Diseases and conditions in pigs, horses and chickens arising from incomplete digestion and absorption of carbohydrates

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Summary
In this review we have described a number of economically important diseases which arise from the incomplete digestion and absorption of carbohydrate in the small intestine. It is evident that processes which occur in the large intestine as a consequence of microbial digestion predispose some animals to certain diseases and conditions. In this review, we have referred to swine dysentery in pigs, laminitis in equines, and wet litter in poultry. Furthermore it is likely that some diseases, such as those caused by Escherichia coli in pigs, are associated with the presence of dietary fibre in the small intestine. The expression of diseases and conditions originating in both the large and small intestine can be controlled by diet (e.g. feeding a diet of low fermentability/high digestibility to pigs to control swine dysentery and E. coli), by the use of antibiotics (e.g. feeding virginiamycin in the form of FounderguardTM to horses), and by the use of enzymes (e.g. wet litter in broiler chickens). The control of acidic conditions in the large intestine and (or) increased digestion of carbohydrates in the small intestine appears to be associated with a reduced incidence of these diseases and conditions.

Introduction
The major components of animal feedstuffs are starch, protein, fat and ‘fibre’. In classical monogastric nutrition, starch, protein and fat have traditionally been considered as ‘nutrients’ whereas ‘fibre’ was considered to contribute little to the nutritional needs of the animal (Annisson and Choct, 1994). From research conducted in the 1970’s and 1980’s, mostly in humans, it has become recognised that some components of ‘dietary fibre’, which includes non-starch polysaccharides (NSP) and resistant starch (i.e. that fraction of starch which escapes digestion in the small intestine), exerted a greater variety of physiological, metabolic, nutritional and health-modulating effects than previously thought. This has been particularly well characterised, for example, in the field of human nutrition where the intake of dietary fibre has been implicated in the etiology of a number of important diseases including coronary heart disease, diabetes, hypercholesterolaemia, appendicitis, haemorrhoids, diverticulus, obesity, osteoporosis, and cancer of the large bowel (Burkitt and Trowell, 1975; Baghurst et al. 1996).

Relatively less interest, however, has been paid to the consequences of the physiochemical properties of dietary fibre in animals and how they can influence animal production and health. In this review, we wish to focus specifically on some diseases and conditions arising from carbohydrates which are incompletely digested and absorbed in the small intestine, and which then enter the large intestine where they are available for microbial fermentation to short-chain fatty acids (SCFA), principally acetate, propionate, and butyrate. We wish to focus specifically on pigs, horses and chickens, and have deliberately avoided a discussion on the classification of carbohydrates and dietary fibre per se.

Nevertheless, it is important to define ‘dietary fibre’ for the purposes of this review. It comprises the non-starch polysaccharides found in plant cell walls, resistant starch, and the a-galactosides (oligosaccharides) of the raffinose series (raffose, stachyose and verbascose) present mainly in legumes. The role of these constituents of feedstuffs in some diseases and conditions in pigs, horses and chickens will be discussed, with the major emphasis being on pigs and the interactions that occur between diet and two important diseases, swine dysentery and post-weaning diarrhoea, or PWD.
Diseases of the intestines in pigs

Interactions between dietary fibre and swine dysentery

Swine dysentery is a mucohaemorrhagic colitis which results from infection by the anaerobic spirochaetal bacterium *Serpulina hyodysenteriae* (Harris and Lysons, 1992). Swine dysentery has been identified as the most economically significant disease of pigs in Australia, costing in excess of $100 per sow per year in affected herds (Cutler and Gardner, 1988). The control of swine dysentery in infected herds has relied heavily on the use of antimicrobials and (or) resistant starch. This approach is expensive, resistant strains of *S. hyodysenteriae* have appeared in Australia, and the public is becoming increasingly concerned about the high level of usage of antimicrobials in meat production (i.e. antibiotic residues). Alternatively, the disease can be eradicated from piggeries by the combined use of medication, destocking, and disinfecting. However, this strategy is not always successful, it is expensive and, even where it has worked, there remains the risk of reintroduction of infection.

Despite there being evidence that around 33% of pig herds in Western Australia are infected with the causative agent of swine dysentery, we know that disease occurs much less commonly than this (Hampson et al. 1992; Mhoma et al. 1992). The reasons for this are not clear, but recent research at Murdoch University has established that the clinical expression of swine dysentery in growing pigs (25 – 40 kg) following experimental infection with *S. hyodysenteriae* is reduced by feeding diets low in rapidly-fermentable dietary fibre, i.e. soluble NSP, oligosaccharides, and (or) resistant starch (Pluske et al. 1996b; Siba et al. 1996). We have shown that diets containing less than about 1% of soluble NSP (= 5% total NSP) showed a reduced incidence of swine dysentery (Figure 2).

There was no correlation between the amount of insoluble NSP nor the measured level of resistant starch in diets and the incidence of disease. However, feeding some diets containing less than ≈1% of soluble NSP (e.g., a diet based on steam-flaked sorghum) was still associated with a high incidence of disease. This was found to relate to higher levels of resistant starch in these diets which appeared to exacerbate the clinical expression of swine dysentery (Pluske et al. 1996b).

We have further examined the role of these fractions of dietary fibre in the aetiology of swine dysentery in two recent experiments by adding them to a diet which completely prevents the development of swine dysentery, i.e. cooked white rice with a supplement based on animal protein sources (Pluske et al. 1996b; Siba et al. 1996). In this way, their precise contribution to swine dysentery could be ascertained.

In the first experiment (Pluske et al. 1996a), a source of soluble-NSP (guar gum, 10%), insoluble-NSP (oaten chaff, 7%), resistant starch (20%, fed as Novelose™, a high-amylose maize starch, and equivalent to 7.6% resistant starch), and a combination of soluble-NSP and resistant starch (5% and 10%, respectively), was added to a diet containing cooked white rice and animal protein. A control diet containing rice and the animal protein supplement was also fed. *Spirochaetes* only colonised the colonic epithelium of pigs fed resistant starch, guar gum, and resistant starch plus guar gum, however the clinical expression of swine dysentery only occurred when pigs were fed guar gum or resistant starch plus guar gum. Expression of swine dysentery was associated with a lower pH in the colon and a heavier large intestine when expressed as a percentage of empty bodyweight (Table 1). These data confirm our previous studies, and implicate the presence of soluble NSP in the clinical expression of swine dysentery. However, the contribution of resistant starch to swine dysentery could not be confirmed despite *Spirochaetes* colonising the epithelium of the large intestine.

In the second study (Pluske et al. unpubl. data), we wished to examine further the role of resistant starch in expression of the disease. Diets containing either 13%, 20% or 27% Novelose™ (equivalent to 5.1, 7.6 and 10.4 % resistant starch) were included in a basal diet containing cooked white rice and an animal protein supplement, and were fed to growing pigs which were then infected with a virulent strain of *S. hyodysenteriae*. In this case, swine dysentery was observed in pigs fed all three levels of resistant starch.

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**Figure 1** The incidence of swine dysentery (%) in pigs fed a range of diets following experimental infection with *S. hyodysenteriae*. From Pluske et al. (1996b).
Incomplete digestion and absorption of carbohydrates

Figure 2  The relationship between the incidence of swine dysentery (%) (y-axis) and (a) soluble NSP concentration (x-axis) \( (y = 9.52 + 56.98x - 8.47x^2, R^2 = 0.561, P = 0.016) \), and (b) total NSP concentration (x-axis) \( (y = -57.97 + 26.85x - 1.10x^2, R^2 = 0.712, P = 0.002) \), in pigs fed different diets. (From Pluske et al. 1996b).

Table 1  Effects of different dietary fibre sources on pH, gut characteristics, colonisation, and the incidence of swine dysentery following experimental infection with S. hyodysenteriae. (From Pluske et al. 1996a).

<table>
<thead>
<tr>
<th>Diet</th>
<th>pH</th>
<th>Empty wt. of large intestine (% bodyweight)</th>
<th>Colonisation</th>
<th>Incidence of swine dysentery (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Caecum</td>
<td>Colon 1†</td>
<td>Colon 2‡</td>
<td></td>
</tr>
<tr>
<td>Control†</td>
<td>6.4</td>
<td>6.6</td>
<td>7.2</td>
<td>1.2</td>
</tr>
<tr>
<td>Oaten chaff</td>
<td>6.8</td>
<td>6.7</td>
<td>7.1</td>
<td>1.7</td>
</tr>
<tr>
<td>RS‡</td>
<td>5.6</td>
<td>6.1</td>
<td>6.5</td>
<td>1.8</td>
</tr>
<tr>
<td>Guar gum</td>
<td>5.8</td>
<td>5.8</td>
<td>5.9</td>
<td>2.0</td>
</tr>
<tr>
<td>Guar gum + RS</td>
<td>5.6</td>
<td>5.8</td>
<td>6.3</td>
<td>2.5</td>
</tr>
<tr>
<td>SED</td>
<td>0.13</td>
<td>0.15</td>
<td>0.19</td>
<td>0.27</td>
</tr>
</tbody>
</table>

†Control = rice + animal protein supplement.
‡Colon 1 = proximal colon; Colon 2 = distal colon.
§S. hyodysenteriae isolated from rectal swabs.
RS: resistant starch.
\*Within columns, values not followed by the same superscript are significantly different \( (P < 0.001) \)
Our data implicate the soluble NSP and resistant starch in the clinical expression of swine dysentery. Also, given the data of Siba et al. (1996), the oligosaccharides contained in legumes such as lupins most likely play a role in the aetiology of swine dysentery. However, ‘classical’ indices of the rate and extent of hind-gut fermentation have not necessarily supported our findings (Pluske et al., 1996b). To date, our research has not unravelled the precise mechanism(s) whereby some diets are more protective than others, and in particular why a diet based on cooked white rice completely prevents the disease. Certainly, the diet based on cooked white rice is fermented less than other cereals in the large gut commensurate with a lighter large intestine, but this did not happen in diets based on stream-flaked maize and steam-flaked sorghum. As suggested by Siba et al. (1996), the protective effect of some diets may be an indirect effect of mucin production, factors affecting the mobility of S. hyodysenteriae to the mucosal lining of the colonic epithelium, factors influencing the ability of S. hyodysenteriae to express haemolysins and/or lipopolysaccharides and effect inflammation of the epithelium, and/or the dry matter content of colonic contents that may inhibit spirochaetal survival in the large intestine. Some support for these notions comes from the significantly drier faeces of pigs fed these protective diets (P.M. Siba, unpubl. data).

**Microbiological interactions and swine dysentery**

Little research has been conducted examining possible interactions between S. hyodysenteriae and other microflora of the large intestine with the type of diet fed, and how this could then predispose pigs to swine dysentery. By definition, swine dysentery is a mixed synergistic infection (Meyer et al. 1974; 1975) and infective combination of resident microorganisms of low pathogenic potential, interacting with the spirochaete and with each other to represent an inherent pathogenic potential. Furthermore, it is well recognised that different components of the microflora of the large intestine can either enhance (Whipp et al., 1979) or reduce (Suenaga and Yamazaki, 1986) colonisation by S. hyodysenteriae, and subsequently can affect the incidence of swine dysentery. It is possible, therefore, that the protective effects of diets operated through some specific or unspecific alteration(s) in the resident microflora. In this regard, we examined the microflora isolated from colonic content and colonic walls from healthy pigs fed different diets. The study revealed that specific diet ingredients can affect both the number and composition of the microflora of the large intestine (Durmic et al., unpublished data). While diets containing resistant starch increased the overall number of bacteria, diets containing soluble NSP encouraged the growth of bacteria which can act synergistically with S. hyodysenteriae. Both events led to the development of swine dysentery following experimental infection. It remains uncertain whether the protection against swine dysentery is attributable to the ratio of Gram-negative and Gram-positive bacteria, since the prevalence of Gram-positive bacteria was observed both in the absence and in the presence of rapidly-fermented dietary fibre in the large intestine.

**The use of enzymes to reduce the clinical expression of swine dysentery**

Based on our previous data, there is good reason to suspect that addition of exogenous enzymes to diets may ameliorate the incidence of swine dysentery by hydrolysing glycosidic linkages of soluble NSP prior to their passage into the large intestine. We have recently investigated this hypothesis in a study comprising a 2 x 2 factorial arrangement of treatments where wheat was fed to pigs either in extruded form (to reduce the contribution of resistant starch to the expression of swine dysentery) or hammer-milled form, and where an exogenous xylanase was added or not added to the diet (Pethick et al., unpublished data). Pigs were infected with a virulent strain of S. hyodysenteriae at \( m = 25 \) kg and monitored for expression of swine dysentery.

Contrary to what we expected, the addition of a xylanase preparation to the diet failed to reduce the incidence of swine dysentery. We observed a significant main effect, however, of enzyme addition on digesta pH but only in the distal part of the colon, such that pigs fed the xylanase preparation had a higher pH than pigs not fed enzyme (6.68 vs. 6.35, \( P = 0.017 \)). These data suggest that the enzyme is having some effect on fermentation, but that it is occurring towards the end of the large bowel. This may allow colonisation by S. hyodysenteriae in the anterior parts of the hind gut, with subsequent expression of swine dysentery. Further investigations with, for example, enzymes of greater specificity and pre-incubation of diets, may cause a greater reduction in hind-gut fermentation with a subsequent decrease in the incidence of disease.

**Interactions between non-starch polysaccharides and Escherichia coli**

One of the keys to improving overall pig productivity is to ameliorate the post-weaning ‘growth check’. There is a considerable body of evidence to show that feeding pigs a ‘highly-digestible’ diet immediately after weaning improves performance, but this is sometimes associated with the overgrowth of pathogenic strains of bacteria within the gastrointestinal tract. Post-weaning diarrhoea (PWD), or colibacillosis, results from the proliferation of certain serotypes of strongly \( \beta \)-haemolytic Escherichia coli in the proximal small intestine. This remains an economically important disease world-wide. The condition is complex and multi-factorial, with both the level of nutrient intake and diet composition having been found to influence the pathogenesis of the disease (Ball and Aheme, 1987; Hampson, 1994; Aurnaire et al., 1995; Rantzer et al., 1996).
Incomplete digestion and absorption of carbohydrates

Addition of dietary fibre has been suggested as a means of ameliorating PWD by accelerating the development of the large intestine (Boudouin et al. 1988). This is the major absorptive organ for water and electrolytes in the newly weaned pig, especially under conditions of disease in the small intestine (Argenzio et al. 1984). Evidence to support this notion, however, is equivocal (van Beers-Schreurs, 1996). Difficulties arise with this work because many studies are retrospective, there has been a lack of controlled infection trials, and most studies have not clearly defined ‘fibre’. In those that have, ‘crude fibre’ is generally given (Batterham 1990) which, in the light of the current understanding and interpretation of dietary fibre (Mugford 1993), is unsatisfactory.

Given this, we have completed some preliminary work to examine the role of soluble NSP on the pathogenesis of haemolytic *E. coli* in weaner pigs (McDonald et al. 1996). Our initial hypothesis was that when pigs are orally challenged with *E. coli* to induce secretory diarrhoea, provision of a source of rapidly-fermentable NSP would promote hind-gut growth and hence promote absorption of water and electrolytes from the large intestine. Enhanced growth of the large bowel would act as a ‘salvage’ mechanism and help to prevent the dehydration and emaciation commonly seen with PWD. We fed a pre-gelatinised diet based on cooked white rice and animal protein to weaner pigs and compared it to the same diet after inclusion of a source of soluble NSP (guar gum added at 10%). Both diets were fed either *ad libitum* or at 1.5 maintenance, and all pigs were orally challenged with 10⁶ enterotoxigenic haemolytic *E. coli* for three consecutive days commencing 48 hours after weaning.

In the first seven days after weaning, McDonald et al. (1996) showed that the presence of haemolytic *E. coli* in the small intestine was influenced by the type of diet fed. Pigs fed soluble NSP had more (*P = 0.08*) colony-forming units (CFU) of haemolytic *E. coli* in the anterior small intestine, and more pigs were colonised with haemolytic *E. coli* (*P < 0.05*), compared to pigs fed the diet containing only rice and animal protein (Table 2). In addition, pigs fed a source of soluble NSP had:

- more acidic contents in their caecum and colon (*P < 0.01*),
- a heavier small (*P < 0.05*) and large (*P < 0.001*) intestine, and
- a lower rate of empty body-weight (EBW) gain (*P < 0.01*) (Table 2).

Similar results for microbiology and effects on gut weights and EBW were found when a diet based on

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**Table 2** Gut pH, gut weights, and haemolytic *E. coli* in the small intestine of weaner pigs fed different diets for seven days after weaning (Values are mean ± SEM, in parentheses). (Adapted from McDonald 1996, and McDonald et al. 1996).

<table>
<thead>
<tr>
<th>Diet¹</th>
<th>Rice</th>
<th>Rice and Guar</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High</td>
<td>Low</td>
<td>High and Low</td>
</tr>
<tr>
<td>pH</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>caecum</td>
<td>6.2 (0.13)</td>
<td>6.0 (0.09)</td>
<td>5.2 (0.05)</td>
</tr>
<tr>
<td>distal colon</td>
<td>6.5 (0.10)</td>
<td>6.5 (0.13)</td>
<td>5.6 (0.18)</td>
</tr>
<tr>
<td>Small intestine, % EBW</td>
<td>4.8 (0.40)</td>
<td>4.6 (0.32)</td>
<td>5.7 (0.26)</td>
</tr>
<tr>
<td>Large intestine, % EBW</td>
<td>1.1 (0.06)</td>
<td>1.2 (0.05)</td>
<td>1.7 (0.11)</td>
</tr>
<tr>
<td>EBW gain over 7 days (g)</td>
<td>828 (0.2)</td>
<td>742 (0.2)</td>
<td>217 (0.1)</td>
</tr>
<tr>
<td>CFU of <em>E. coli</em> per g²</td>
<td>1.3 x 10⁴</td>
<td>8.0 x 10⁹</td>
<td>*</td>
</tr>
<tr>
<td>% haemolytic <em>E. coli</em> in small intestine³</td>
<td>3.6 (1.40)</td>
<td>26.8 (8.93)</td>
<td>**</td>
</tr>
</tbody>
</table>

¹ Both diets contained cooked white rice plus a “supplement” consisting mainly of animal protein sources; no anti-microbials were added to any diets.

² See text for details.

³ CFU of haemolytic *E. coli* per gram of mucosal scraping in the small intestine.

³ Mean % β-haemolytic *E. coli*, expressed as a percentage of total bacterial population in small intestine.

* P < 0.1; ** P < 0.05; *** P < 0.01; **** P < 0.001; NS: not significant (*P > 0.1*).
increase and when a cereal containing a high proportion of starch (e.g., maize) is fed (Rowe et al. 1994b) and when a cereal containing a high proportion of starch (i.e., maize) is fed (Rowe et al. 1994a). This is associated with decreased concentrations of circulating D-lactate and a higher faecal pH indicative of reduced hind-gut fermentation. It appears, however, that Founderguard™ is only effective in controlling acidosis and laminitis when given prior to a carbohydrate overload (Rowe et al. 1995a).

Furthermore, horses fed high amounts of cereal (72% wheat) and given Founderguard™ demonstrated a reduced incidence of adverse behaviours generally associated with feeding high-grain diets (Figure 3), and this was linked to a decrease in the acidity of the faeces (Rowe et al. 1995b). These observations agree with those of Willard et al. (1976) who reported that behaviour such as wood chewing and the consumption of bedding was related to the concentration of D-lactate in faecal contents, and provide convincing evidence of a direct link between fermentative events in the large intestine and both health and behaviour. This is a fascinating discovery, and corollaries could be drawn between lameness in horses with other monogastric animals which are primarily kept indoors on solid surfaces, and where lameness is a common cause of culling (e.g., pigs).

**Wet litter in poultry production**

**Effects of NSP on nutrient digestion in poultry**

It is well established that soluble NSP depress the performance of, and impair the nutrient digestibility by, poultry. For example, the high amounts of soluble arabinoxylans in rye and β-glucans in barley are responsible for the cereals’ poor nutritive value to poultry (Antoniou et al. 1981; Campbell et al. 1989; Chocot and Anisson, 1992; Chocot et al. 1996), and the concentrations of soluble pentosans in Australian wheats are inversely correlated with their AME in broiler chickens (Annison, 1991). It is believed that the viscous
nature of these cell wall polysaccharides is the primary cause for their anti-nutritive effect in poultry, because the increased bulk and viscosity of the intestinal contents decrease the rate of utilisation of substrates and digestive enzymes and hinder their effective interaction at the mucosal surface (Choct et al. 1996). Interaction of soluble NSP with the gut microflora is also implicated in reduced nutrient utilisation (Choct et al. 1996). Similarly, it is well established that the adverse effects of soluble NSP on performance and digestion can be ameliorated by the addition of exogenous xylanases (Campbell and Bedford, 1992, and Bedford and Morgan, 1996).

A condition in broiler chickens believed to arise from both the physical (viscosity) effect of NSP and the interaction between the gut microflora is ‘wet litter’, or ‘sticky droppings’. Apart from the obvious concern of reduced production, wet litter also poses a management problem, particularly when the humidity is high and external temperatures are low (Marquardt et al. 1996). In layers, however, the story appears to be much more complicated. In addition to NSP, dietary electrolyte balance, renal problems, mineral x strain interactions, disease conditions etc., may all be involved (M. Choct, pers. comm.).

Are intestinal spirochaetes a contributing factor to wet litter?

Of recent interest is the finding from Murdoch University that intestinal spirochaetes of the same genus that cause swine dysentery in pigs were found to colonise 16/30 (53.3%) broiler breeder flocks and 13/37 (35.1%) layer flocks in Western Australia (Table 3). Intestinal spirochaetes were isolated from 6/12 (50%) layer flocks showing signs of either diarrhoea or reduced production, compared to only 5/23 (21.7%) found to be disease free (P < 0.10 by chi-square test). No data were available from the broiler breeder flocks. When the results for both layer and broiler breeder flocks for which an adequate history was available were combined, intestinal spirochaetes were isolated from 16/25 (64%) of flocks with disease signs, compared to only 7/25 (28%) of flocks which appeared clinically normal (P < 0.02 by chi-square test). These data concur with those from layer flocks in both The Netherlands (Dwars et al. 1989) and the USA (Trampel et al. 1994).

Given the high prevalence of infection with intestinal spirochaetes in WA poultry flocks, it is possible that some of the spirochaetes isolated may have been commensals and not responsible for any disease condition at all (McLaren et al. 1996). This may partly obscure the significance of the relationship between prevalence and signs of wet litter and reduced production. Nevertheless, and given the relationship between NSP and the clinical expression of swine dysentery in pigs discussed previously, this finding in poultry is of interest and warrants further research, especially since McLaren et al. (1996) recovered an isolate resembling the porcine pathogen responsible for swine dysentery from a broiler breeder flock.

### Table 3

Results of the culture of faecal samples from broiler breeder and layer flocks for the presence of intestinal spirochaetes. (From McLaren et al. 1996).

<table>
<thead>
<tr>
<th></th>
<th>No. of flocks tested</th>
<th>Number of flocks culture-positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broiler breeder</td>
<td>30</td>
<td>16 (53.3%)</td>
</tr>
<tr>
<td>Layer</td>
<td>37</td>
<td>13 (35.1%)</td>
</tr>
<tr>
<td>Total</td>
<td>67</td>
<td>29 (43.3%)</td>
</tr>
</tbody>
</table>

Conclusion

In this review, we have described several important diseases and conditions of pigs, horses and chickens in relation to the incomplete digestion and absorption of carbohydrate in the small intestine. It is evident that carbohydrate passing into the large intestine can, as a consequence of microbial fermentation, compromise both the health and productivity of some animals. Furthermore, indigestible carbohydrate remaining in the small intestine may be linked to disease, as evidenced by the increased proliferation and colonisation of haemolytic *E. coli* which occurred in the small intestine when a diet containing a source of soluble NSP was fed to newly-weaned pigs which were infected with *E. coli*. Antibiotics have traditionally been used to control enteric infections and conditions, and we have described the use of the non-therapeutic antibiotic *Founderguard*™ to control laminitis in the horse. One may also speculate on the possible role of NSP in the aetiology of this condition. However, the widespread use of in-feed antibiotics in intensive animal production is coming under closer scrutiny. Our finding that the clinical expression of swine dysentery can be abrogated by feeding a lowly-fermentable diet based on cooked white rice and an animal protein supplement, therefore, is both novel and unique. Diets based on steam-flaked sorghum and steam-flaked maize reduced the incidence of swine dysentery in comparison to more traditional diets based on the cereals wheat, barley and oats. The association between ‘wet litter’ and the isolation of *S. hyodysenteriae* in poultry herds may also be linked to the type of diet fed, and it has been shown that the addition of xylanases to poultry diets reduces the incidence of wet litter and increases production. Collectively, these findings suggest there may be
potential for the use of exogenous enzymes in diets based on more traditional feed ingredients to control diseases in pigs, although results from our preliminary study did not show a reduction in the incidence of swine dysentery when a xylanase was added to a wheat-based diet. Alternatively, the severity of diseases such as swine dysentery and post-weaning diarrhoea may be reduced by the judicious selection and (or) processing of feedstuffs which are more digestible in the small intestine.

References


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