each of the associated diseases occurs predominantly in pigs of certain ages or classes; examples are *Escherichia coli* diarrhoea in suckers and/or weaners, and swine dysentery and porcine intestinal spirochaetosis in

growers and finishers (Hampson *et al.* 2001). Presently the major form of control of these diseases is the use of antibiotics, and these are given to pigs to treat overt disease, to provide prophylaxis in situations where disease is liable to occur, and to improve growth rates in the absence of disease. In a number of European countries, however, the use of most antibiotics in pig diets for growth promotion has been banned, or in some instances imposed on a voluntary basis, as fears mount regarding the implications for both animal and human health about continued use of antibiotics in the intensive livestock industries. These fears include, for example, the transfer of multi-drug resistant zoonotic pathogens such as *Salmonella* spp. and *Campylobacter* spp. from pigs to humans, the direct or indirect transfer of resistance genes from the porcine intestinal microflora to human bacterial strains, and the presence of antimicrobial drug residues in pig meat (Hampson *et al.* 2001).

Other considerations include issues such as the protection of export markets, sustained domestic consumption of pork in light of growing fears about the overall 'safety' of meat production, and the continued use of animal protein sources such as meat and bone meal in light of concerns about its safety as a livestock feed. Consequently a high priority for the Australian pig industry should be investigations into the development of alternative means of controlling bacterial infections to safeguard markets while maintaining international competitiveness. The aim of this paper is to review aspects of current and past research related to the nutritional management of the GI tract in the expression of certain enteric infections. We will focus on post-weaning diarrhoea (PWD) caused by enterotoxigenic strains of *E. coli* and diseases caused by *Brachyspira* spp. To do this requires some background about the microbiota of the GI tract of the pig and the types of substrates the resident bacteria require to live and reproduce, between which we suspect an important association exists that determines whether or not a pig succumbs to enteric disease.

What comprises the normal microbiota?

The microbiota is characterized by its high population density, extensive diversity, and complexity of interactions throughout the GI tract. The stomach and proximal small intestine contain relatively low numbers of bacteria due to low pH and rapid flow. In the small intestine for example, digesta flow rate and hence the rate of bacterial washout exceeds the maximal growth rates of most bacterial species. In contrast the distal small intestine harbours a more diverse and numerically greater population of bacteria. The large intestine is a major site of microbial colonisation because of slow digesta turnover. The contents of the colon support in excess of 500 different bacterial species with numbers as high as 10^{10} and 10^{11} culturable bacteria per g (wet weight) of digesta (Gaskins 2001). The majority of these bacteria are strictly anaerobic Gram-positive bacteria, the major bacterial groups isolated from pig faeces (in order of prominence) being *Streptococcus*, *Lactobacillus*, *Eubacterium*, *Fusobacterium*, *Bacteroides*, *Peptostreptococcus*, *Bifidobacterium*, *Selenomonas*, *Clostridium*, *Butyrivibrio* and *Escherichia* (Moore *et al.* 1987). The Gram-negative organisms comprise about 10% of total culturable bacteria, with most isolates belonging to the *Bacteroides* and *Prevotella* groups (Russell 1979). The hindgut flora is considered to be both diverse and stable, the many species and strains appearing to coexist without one or few ever becoming dominant.

There are many uncertainties about what constitutes the composition of the 'normal' microflora of the pig. Three major problems with the study of the intestinal microbial ecosystem are:

- culturing can only be performed on those organisms for which nutritional and growth requirements are known
- the lack of a phylogenetically based classification scheme
- the unavoidable bias introduced by culture-based enumeration and characterisation techniques (Gaskins 2001).

Modern molecular techniques based on comparative sequence analysis of small subunit ribosomal RNA (16S rRNA) molecules can be used to provide molecular characterisation (Simpson *et al.* 1999), while at the same time provide a classification system that predicts natural evolutionary relationships (Pace 1997). A recent study of microbial diversity in the mucosal layer of the pig colon with molecular analysis compared with culture-based methods highlights the disparity between these two approaches. Pryde *et al.* (1999) demonstrated that *Streptococci* and *Lactobacilli* comprised the majority of isolates recovered (54%) from the colon wall by culturing, but these groups accounted for only one-third

of the sequence variation for the same sample from random cloning. In addition, 59% of randomly cloned sequences showed less than 95% similarity to database entries or sequences from cultivated organisms. These data show the problem of cultivation bias and illustrate the need for molecular-based techniques if there are to be advances in our understanding of nutrition-disease interactions.

The diversity of the hindgut microflora, and that of the distal region of the small intestine, reflects in part the variety of nutrient substrates found in their environments. The diversity of bacterial populations within a particular ecosystem is directly related to the number and composition of limiting nutrients, since each of these will support the one bacterial species or strain that is most efficient in utilizing it. Moreover, the stability of these bacterial populations will also be influenced by the inhibitory actions of a number of compounds such as short-chain fatty acids (SCFA), H_2S , deconjugated bile salts and bacteriocins (Gaskins 2001). In this regard we suspect that certain nutrients and their associated physicochemical effects might also play a major role in maintaining the balance of the microflora in these parts of the GI tract, and subsequently in determining whether a pathogenic bacteria proliferates to cause overt expression of disease.

Nutrition of the lower gut

The main source of growth substrate for the gastrointestinal microflora comes from the diet, although endogenous secretions can also be utilised by different classes of bacteria. Simple sugars tend to act as the main growth substrate for bacteria in the upper part of the GI tract, whilst in the large intestine, where the main bacterial biomass is located, dietary fibre (DF; predominantly NSP) serves as the major substrate. Non-starch polysaccharides include a large variety of polysaccharide molecules, often in association with phenolic lignified polymers, protein and starch, that have glucosidic bonds other than the α -(1→4), (1→6) bonds of starch. The building blocks of plant cell wall polysaccharides are the pentoses (arabinose and xylose), hexoses (glucose, galactose and mannose), 6-deoxyhexoses (rhamnose and fucose) and uronic acids (glucuronic and galacturonic acids). These monomers are chemically linked to each other to build various NSP in the plant cell walls of both cereals and legumes. The major NSP of plant cell walls, therefore, are cellulose (linear β -glucan chains), non-cellulosic polysaccharides (arabinoxylans, mixed-linked β -glucans, mannans, galactans, xyloglucan), and pectic polysaccharides (polygalacturonic acids, which may be substituted with arabinan, galactan, and arabinogalactan) (Theander *et al.* 1989; Bach Knudsen 1997; Choct 1997). The NSP, in turn, can be divided into a 'soluble' fraction and an 'insoluble' fraction. In chickens, and to a lesser degree in pigs, the soluble fraction of NSP can cause formation of a viscous digesta

that reduces nutrient digestibility and absorption. Increased viscosity will also decrease the transit time of digesta through the upper small intestine of pigs (Cherbut *et al.* 1990) and this process might provide more time for microbial pathogens such as *E. coli* to proliferate. This will be discussed in a later section.

The amount and type of NSP available to the microbiota in the (lower) GI tract, and the age of the pig (Shi and Noblet, 1993), will largely determine the extent and nature of the fermentation that occurs. Consequently different forms of dietary NSP can broadly influence the composition and metabolic activity of the large intestinal microflora (see Pluske *et al.* 1999, 2001 for reviews; Reid and Hillman 1999). Currently, however, and even where addition of appropriate substrate is known to stimulate proliferation of specific groups of resident bacteria, little is known about the way in which these bacteria interact with pathogenic species of bacteria. This lack of information therefore makes it difficult to predict how a given dietary component could be used to indirectly influence a given enteric pathogen.

Durmic *et al.* (1998) used culturing techniques and found that pigs experimentally infected with *B. hyodysenteriae* after being fed diets known to allow expression of swine dysentery (SD) showed changes in bacterial populations consistent with those that occur in the natural disease (Whipp *et al.* 1979). Leser *et al.* (2000), using a molecular-based technique, did not detect the same synergistic bacteria in pigs infected with *B. hyodysenteriae*, although they did report changes in bacterial populations when pigs were fed either a cooked rice diet or a fermented liquid feed following infection with the causative agent.

Besides influencing the commensal microflora, diet could also influence colonisation by pathogens through other routes. For example it could act by modulating the amount of specific substrate available for the pathogen at a given site, by influencing viscosity of the intestinal contents and hence altering accessibility of receptor sites and (or) affecting intestinal motility, and by direct or indirect effects on the intestines (Brunsgaard 1998). Similar changes may occur in specific colonisation sites or bacterial receptors on the enterocytes. The diet also might influence intestinal function; for example, components in boiled rice inhibit secretions of electrolytes, particularly chloride, in the small intestine and hence reduce the magnitude of secretory diarrhoea due to pathogens such as enterotoxigenic *E. coli* (Mathews *et al.* 1999).

Despite a lack of detailed knowledge, the general contention that dietary components can in some way influence colonisation or disease expression by pathogens is consistent with reports from the field, where it is often observed that changes to the diet result in either increased or decreased enteric disease. For example it has been reported that units adopting liquid feeding of by-products or using fermented wet feed have a lower incidence of Salmonellosis than herds using dry

feed (Stege *et al.* 1997). Pearce (1999) demonstrated a strong positive association between levels of NSP in diets provided for growing pigs on commercial farms in the UK and the prevalence of the endo-parasites *Ascaris suum* and *Trichuris suis*. Collectively these data suggest that interactions occur between the feed presented to a pig and the proliferation and expression of certain enteric diseases in pigs. At Murdoch University and Agriculture WA we have been interested in the interactions between dietary components such as DF and starch on enteric diseases of pigs. The rest of this paper reports in more detail on some of the diseases we have studied.

Post-weaning diarrhoea

The growth checks and diarrhoea that occur in many pigs after weaning are a serious industry problem. Colonisation of the small intestine by enterotoxigenic strains of *E. coli* in this period results in severe secretory diarrhoea (post-weaning diarrhoea, PWD). Besides mortalities and the requirement for antimicrobial medication, the associated growth checks result in increases in the time pigs take to reach market weight (Hampson *et al.* 2001). Post-weaning diarrhoea is a multifactorial condition, and there are various dietary influences on the disease. For example, diets containing high concentrations of protein (210 g/kg) have been shown to predispose piglets to the condition (Prohászka and Baron 1980). Some highly digestible and milk-based weaner diets have been associated with increased PWD, whilst conversely it has been suggested that the inclusion of DF in weaner diets will reduce its incidence and severity (Bertschinger *et al.* 1978; Bolduan *et al.* 1988). Aspects of this will be discussed later in this section.

Recent research on PWD by D.E. McDonald and colleagues at Murdoch University has examined the interrelationships between different sources of NSP and proliferation of *E. coli* in weaner pigs. Increased proliferation of pathogenic *E. coli* in both the small and large intestines has been seen with addition of either guar gum (McDonald *et al.* 1999) or pearl barley (McDonald *et al.* 2000a) to the diet of 21-day-old weaner pigs. For example, McDonald *et al.* (2000a) assessed the effect of adding a soluble NSP source (pearl barley) to a cooked white rice-based diet on the performance, gastrointestinal physiology and intestinal proliferation of enterotoxigenic *E. coli* in weaned pigs experimentally inoculated with *E. coli* O8; K87; K88. Pigs were infected at 48, 72 and 96 hours after weaning and were allowed *ad libitum* access to their feed. Pigs were euthanased seven to nine days after weaning. Pigs fed the rice-based diet grew faster, had a greater empty-bodyweight gain, and had a reduced large intestinal weight (expressed as a proportion of empty bodyweight) than pigs fed the rice-based diet supplemented with pearl barley (Table 1). Higher concentrations of volatile

fatty acids and a lower pH of digesta in the large intestine (data not shown) indicated greater fermentative activity in pigs fed the pearl barley-based diet. Pigs offered the rice-based diet showed less of a reduction in empty bodyweight gain associated with *E. coli* infection (Table 1) and showed less proliferation of *E. coli* (2 to 2.5 log units) in the small and large intestine than their barley-fed counterparts. Addition of a β -glucanase enzyme to the pearl barley-based diet failed to reduce viscosity (data not shown) or influence bacterial counts.

The addition of pearl barley to the rice-based diet altered the physico-chemical properties in the intestines, increased the ileal viscosity and altered the site of microbial fermentation. The energy expended in adapting the intestinal tract for digestion of NSP caused a depression in carcass growth, and this was exacerbated by PWD. These data suggest that the presence of soluble NSP in weaner diets is detrimental for piglet growth and causes proliferation of *E. coli* in the small intestine. It also indicates that there are benefits in feeding a highly-digestible rice-based diet to weaners, although it is not at present known what mechanism(s) promote this protection. Further understanding is needed because highly-digestible rice-based diets may prove to be a viable option to the use of growth promoting antibiotics and minerals currently used in the control of PWD.

The two types of soluble NSP used by McDonald et al. (1999, 2000a) were highly fermentable and viscous in nature, which raises the question of whether fermentability, viscosity or combinations of both are likely to influence the small intestinal microbiota. To further investigate the potential detrimental effects of increased intestinal viscosity in weaner pigs on proliferation of enterotoxigenic haemolytic *E. coli*, McDonald et al. (2000b) fed experimental diets supplemented with two sources of carboxymethylcellulose (CMC) to 21-day-old weaned pigs for 10 days. CMC is a water-soluble synthetic viscous polysaccharide resistant to microbial

fermentation. The pigs were then euthanased and the effects of two types of CMC, either low-viscosity (50–200 cP *in vitro*) or high viscosity (400–800 cP *in vitro*), on gastrointestinal development, growth performance, faecal dry matter and proliferation of haemolytic *E. coli* were monitored.

Dietary CMC increased the viscosity along the entire lumen of the small intestine and in the caecum, and resulted in increased intestinal weights. Pigs fed the rice-based diet remained healthy, whereas those fed either low- or high-viscosity CMC developed diarrhoea within seven days of weaning which continued until they were euthanased on day 10. Pigs fed the low- or high-viscosity CMC diets shed more haemolytic *E. coli* (serovar 0141; K88) daily than pigs fed the rice-only based diet (Table 2).

The presence of CMC might provide a favourable luminal environment for the establishment and growth of bacteria, especially *E. coli* (McDonald et al. 2001). *Escherichia coli* possess fimbriae that allow it to attach to the brush border of the small intestinal villi, but also allow the bacteria to attach to the mucus lining the intestinal tract (Conway 1994). Of particular interest is

Table 2 The effect of dietary CMC (40 g/kg) added to a cooked rice-based diet on the presence of diarrhoea in pigs after weaning (number of pigs with diarrhoea expressed as a proportion of total number of pigs in group) (after McDonald et al. 2000b).

Diet	Day 7 [*]	Day 8	Day 9	Day 10
Rice	0/8 ^a	1/8 ^a	0/8 ^a	0/8 ^a
Rice + low viscosity CMC	5/8 ^b	3/8 ^b	4/8 ^b	4/8 ^b
Rice + high viscosity CMC	7/7 ^b	7/7 ^b	7/7 ^b	5/7 ^b
<i>P</i> -value	<0.05	<0.05	<0.05	<0.05

^{a,b} Values within a column with different superscripts differ significantly ($P < 0.05$)

^{*} Number of days after weaning (weaning is day 1)

Table 1 Performance, large intestinal weights and ileal viscosity in non-infected and infected pigs fed either a rice-based diet or one containing pearl barley (after McDonald et al. 2000a).

Item	Non-infected pigs		Infected pigs		<i>P</i> -value ³	
	Rice ¹	Barley ²	Rice	Barley	Diet	Infection
Total weight gain (kg)	1.4 (0.36) ³	0.9 (0.76)	1.4 (0.36)	-0.6 (0.12)	*	**
EBW ⁴ gain (g/day)	74 (30.3)	26 (6.5)	-28 (10.9)	-56 (16.1)	**	****
Large intestine as						
% pig weight	2.7	3.8	2.6	3.2	****	NS
Ileal VFA (mmol/pig)	18.2	9.1	11.5	13.5	Interaction = 0.053	
Viscosity in ileum (cP)	2.1 (0.31)	2.8 (0.56)	1.6 (0.22)	2.3 (0.29)	**	**

¹ Rice: cooked white rice (702 g/kg, 4 g/kg dietary soluble NSP content) plus animal protein sources (197 g/kg)

² Barley: pearl barley (500 g/kg; 25 g/kg dietary soluble NSP content) plus rice (275 g/kg) and animal protein sources (200 g/kg)

³ Data are means \pm SEM; NS: not significant, * $P < 0.10$, ** $P < 0.05$, *** $P < 0.01$, **** $P < 0.001$

⁴ EBW: empty bodyweight (weight of pig at slaughter minus weight of the contents of the gastrointestinal tract)

the fact that CMC adheres to and thickens porcine mucin (Rossi *et al.* 1996), and may also alter its composition. These events may enhance the ability of haemolytic *E. coli* to bind to the mucus lining the intestinal villi and cause diarrhoea, as there is evidence (reviewed by Deplancke and Gaskins 2001) that pathogenic microbes are capable of mucolysis.

Altered microbial activity also has been noted in other species in association with increasing viscosity of the small intestinal contents (e.g. Choct *et al.* 1996; Smits *et al.* 1998; Langhout 1998; Langhout *et al.* 2000). For example, *E. coli* numbers and total anaerobic counts in the ileum of chickens increased significantly with the addition of citrus pectin (Langhout 1998), whilst total microbial counts (aerobic and anaerobic) increased in the duodenum and jejunum of chickens fed diets containing CMC (Smits *et al.* 1998). Wyatt *et al.* (1988) found that the addition of CMC in diets for rats failed to increase the density of bacteria in the caecal or colonic contents, but the bacterial populations changed significantly such that the aerobic bacteria, in particular *E. coli*, were more numerous in the large intestine.

Interactions with dietary protein?

Research to date on the influence of diet on PWD has largely focused on the role of carbohydrates. Less attention has been given to the digestion and metabolism of resistant protein by intestinal bacteria in pigs, and consequently knowledge of the proteolytic activities of gut bacteria in the pig is relatively poor. It is recognized, however, that a number of bacterial groups including *Bacteroides*, *Clostridium*, *Enterobacterium*, *Lactobacillus* and *Streptococcus* possess the ability to produce amines, such as putrescine, cadaverine, histamine and tyramine, via decarboxylation of amino acids and breakdown of polyamines (Gaskins 2001).

These amines have often been implicated in the aetiology of PWD. In the 3-week old weaned pig for example, Porter and Kenworthy (1969) observed that increased urinary heterocyclic amine excretion was associated with diarrhoea after weaning, with putrescine and cadaverine levels being particularly high. They commented that it was not likely to be the absolute amount of these amines produced but their site of production that might precipitate PWD. Porter and Kenworthy (1969) reported that in severely diarrhoeic pigs the small intestine was the main site of amine production, whereas in clinically unaffected pigs there was only a low level of amine production in the small intestine. Prohászka and Baron (1980) reported increased numbers of haemolytic *E. coli* strains when pigs were fed 210 g/kg crude protein as opposed to 130 g/kg crude protein. Furthermore, Hampson (1987) reviewed the work of several authors also implicating the type and quantity of protein fed in the aetiology of PWD. More recently Aumaitre *et al.* (1995) showed that the activity of major gut proteases (trypsin, chymotrypsin) were stimulated after weaning by an increase in the level of protein in the weaning diet of up

to, but not exceeding, 200 g/kg. This helps to explain, at least partly, data showing a decrease in the apparent ileal digestibility of nitrogen in pigs fed diets containing more than 225 g/kg crude protein.

Furthermore, and in agreement with earlier work, Aumaitre *et al.* (1995) suggested that the activity of proteases are reduced after weaning either by the presence of fish meal or fish protein concentrate or by the presence of high amounts of soybean meal in the diet. The extra protein would move distally and could be decarboxylated to amines that, in turn, could predispose the young pig to diarrhoea. In contrast, other authors (e.g. Pouteaux *et al.* 1982; Etheridge *et al.* 1984) have failed to find any association between dietary protein source and the incidence of diarrhoea after weaning.

There are many possible reasons for these discrepancies between studies. For example, very few (if any) studies have used controlled infection studies. Another possible reason might be the influence of DF and its interaction with indigestible protein. Bolduan *et al.* (1988) commented that the crude fibre content of a starter diet should be about 50 g/kg in order for the hindgut to be 'activated' after weaning. Bolduan *et al.* (1988) also presented evidence showing that the production of diamines in the colon reduces linearly with an increase in the crude fibre content of a weaner feed.

On the basis of this work, it is possible that 'appropriate' fibrous feedstuffs could be added to weaner diets to supply fermentative substrates to the flora of the large intestine, thereby promoting physiological and functional development. In turn, stimulation of acid fermentation based on these NSP could decrease the formation of diamines in the colon that have been implicated in the aetiology of PWD. Further research is required to identify 'appropriate' fibre sources, with control of protein source and level to separate the potential confounding effects with type and level of DF on PWD.

Swine dysentery and porcine intestinal spirochaetosis

Swine dysentery (SD) causes substantial economic loss for the pig industry in many parts of the world. The disease results from infection of the caecum and colon with an anaerobic intestinal spirochaete, *Brachyspira (Serpulina) hyodysenteriae*. The spirochaete colonises the mucus layer and crypts of the large intestine and induces a severe mucohaemorrhagic colitis and dysentery. The disease is most usually seen in grower and finisher pigs, and can result in severe growth depression and variable mortality. Numerous reviews by our group (e.g. Pluske *et al.* 1997; Hampson *et al.* 2001) have covered aspects of the nutritional control of SD, although it is worth reiterating the relationship discovered between NSP content of the diet and the

incidence of SD (Figure 1). The amount of amylase-resistant starch in a diet was also related to the incidence of SD (Pluske et al. 1996).

Porcine intestinal spirochaetosis (PIS) is a chronic diarrhoeal disease of weaner and grower/finisher pigs, resulting from colonisation by the anaerobic intestinal spirochaete *Brachyspira pilosicoli* (Trott et al. 1996; Ochiai et al. 1997). As with the closely-related *B. hyodysenteriae*, *B. pilosicoli* colonises the caecum and colon. Unlike *B. hyodysenteriae*, however, which is chemotactic to mucus and moves deep into the crypts, *B. pilosicoli* remains largely in the lumen of the intestine, or may attach by one cell end to the epithelium adjacent to the intestinal lumen (Hampson and Trott 1995).

In view of the close similarity between *B. pilosicoli* and *B. hyodysenteriae*, their very similar habitats in the hindgut, and reports from veterinarians in the field of dietary influences on PIS, an investigation was made into whether the cooked rice diet that protects from swine dysentery might also protect from PIS (Hampson et al. 2000). Two groups of weaner pigs were fed either a standard commercial wheat/lupin weaner diet (n = 8) or the rice-based diet described above (n = 6) for three weeks after weaning. All pigs were then challenged orally over three days with 10^{10} active mid-log phase cells of a Western Australian field strain of *B. pilosicoli* (strain 95/1000). The pigs were killed 3–4 weeks post inoculation (p.i.). All animals became colonised with *B. pilosicoli* strain 95/1000, but this occurred significantly later (mean of 10 days p.i. compared to 3 days), and lasted for significantly less time (mean of 5 days compared to 16 days), in the pigs fed the cooked white rice diet compared to those fed the wheat-lupin based diet. One pig fed the wheat-lupin diet developed an acute and severe erosive colitis with

severe watery diarrhoea within three days p.i., and was euthanased. All the other pigs on both diets developed mild transient diarrhoea, lasting only 2–3 days. Small areas of mild patchy colitis were observed at post-mortem, but no spirochaete attachment to the epithelium was detected. This study demonstrated that as with *B. hyodysenteriae* in grower pigs, colonisation by *B. pilosicoli* can be influenced by diet. In this case the rice-based diet did not prevent colonisation, but only retarded it.

Conclusions

We have made several observations showing associations between the type of diet fed to pigs and the proliferation and expression of several economically important diseases affecting the Australian pig industry. Whilst it is apparent that several dietary components are implicated in the expression of these diseases, the exact mechanism(s) whereby this protection is afforded is largely unknown. Future advances in the area of diet-disease interactions will be made by increasing our understanding of the events that occur as a consequence of nutrition at the interface between the microflora and the host, that is, at the mucosal epithelium. Detailed studies are required to establish the composition of the microflora under 'normal' and 'diseased' conditions.

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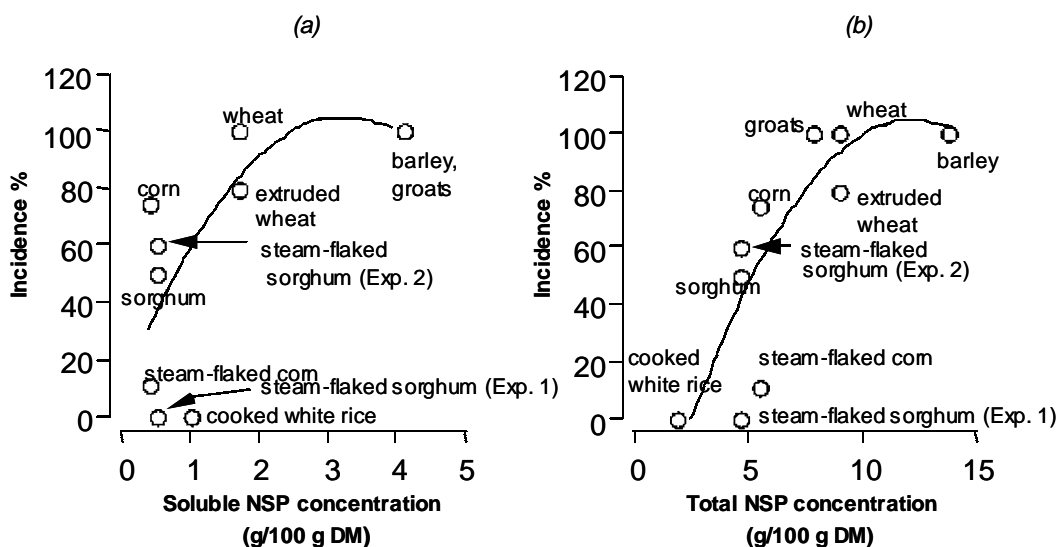


Figure 1 The relationship between the incidence of swine dysentery (Y) and (a) soluble NSP concentration ($Y = 9.52 + 56.98X - 8.47X^2$; $R^2 = 0.561$, $P = 0.016$), and (b) total NSP concentration ($Y = -57.97 + 26.85X - 1.10X^2$; $R^2 = 0.712$, $P = 0.002$), in pigs fed different diets (after Pluske et al. 1996).

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