

Feed enzymes and betaine in antibiotic free poultry diets

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June 1999 saw a further restriction in the use of prophylactic antibiotic growth promoters (AGPs) used in monogastric diets in the EU. In response feed and poultry producers have focussed on testing alternative products which may relieve some of the negative impacts of this ban on animal production. The following article examines the benefits of using feed enzymes and betaine, and how their mode of action may aid animal performance in AGP-free diets through improving nutrient digestion and the gut environment.

The benefits of feeding antibiotic growth promoters

AGPs have a long history of prophylactic use in animal feed, where they contributed to improved animal performance and health status by targeting and destroying the intestinal microflora. They can achieve this through several mechanisms: interfering with the bacteria's ability to reproduce, damaging cell membranes or disrupting essential cellular activities. Most AGPs target gram-positive organisms that are commonly associated with poor health and reduced animal performance. The economic benefit of feeding prophylactic AGPs can be hard to quantify in individual crops of animals, but analysis of long-term effects has provided evidence of their value in improvement of growth, health and uniformity. The size of response to AGPs is dependent on farm management, exposure to pathogens, environmental stresses and diet. The poorer the conditions the animals are grown under, the greater will be their response to AGPs.

Microflora reduce animal performance by directly competing for nutrients released from the diet in the ileal region of the gut. Bacteria deplete the animal's body reserves by stimulating energy-consuming immune responses to microbial adherence to the gut wall and pathogenic invasion. This has been demonstrated by Muramatsu *et al.* (1994), who compared the growth of bacteria-free and conventional chickens, and saw a reduction in weight gain but an increase in available energy (ME) in the normal birds. Bird weight gain decreased due to increased utilisation of available nutrients by the bacteria and possibly some diversion of energy into immune response. The ME increased because of bacterial fermentation of undigested nutrients. Bacterial fermentation results in the breakdown of substrate to volatile fatty acid components (VFAs), which can be absorbed from the caeca and used as a source of metabolisable energy by the host animal.

The effect of nutrient flow in the ileum on microbial populations.

Like all organisms, microbes need a supply of nutrients and a suitable environment to inhabit (e.g. the caeca). It has been known for many years that the diet fed to the host animal influences microflora development and distribution. Feeding high viscosity cereal grains to broilers, such as wheat and barley, has been shown to result in larger microbial populations in the ileum compared with low viscous corn-based diets (Hofshagen & Kaldhusdal, 1992; Wagner & Thomas, 1987). Similar effects have been observed in broilers fed corn diets supplemented with pectin (to artificially raise viscosity), where rate of digestion was reduced in the pectin diets (Schutte & Langhout, 1999). This was especially pronounced in the

broilers with normal microflora populations. Viscous diets respond particularly well to inclusion of AGPs and other feed additives such as enzymes (Elwinger & Teglof, 1991; Vranjes & Wenk, 1997). A viscous environment slows down digestion processes, and encapsulates nutrients, making them inaccessible to digestive enzymes. Viscous gels are formed in the digesta by the non-starch polysaccharides (NSP) arabinoxylan and betaglucan, which cannot be digested by the animal's own enzymes, and so have been a major target substrate in the development of feed enzymes.

Unlike the field of ruminant nutrition, monogastric nutrition often neglects the impact of diet on intestinal microflora development and the consequential effect on the host animal. VFAs from bacterial fermentation can be a source of energy for the host. Efficiency of digestion is considered to be the key to microflora development. When digestive conditions are optimised by feeding a good quality diet with low stress (disease, heat) levels, the high rate of nutrient digestion and absorption from the intestine limits the amount of nutrients passing to the caecal microflora for fermentation. This restricts the growth and colonisation of microbial populations in the intestine. When digestion is inefficient due to poor raw material quality or gut wall damage, more starch and protein escape ileal digestion, and flow to the lower intestine where they 'feed' the microflora, encouraging proliferation. If there is a change in relative substrate levels (e.g. higher starch and protein compared to fermentable fibre) certain bacterial populations may be encouraged, changing species balance and dominance (Wagner & Thomas, 1987) that may initiate intestinal disorders

(Deloyer *et al*, 1996). This occurs especially in high viscosity, heat-damaged starch and protein diets (Vahjen *et al*, 1998). Poor digestion can induce physiological adaptations in the animal, in an attempt to improve nutrient uptake, e.g. increased pancreatic enzyme secretion, gut length and surface area (Angkanaporn *et al*, 1994; Brenes *et al*, 1993).

Finnfeeds enzymes have been tailor-made for different species (e.g. broilers, pigs) and main dietary components (e.g. corn-soy diets) and are specially formulated to increase nutrient digestibility in the ileum. They reduce digesta viscosity and water holding capacity through breakdown of NSPs (arabinoxylan and betaglucan), starch and protein. Surveys on the most commonly used raw materials in feeds have revealed that variability in broiler performance is of a similar magnitude between wheat, barley and corn, with 0.043, 0.039 and 0.042 standard deviation in FCR respectively (Finnfeeds own data; Leeson *et al*, 1993). This demonstrated that corn-soy diets are not always delivering best animal performance, and have the same scope for improvement as viscous cereal diets. Feed enzymes can improve overall diet digestibility with greater effects on lower versus higher digestible diets, giving corresponding improvements in uniformity of animal performance. As well as improving digestion and absorption in the ileum limiting the nutrients available for microfloral utilisation, fermentation of NSP subunits (liberated by feed enzymes) produces the VFA butyrate, which increases villi growth in pigs thereby improving absorptive surface area (Mosenthin, 1999). There is also increasing evidence that short-chained xylose-oligomers generated through feed enzyme activity may be a specific substrate for certain useful fermentative bacteria (Apajalahti & Bedford, 1998). In corn-soy diets the digestibility of starch and protein has been shown to be highly variable, and an enzyme product (Avizyme 1500) has been specifically developed for corn-soy diets as a result. Excessive drying or heat processing of feeds damages the ileal digestibility of

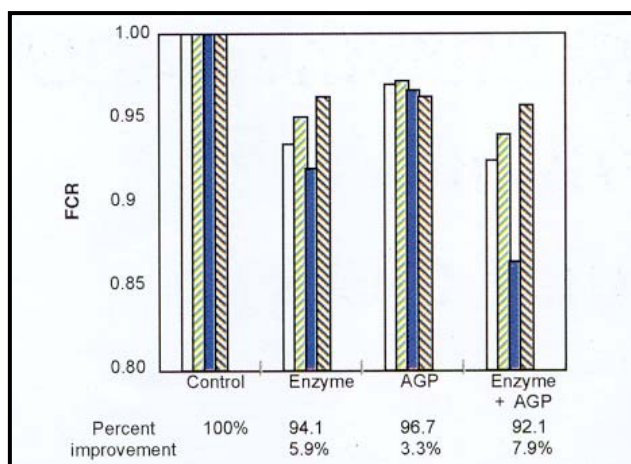


Figure 1: Effect of feed enzyme on FCR in four trials (relative to 100% FCR in control diets) in diets with and without AGP's (Elwinger & Teglof, 1191; Hock *et al*, 1997; Moran & McGinnis, 1968; Vranjes & Wenk, 1996).

starch and protein, providing more nutrients for hindgut bacterial proliferation. In the USA, Finnfeed's corn-soy enzyme has been shown to improve ileal starch digestion by 13.5% and ME by 6%, decreasing the amount of starch digested in the caecum by 5%, and improving ME by nearly 1 % (Coon *et al* 1998).

Recent trials comparing broilers with and without microflora showed that negative effects of poor diet digestibility are only evident when gut bacteria are present (Schutte & Langhout, 1999; Smits & Annon, 1996), so increasing bacterial populations through AGP-removal will reduce growth. In the absence of dietary prophylactic AGPs reliable improvements in animal performance through supplementation with proven enzymes will become more important. In four published trials, use of enzyme alone led to an average 5.9% improvement in FCR while growth promoters gave a 3.3% advantage (figure 1). The combination of the two gave the greatest (7.9%) response, demonstrating that the mode of action of the two products is quite different. As most producers in the EU have been using AGPs and enzymes for many years, the loss of the 3% performance on AGP withdrawal will enhance the economic significance of the 5.9% improvement by enzyme alone.

Betaine - its role in poultry diets

Betaine is used in animal feeds as a source of essential methyl groups for metabolic reactions and as an osmoregulant. Its osmotic function is useful in maintaining gut wall integrity, which has been observed as increased gut strength and reduced dehydration of cells under stressful conditions. When cells dehydrate they cease to function efficiently and become more susceptible to attack from pathogens. This highlights the requirement for preserving the integrity and functioning of the gut wall through increased cell hydration which can be achieved by feeding betaine. In Europe, poultry production is under increasing threat from the disease necrotic enteritis, caused by *Clostridium perfringens*. Challenging birds with the coccidiosis pathogen *E. maxima* has been shown to cause the spontaneous development of necrotic enteritis, as the gut wall damage by coccidiosis facilitates invasion by clostridia (Waldenstedt *et al*, 1998). Removal of AGPs could lead to an increase in necrotic enteritis due to reduced control of *Clostridium perfringens*, making pathogenic invasion more likely, following even low levels of coccidiosis damage. Figure 2 shows how betaine consistently improves the hydration and integrity of the gut wall, manifested as reduced coccidiosis lesion scores and

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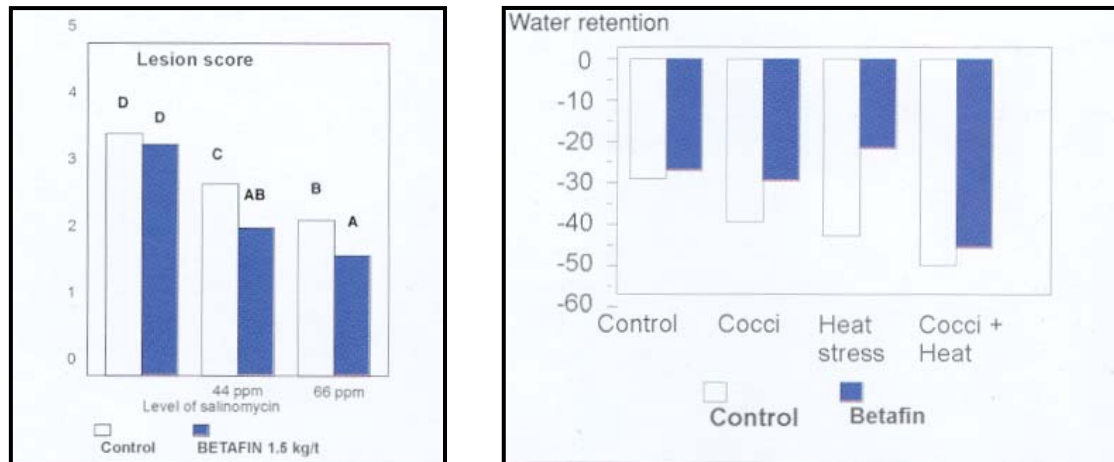


Figure 2: The effect of coccidiostat and Betafin on water retention and gut wall damage in broilers challenged with coccidiosis (ref. Finnfeeds Research Report No B.CC.PC. 92.1).

reduced water loss (calculated as water retention = water intake-(urinary loss + evaporation)). Poor protein digestion results in high caecal nitrogen levels which are currently considered to encourage growth of *Clostridium perfringens*. Under normal gastric conditions, nitrogen is well digested, little enters the caeca and necrotic enteritis is uncommon. However, when viscous diets are fed or coccidiosis damage reduces small intestinal absorptive function, less nitrogen is taken up by the animal, and more enters the caeca resulting in a more frequent occurrence of necrotic enteritis. The removal of AGPs from monogastric diets is anticipated to lead to far greater incidence of necrotic enteritis (Broussard *et al*, 1986; Bywater, 1998; Elwinger *et al*, 1998). By reducing the impact of

coccidiosis infection on gut cells by assisting in cell hydration and function this threat can be lessened.

Overall Conclusions

The ban on AGPs in Europe will cause difficulties for commercial pig and poultry producers through poorer growing efficiency and increased stress and disease levels in their animals. While feed enzymes and betaine cannot replace AGPS, they can promote optimum digestion conditions in the intestine. This creates an environment where nutrients are taken up and used by the host animal, rather than by the resident bacteria. Removal of dietary AGPs are likely to increase variability in broiler performance. Animals fed poorer quality diets are at risk of developing larger intestinal microbial

populations particularly in stressful environments. Increased bacterial numbers will then compete for nutrients and could proliferate to pathogenic levels. Finnfeed's enzymes improve diet digestibility, limiting proliferation of hind gut bacteria by restricting the nutrients available to them, while betaine helps to maintain intestinal cell function by osmoprotection, particularly in the presence of a coccidiostat or under heat stress conditions.

Reference

The references for this article are available from the author and AFMA. It has also been put on the AFMA website at www.landbou.com/afma (www.afma.co.za).

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