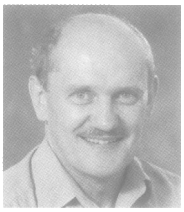
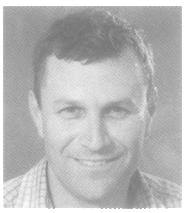
Effect of diet on faecal consistency in normal pigs of 25 kg bodyweight. (left) Soft faeces from a pig fed a wheat-based diet and (right) hard, black pelleted faeces from pigs fed a diet based on highly digestible cooked rice

Role of diet in managing enteric disease in pigs

DAVID HAMPSON AND JOHN PLUSKE



David Hampson graduated from the Royal Veterinary College in 1979. After postgraduate studies at the Universities of Cambridge and Bristol, he worked at Massey University in New Zealand and then joined Murdoch University in Western Australia where he is currently professor of veterinary microbiology. He holds a PhD for studies on porcine postweaning diarrhoea.



John Pluske graduated from the University of Western Australia with a degree in agricultural science in 1988. He is currently associate professor of animal physiology and nutrition at the School of Veterinary and Biomedical Sciences, Murdoch University. He holds a PhD for studies on nutrition and intestinal physiology of the weaner pig.

GASTROINTESTINAL diseases in pigs can have a large number of different causes and, depending on their severity and distribution in a herd, may result in significant economic losses due to mortalities and/or poor and uneven growth rates. Specific enteric bacterial pathogens are responsible for a large proportion of this disease burden and the control of such infections can be difficult. Effective vaccines are available for only a few of these diseases and thus control often requires the implementation of a combination of different management strategies, usually involving considerable routine prophylactic antimicrobial usage. Unfortunately, the associated bacterial pathogens are increasingly found to have reduced susceptibilities to antibiotics, and this coincides with mounting societal and regulatory pressures to reduce routine antimicrobial therapy for all animal species. Veterinary surgeons and producers therefore need alternative means of controlling such infections. This article outlines how the management of some enteric diseases in pigs might be improved by dietary modification.

ENTERIC DISEASE AND DIET

Enteric diseases result in disturbed gastrointestinal function. The underlying disease may have a physical basis (eg, intestinal obstruction) but, in the case of pigs, is frequently due to a nutritional or infectious cause. From a clinical perspective, the most obvious outcome is diarrhoea, usually accompanied by reduced growth rates caused by impaired digestive function and/or the presence of specific pathological lesions that may accompany the disease. Reduced and uneven growth rates, commonly resulting in increased variation in slaughter weights, are often responsible for the greatest economic impact at herd level.

Infectious diarrhoea due to common endemic agents occurs in various age groups of pigs and at each stage is associated with a relatively limited range of specific pathogens. Generally, these diseases are rare in adult breeding animals. Bacterial infections of young and growing pigs that have been reported to be responsive to dietary changes are listed in the table on the facing page.

'Nutritional diarrhoea', from which no specific infectious agents are routinely isolated, is most likely to be seen in piglets before or just after weaning, before their digestive function is fully developed. It is usually associated with the consumption of unsuitable ingredients, and has been especially linked with diets containing certain wheats, soya bean meal and poor quality animal proteins (in countries where animal proteins are still fed to livestock).

General principles of dietary control

Before prophylactic or therapeutic changes are implemented to a diet, it is vital to make an accurate diagnosis of the disease present. This is essential as no single dietary change has proved useful in helping to control all of the various conditions that may occur. Many piggeries may also have different combinations of potential pathogens present and these should all be considered before dietary changes are made. In addition, the possible involvement of exotic or notifiable diseases needs to be reviewed and ruled out.

Dietary manipulation should best be considered as a means to complement other methods of disease control. Most dietary 'treatments' are more appropriately used prophylactically rather than therapeutically. This is because it generally takes days or weeks for the microenvironment in the gastrointestinal tract to stabilise after a dietary change and, in many cases, an individual animal may have already recovered before any diet-related changes in the microenvironment have had an effect.

Infections can occur variously in different parts of the gastrointestinal tract, ranging from the stomach through to the colon, and dietary components can become physically modified, digested and absorbed as they progress along the tract. The interactions between dietary components, the normal microbiota of the tract, the underlying digestive processes and the potential pathogens are enormously complex. Hence, dietary effects are variable, and rarely offer complete protection from enteric disease.

Any dietary change has to be economically viable. In other words, it must not be excessively expensive or impractical for routine use, and must not damage the overall production and profitability of a herd.

POSTWEANING COLIBACILLOSIS

Watery, alkaline (secretory) diarrhoea is frequently seen in piglets within three to 10 days of weaning. The condition is commonly called postweaning colibacillosis (also known as postweaning diarrhoea) to acknowledge the important role of certain serotypes of (usually) enterotoxigenic haemolytic *Escherichia coli* in its aetiology. There are numerous risk factors associated with the development of this condition, but it is generally accepted that the activity of these bacteria, which proliferate in the small intestine after weaning, is central in causing the diarrhoea.

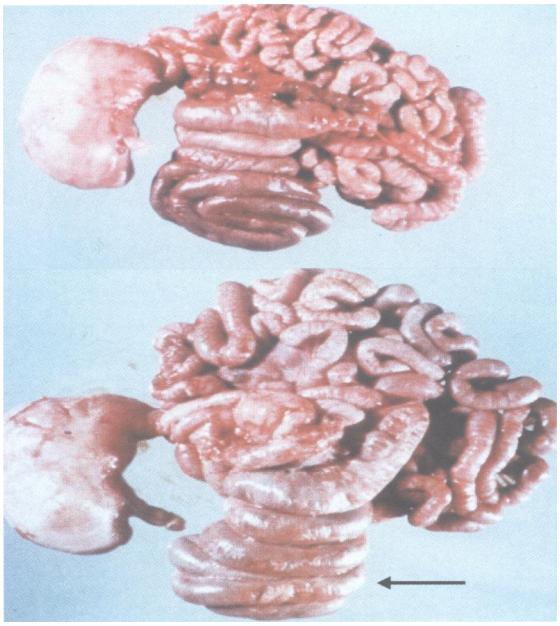
It is very evident that postweaning colibacillosis is subject to some important dietary influences. For example, the condition can be brought under control by imposing severe dietary restrictions over the period of greatest susceptibility. Unfortunately, although this may stop the diarrhoea, it also has an unacceptable impact on growth rate. Generally, it is considered advisable to encourage early and sustained consumption of weaner diets to promote weight gain.

Certain dietary components are considered to impose an increased risk of developing postweaning colibacillosis. These include:

- Unrefined soya bean meal containing antinutritional factors, such as trypsin inhibitors, and potential allergens;
- Certain wheats and other cereals (eg, rye) that are rich in soluble non-starch polysaccharides;
- Poor quality animal proteins, particularly where these have a high acid-binding capacity in the stomach (eg, poor quality fishmeal).

High levels of protein (eg, >24 per cent) are generally thought to be detrimental immediately after weaning, although the dietary balance between protein level and the amount of dietary fibre could also influence the extent and severity of diarrhoea.

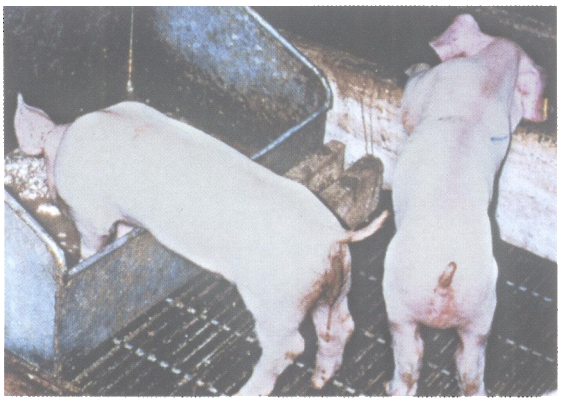
Highly digestible diets are considered to offer protection from postweaning colibacillosis. An example is a diet based on cooked white rice (McDonald and others 2001). Although relatively expensive, the diet is only fed for a short period after weaning and, hence, probably has an acceptable cost/benefit ratio. Cooking rice on a large scale is not practical, but the use of extruded/expanded rice is an effective substitute. Interestingly, there is also good evidence that the addition of insoluble non-starch polysaccharides ('fibre'), such as those found in sugar beet pulp and barley, can reduce the occurrence of postweaning colibacillosis by promoting the rapid development of the fermentative function of the large intestine, which in itself may provide health benefits.



Effect of weaning on the development of the gastrointestinal tract. Gastrointestinal tract from (top) a 26-day-old unweaned piglet and (bottom) from a 26-day-old littermate of the same weight, five days after being weaned. The tract has increased in size after weaning, particularly the caecum and spiral colon (arrow) where fermentation occurs

COMMON BACTERIAL CAUSES OF ENTERITIS IN GROWING PIGS REPORTED TO BE INFLUENCED BY DIET

Disease	*Agent*	*Usual age goup*
Postweaning colibacillosis	Enterotoxigenic *Escherichia coli*	3 to 10 days postweaning
Proliferative enteropathy	*Lawsonia intracellularis*	Weaners/growers
Porcine intestinal spirochaetosis	*Brachyspira pilosicoli*	Weaners/growers
Swine dysentery	*Brachyspira hyodysenteriae*	Weaners/growers/finishers
Salmonellosis	*Salmonella enterica*	Weaners/growers/finishers
Stomach ulcers	*Helicobacter* species?	Growers/finishers



Recently weaned piglets with postweaning diarrhoea associated with proliferation of enterotoxigenic *Escherichia coli* in the small intestine

PROLIFERATIVE ENTEROPATHY

Proliferative enteropathy is widespread, particularly in weaner and grower pigs. The condition is associated with colonisation by the intracellular bacterium *Lawsonia intracellularis*, particularly involving enterocytes in the crypts of the ileum. Subsequent changes in enterocyte differentiation, with elongation of the crypts, are responsible for reduced absorption. Groups of affected pigs may show porridge-like 'osmotic' diarrhoea, and poor and uneven growth rates. If infection occurs in replacement gilts or young boars, it can result in extensive, and often fatal, haemorrhage into the intestinal lumen.

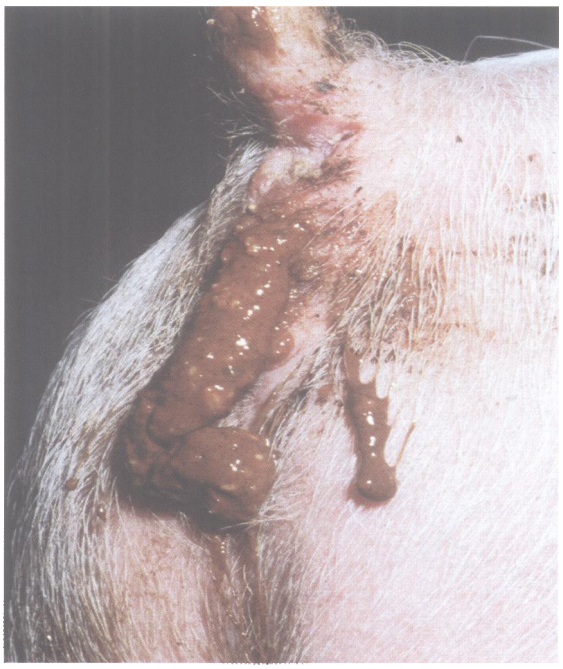
There is some suggestion that diet may affect proliferative enteropathy and, hence, the condition may

respond to dietary changes. For example, in a survey to identify risk factors associated with enteropathogenic bacteria in Danish finishing herds, the use of home-mixed feed and/or non-pelleted diets was linked to a reduced risk of infection with *L intracellularis* (Stege and others 2001). Whether protection is due to specific dietary ingredients that are more commonly found in home-mixed diets than in commercial pelleted diets is unclear. Interestingly, however, pelleted diets confer a greater risk for a number of enteric infections (see below). The high temperatures used for pelleting may have physical effects on the ingredients, causing gelatinisation and retrograding of starch and/or solubilisation of the non-starch polysaccharides, the components of which, in turn, act as a substrate for fermentation, causing alterations in the intestinal microenvironment. Nevertheless, pelleted diets are widely used because they generally cause less food wastage and promote greater growth rates; any recommendation to move to a meal-based diet should therefore be made carefully.

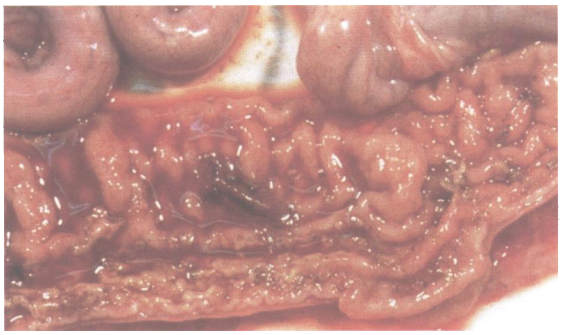
PORCINE INTESTINAL SPIROCHAETOSIS

Porcine intestinal spirochaetosis is a condition of weaner, grower and, sometimes, finisher pigs. It is associated with colonisation of the large intestine with *Brachyspira pilosicoli*, an anaerobic, weakly haemolytic intestinal spirochaete. Infection is common in herds in many countries and, often, multiple strains of the spirochaete may be present in a herd. Colonisation may result in a mild patchy colitis, usually with a mild mucoid diarrhoea.

In an experimental study involving infection of weaner pigs, it was shown that animals fed a highly digestible diet based on cooked white rice were colonised by *B pilosicoli* for a significantly shorter period of time compared with pigs fed a more standard wheat-based, non-pelleted diet (Hampson and others 2000). It was suspected that this protection might be due to the less viscous intestinal contents of those pigs fed the rice. This idea was supported by a subsequent study where the addition of viscous-forming carboxymethylcellulose to a rice-based diet appeared to reinstate the susceptibility of the pigs to infection with the spirochaete (Hopwood and others 2002). The relative protective effects of a rice-based diet on colonisation by *B pilosicoli* have recently been confirmed by Lindecrona and others (2004). Interestingly, in this study, the use of a pelleted diet based on wheat and barley also led to more colonisation and more clinical signs than the same diet in a non-pelleted form.



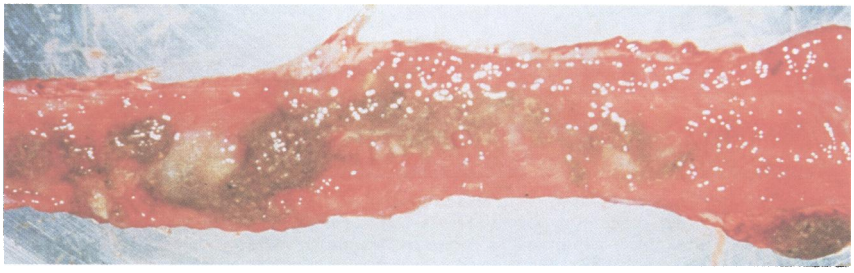
A grower pig showing moist mucoid diarrhoea containing undigested grains. The large intestine of this pig was found to be colonised with the intestinal spirochaete *Brachyspira pilosicoli*



Luminal surface of the ileum of a pig with proliferative enteropathy due to *Lawsonia intracellularis* infection. Note the thickening of the mucosa and haemorrhage

SWINE DYSENTERY

Swine dysentery is a severe mucohaemorrhagic diarrhoea caused by colonisation of the large intestine by the strongly haemolytic intestinal spirochaete *Brachyspira hyodysenteriae*. The disease is still common in a number of important pig-rearing countries, where isolates with reduced susceptibility to various antimicrobial agents are now frequently being found. The disease is complex and its expression is known to be influenced by diet. Several studies have indicated that the incidence of swine dysentery can be reduced by feeding highly digestible diets that result in less fermentation in the large intestine (eg, Pluske and others 1998). Diets based on cooked white rice, which are low in soluble non-starch polysaccharides, were found to be particularly protective. More recently, others have not been able to reproduce these protective results with rice (Lindecrona and others 2003), although a possible explanation could be that different rice varieties and physical processing were used in the different experiments. Interestingly, Lindecrona and others (2003) did find that feeding a fermented liquid diet reduced the incidence of swine dysentery in experimentally infected pigs, and it is possible that the four to five days of pre-fermentation reduced the level of starch and non-starch polysaccharides in the diet, and thus the amount of fermentable substrate reaching the large intestine.



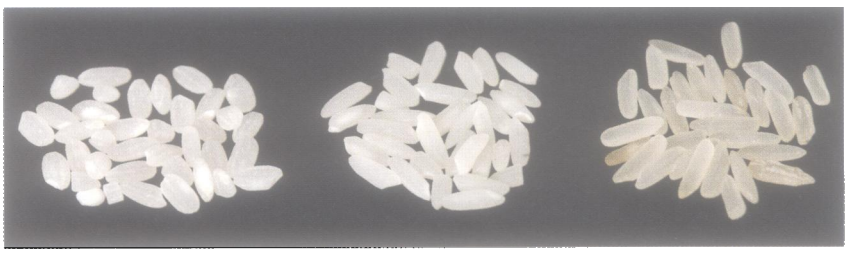
Luminal surface of the colon of a pig with swine dysentery due to *Brachyspira hyodysenteriae* infection. Note the extensive haemorrhagic colitis with large plugs of mucus overlying the mucosa

The pigs in the foreground are being fed on a standard wheat-based diet. The pigs in the background are being fed a highly digestible diet based on cooked white rice. Note the comparative scarcity of faeces on the cage floor of the pigs fed rice compared with those fed wheat

Based on the available evidence so far, and where it is practical and economically viable, the feeding of diets based on cereals with lower levels of soluble non-starch polysaccharides (eg, maize) may provide some protection against swine dysentery. The use of pre-fermented diets should also be considered.

SALMONELLOSIS

Severe salmonellosis (enteritis/septicaemia) in pigs is not particularly common, although subclinical intestinal carriage and subsequent zoonotic spread of salmonellae has been an important issue for the pig industry. Attempts to control salmonellosis in pigs are mainly driven by issues of food safety. Problems in this regard include the fact that the numerous non-host adapted serovars (eg, *Salmonella enterica* serovar Typhimurium) can be acquired by pigs through contaminated diets or through contact with faeces from other animals such as rodents or wild birds. A number of studies have looked for evidence of dietary influences on *Salmonella* species carriage in pigs, and there is general consensus that feeding pelleted diets increases the risk of infection, while feeding liquid by-products or fermented liquid feed reduces the risk (Brooks and others 2001). Furthermore, it has been demonstrated in vitro that pigs fed a coarsely ground meal-based diet have an increased death rate of *Salmonella* species in their stomach contents compared with pigs fed fine non-pelleted, coarse-pelleted or fine-pelleted diets (Mikkelsen and Jensen 2003).



(left to right) Medium grain, long grain and parboiled polished rice. There are many different types of rice containing varying levels and forms of starch (eg, resistant starch). The availability and, hence, biological activities of this starch within the gastrointestinal tract can be further modified by the type of processing or cooking used before it is eaten

In the light of these dietary influences on *Salmonella* species carriage, the composition and form of the diet should be considered when devising control programmes for salmonellosis in pigs.

STOMACH ULCERS

Erosions and/or ulcers of the pars oesophagea of the stomach are often observed in pigs at slaughter, and associated perforations of the stomach and consequent internal bleeding are important causes of death in finishers and sows in some piggeries. Lesions may also be observed in weaners and, presumably, can be present throughout the grower phase of production. It is believed that the lesions have an impact on pig growth and productivity, and are a potential welfare problem.

Some studies have suggested a link between the presence of bacteria (*Helicobacter* species) and ulceration in pigs, although the associated lesions in colonised animals are more usually found in the fundus of the stomach rather than in the pars oesophagea.

Whether or not there is bacterial involvement in the aetiology of stomach ulceration in pigs, numerous studies have found a positive association between ulcerative lesions in the pars oesophagea and feeding finely ground or pelleted diets (eg, Robertson and others 2002). The basis for this association is not clear, but may be due to disruptions to the normal pH gradient in the stomach. Automated feeding also increases the risk of stomach ulcers being present. Unfortunately, the use of pelleted feed and automated feeders is integral to most modern pig producing units, so it is not necessarily practical to alter these systems. Furthermore, as mentioned earlier, pelleted feeds result in improved feed conversion efficiency, so any recommendation to change to another form of diet should be considered carefully.

Opened stomach from (top) a normal grower pig and (bottom) one with erosions of the pars oesophagea (arrow)

A FAVOURABLE SOLUTION?

It has been shown that the occurrence of a number of important enteric bacterial infections in pigs can be influenced by the diet consumed. Producers are only likely to agree to implement major dietary changes to overcome these problems if the diseases are particularly costly and intractable. Changing diets may solve some problems, but create others. A number of infections can be limited by switching from pelleted diets to those based on meal, but this can also impose penalties in terms of feed conversion efficiency. Similarly, a switch to fermented liquid diets requires considerable financial and technical input, and may not be practical in many cases. Nevertheless, at a time when antimicrobial resistance is increasing and the availability of antimicrobials is diminishing, the existence of potential dietary solutions to some enteric infections should not be overlooked.

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Notes