

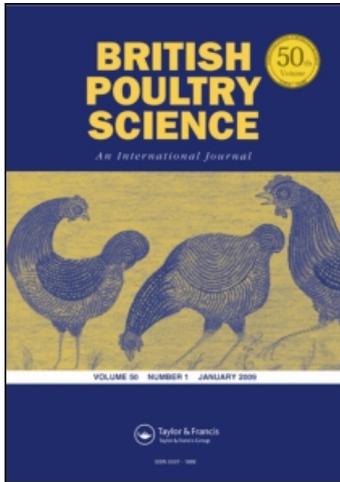
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GORDON MEMORIAL LECTURE

Managing gut health through nutrition

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Abstract 1. To study gut health, a multi-pronged approach is necessary. It should be considered from the point of view of immunology, microbiology and nutrient supply.
2. The impact on gut health often comes from microbial imbalance in the gut, which will be exacerbated if antibiotics are withdrawn from feed.
3. Any gut damage caused by pathogens will lead to poor gut health, which will, in turn, affect nutrient utilisation efficiency. Subclinical forms of infection with no obvious signs of lesions are often financially more devastating than acute, short-term infections. Necrotic enteritis in poultry is one such example.
4. Dietary factors that modulate the immune system and gut microflora should be considered when formulating diets and managing feeding practices.

INTRODUCTION

Formulating diet for its effects on gut health is fast becoming a reality in the monogastric animal industries. This is because maintenance or enhancement of gut health is essential for the welfare and productivity of animals when antibiotics are not allowed in feed. Gut health research has its origin in human health programmes where nutritional interventions, such as probiotics and prebiotics, are used to ameliorate conditions such as inflammatory bowel disease (Guarner *et al.*, 2002; Damaskos and Kolios, 2008) and irritable bowel syndrome (Fooks and Gibson, 2002).

Today, gut health is a major topic for research not only in humans but also in animals. It is now generally conceded that maintenance or enhancement of 'gut health' is far more complex than just the modulation of the gut microflora through probiotics or prebiotics. This is not surprising considering that the gut harbours more than 640 different species of bacteria, contains over 20 different hormones, digests and absorbs the vast majority of nutrients, and accounts for 20% of body energy expenditure. It is also the largest immune organ in the body (Kraehenbuhl

and Neutra, 1992). Thus, anything that affects the health of the gut will undoubtedly influence the animal as a whole and consequently alter its nutrient uptake and requirements. Consequently, 'gut health' is highly complex and encompasses the macro- and micro-structural integrity of the gut, the balance of the microflora and the status of the immune system. Further complexity arises from their interactions and the resulting changes in gene expression, and possibly, endocrine regulation. This, in turn, may affect the way nutrients are partitioned and utilised for organ development, tissue growth and immune system maturation (Kelly and Conway, 2001; Kelly and King, 2001).

Using data generated in poultry studies, this paper will discuss the link between gut health and nutrition in a broad sense, covering gut development, gut microflora and gut microstructure.

FEED CONSTITUENTS AND GUT DEVELOPMENT

Most feed ingredients of plant origin contain considerable amounts of fibre (non-starch polysaccharides, NSP, plus lignin), with the majority

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being insoluble (Bach Knudsen, 1997). Insoluble fibre has traditionally been regarded as an inert nutrient diluent with little or no nutritive value in monogastric animal diets. However, recent findings suggest that this is not true; instead it has various roles in improving gut health, enhancing nutrient digestion and modulating the behaviour of animals (Hartini *et al.*, 2002; Hetland *et al.*, 2003). It is postulated that monogastric animals have a 'fibre requirement' because their gut development requires physical stimulation by hard, solid particles of feed (Hetland *et al.*, 2004b).

A number of recent reports show that chickens consume a considerable amount of their bedding material (Hetland *et al.*, 2004b) and a laying hen fed on a finely ground diet lacking fibre consumes feathers, be it her own or a fellow hen's (Hetland *et al.*, 2004a). A sow obtains up to 10% of her intake from the bedding material (van Barneveld *et al.*, 2003). Bedding materials, such as straw, sawdust, wood chips and wood shavings, are composed primarily of hard fibre (insoluble NSP plus lignocellulose compounds). Hetland *et al.* (2003) demonstrated in laying hens that consumption of 4% of feed as wood shavings resulted in a 50% heavier gizzard.

Not only is the gut the major organ for nutrient digestion and absorption, it also works as the first protective mechanism to exogenous pathogens which can colonise and/or enter the host cells and tissues (Mathew, 2001). The gut is also the largest immunological organ in the body. Thus, it is often implied that a more robust gut will make a healthier animal, which, in turn, digests and utilises nutrients more efficiently. This link between enzyme activities, gut weight and growth performance has been elucidated by Hetland and his colleagues (Hetland and Svihus, 2001; Hetland *et al.*, 2003) where the inclusion of oat hulls in a wheat-based broiler diet increased the gizzard weight, which coincided with a significant improvement (from 97 to 99%) in the digestibility of starch—the most important energy source in broiler diets—in the ileum. This was probably due largely to the massive increase in the amount of starch-degrading enzyme, amylase, secreted. In addition, the gizzard bile acid level increased in proportion to the amount of wood shavings retained in the gizzard (Table 1). Since bile acids enter the intestine through the posterior duodenal loop, their concentrations in the gizzard contents give a good indication of gastroduodenal reflux, supporting the hypothesis that digesta reflux between the gizzard and the duodenum is increased by inclusion of insoluble fibre. Bile acids are strong emulsifiers and they facilitate nutrient solubilisation in the gizzard by effective emulsification of liberated lipids. Lipids are released continuously from the diet by water

Table 1. Performance, jejunal digestive components and starch digestibility in broilers (Hetland *et al.*, 2003)

	Wheat diet, no oat hulls	Wheat diet, oat hulls added
Weight gain, g/21 d	1463	1435
Feed intake, g	2293	2357
FCE ¹	0.64	0.66 (0.61)
Gizzard weight, g/kg	20.6	26.0
Ileal starch digestibility	0.97 ^b	0.99 ^a
Pancreas, g	3.7	4.0
Pancreas, g/kg live weight	2.1	2.2
Amylase, U/g jejunal DM	146 ^b	255 ^a
Bile acid, jejunum mg/g	11.7 ^b	18.0 ^a

¹FCE is presented as corrected means for total dietary fibre contents of oat hulls with uncorrected means in parentheses.

^{a,b}Means in the same row not sharing a common superscript are significantly different.

dilution and protein degradation. An incomplete emulsification of dietary lipids could lead to formation of a protective lipid coating of nutrients in the lumen, resulting in impaired solubility, and hence eventual digestibility, of nutrients. The improvement in starch digestibility may, in part, be due to enhanced emulsification of lipids as a result of more bile acids being available.

There is another side to the relationship between gut health and nutrition. An infected gut (coccidiosis, necrotic enteritis, etc.) is not a healthy gut, and is not efficient in digesting and transporting nutrients. As presented earlier, a heavier and more muscular gizzard appears to relate closely with better utilisation of nutrients; there is also much evidence suggesting that a well-developed gut is essential for the ability of poultry to resist disease (Ao and Choct, 2006). This may mistakenly lead to the notion that a 'heavy gut' represents a 'healthy gut'. It is not so. For instance, the size of the intestine is reduced and the mucosal layer is substantially thinned when antibiotics are added to animal diets (Hill *et al.*, 1957; Henry *et al.*, 1986). This suggests that gut health is related not only to the physical development as a result of stimulation by food and solid particles, but is determined by the organisms harboured in the gut.

GUT MICROFLORA EFFECTS ON HEALTH AND NUTRITION

The diversity of bacterial species in the gut is one of the most important factors for the establishment of a stable ecosystem in the intestinal tract. This is suggested by the observation that, until the bacterial populations are fully established, young animals have fewer bacterial species in the intestinal tract than adult birds, making their gut microflora more susceptible to disturbances than

that of adult animals (Mead, 1989). A stable flora is essential for an animal to resist infections, particularly in the gut. This phenomenon has been described as bacterial antagonism (Freter, 1956), bacterial interference (Dubos, 1963), colonisation resistance (van der Waaij *et al.*, 1971) and competitive exclusion (Lloyd *et al.*, 1977).

The evidence for the protective role of the indigenous microflora of animals against infections of pathogenic micro-organisms has been obtained predominantly from studies with either germ-free or antibiotic-treated experimental animals, which are much more susceptible to infections with intestinal pathogens than conventional animals (Hentges, 1980). Collins and Carter (1978) demonstrated that a germ-free mouse can be killed with 10 cells of *Salmonella enteritidis*, although it requires 10⁶ cells to kill a conventional mouse. The presence of the gut microflora is an important factor in this difference because the LD₅₀ (half live dose) for germ-free and conventional mice is the same whether the animals are challenged intravenously or intraperitoneally. In these cases, the antimicrobials suppress the protective microflora, allowing the pathogen to survive. Furthermore, resistance against colonisation by pathogens may partly result from improvement of the immune system. Data in the literature indicate that the microflora affects the immune status of the bird through its influence on the intestinal wall. In the present context, an animal's immunity is its ability to build up resistance against invasion of pathogenic organisms. Bienenstock and Befus (1980) suggested that the immunity of the animal is affected after a change in the gut microbial activity. The numbers of lymphocytes, plasma cells and intra-epithelial lymphocytes are lower in germ-free animals than in conventional animals (Crabbe *et al.*, 1970). In addition, Peyer's patches in germ-free animals, unlike in their conventional counterparts, are smaller and do not show fully developed germinal centres (Crabbe *et al.*, 1970). Located along the intestinal tract, Peyer's patches are lymphoid tissues containing all components needed to stimulate an immune response.

Perhaps the mostly commonly used method to modulate the gut microflora is the use of live bacteria considered to be beneficial to the host (Morland and Midtvedt, 1984; Perdigon *et al.*, 1990; Havenaar and Spanhaak, 1994). Pollman *et al.* (1980) showed that the inclusion of lactobacilli in the diet of gnotobiotic pigs activated the immune system through an increase in the number of leucocytes. Also, the addition of lactobacilli to the diet of pigs (Fuller, 1989) or mice (Perdigon *et al.*, 1987) stimulates the production of antibodies and the activity of phagocytes against pathogenic bacteria in the intestine.

The presence of antibodies, in particular secretory IgA, is considered to confer a primary line of defence against pathogenic invasions (Fubara and Freter, 1973).

The naturally established protective flora is very stable, but it can be influenced by dietary, disease and environmental factors. For example, hygiene conditions (clean *vs.* dirty environment, pathogen load of the ingredients, humidity of the shed, litter type and usage, etc.), feed additives (antibiotics, coccidiostats, buffers or acidifiers that influence gut pH) and stress (change of feed, sudden disturbances, heat or water stress) can also affect gut microflora. However, diet is perhaps the most important factor influencing gut microflora. Dietary factors, such as composition, processing, digestibility and feeding method, may all disturb the balance in the gut ecosystem, especially in young animals (Choct *et al.*, 1996; Langhout *et al.*, 1999, 2000; Apajalahti *et al.*, 2004). It certainly appears to be the case in poultry that the fermentative characteristics of the gut microflora can be manipulated by diet. Thus, Choct *et al.* (1996) demonstrated that addition of soluble NSP to a broiler chicken diet drastically increased volatile fatty acid (VFA) production in the ileum, which was easily reversed when the NSP were depolymerised with an enzyme. As shown in Table 2, the VFA levels in the ileum were negatively correlated with apparent metabolisable energy (AME) and starch digestion. Interestingly, the antibiotic (amoxil) had little effect on any of the parameters measured in the study.

Other feed additives such as prebiotics, probiotics and enzymes can modulate the gut microflora and performance of broiler chickens. In a recent study, Yang *et al.* (2008) demonstrated significant changes in the ileal populations of lactobacilli and coliforms in broilers fed on diets containing manno-oligosaccharides (MOS) or zinc-bacitracin. In addition, bird performance was enhanced and energy utilisation improved. This shows that a single feed additive can have profound effects on gut health. This point is

Table 2. Volatile fatty acid (VFA) concentrations ($\mu\text{moles/g}$ fresh digesta) in the ilea and caeca of broilers fed on NSP-enriched diets with or without enzyme or antibiotic (after Choct *et al.*, 1996)

Diet ¹	Ileum	Caeca	AME (MJ/kg DM)	Ileal starch digestibility (%)
Control	8.3 ^b	312.3 ^b	13.8 ^a	90 ^a
NSP	118.2 ^a	369.0 ^b	10.9 ^b	56 ^b
NSP + enzyme	5.1 ^b	930.0 ^a	14.1 ^a	92 ^a
NSP + antibiotic	178.9 ^a	413.5 ^b	10.1 ^b	50 ^b

¹Values are means of 8 replicates.

^{a,b}Means followed by different superscripts within a column differ at $P < 0.05$.

elegantly illustrated in the study of Torok *et al.* (2006), who fed broiler chickens on a barley-based diet with or without a β -glucanase, an exogenous enzyme that degrades the anti-nutritive viscous NSP β -glucans. They used the T-RFLP (terminal restriction length polymorphism) method to profile the gut microbial communities. Their data suggested that the two diets resulted in two distinct gut microbial communities. It is, however, too early to draw a conclusion as to a definitive link between a well-performing flock with a 'good microflora' and a poorly performing flock with a 'bad microflora' because it is not known what actually constitutes good or bad flora. Dawson (2001) suggested that an ideal flora should promote the absorption of nutrients during the digestive process, whilst also ensuring that the host is capable of mounting an effective immune response in the event of pathogenic challenge. However, defining what an 'ideal microflora' means in terms of their interactions with each other as well as with the host to yield favourable health and nutritional outcomes remains a key challenge. This is because even with the advent of molecular techniques making it possible to identify up to 640 different species of bacteria in poultry gut, 90% of these species are previously unidentified organisms and their role and functions are totally uncharacterised (Apajalahti *et al.*, 2004).

GUT MICROSTRUCTURE AND NUTRITION

Today, a newly hatched chick increases its body weight by 25% overnight and 5000% by 5 weeks, to 2 kg. This astonishing performance of the modern chicken comes from: (a) intensive selection for growth rate; (b) meticulous attention to health and husbandry; and (c) advances in feed formulation, matching the nutrient contents of the feed with the nutrient requirements of the bird. As the growth period is progressively shortened and feed efficiency continuously improved, the health care and nutrition of the bird are becoming more demanding. This makes it more important to pay attention to the minute changes that occur in the gut, which are often overlooked because the damage is subtle and usually characterised by microscopic changes in the mucosal layer. These minute changes underpin the efficiency of nutrient assimilation because underneath the mucosa is a vast surface of epithelial cells of the absorptive type essential for the transport of nutrients into the enterocytes.

The surface of the mucous membrane over and between the folds is studded with tiny projections called villi. The surface of each

villus is covered by simple columnar epithelium, with cuticular borders, and resting upon a core of connective tissue, the lamina propria. Between the villi are deep pits, the crypts, extending to the muscularis mucosae. Scattered lymph nodules appear in the lamina propria in all parts of the intestine. The villi of the duodenum and jejunum are broader and tongue shaped, becoming finger shaped in the ileum. In general, length and surface area are maximal at the beginning of the small intestine, decreasing gradually to reach a minimum in the ileum just before the ileo-caecal junction.

The development of the gastrointestinal tract (GIT) and nutrient utilisation are intricately related. Hydrolysis of macromolecules in the small intestine is achieved, to a large extent, by pancreatic enzyme activities, which are correlated with body weight and intestinal weight (Sklan and Noy, 2000). It is reported that early access to nutrients and water stimulate the activity of the GIT and digestive organs (Sklan, 2003). Development of the GIT is an important aspect of growth, especially during the early post-hatching period (Sell *et al.*, 1991). Close to and shortly after hatch, segments of the GIT and digestive organs increase in size and weight more rapidly in relation to body weight than do other organs and tissues (Lilja, 1983; Noy and Sklan, 2001). Morphological measurements of the small intestinal mucosa in chicks indicate that villus height increases twofold in the 48 h after hatching and reaches a plateau at 6 to 8 d in the duodenum, but after 10 d or more in both the jejunum and ileum. The width of the villus also increases slightly; thus the growth in surface area tends to mirror the change in villus height. From these data the total surface area of the various segments can be estimated and parallel increases were shown to occur in all segments until 3 d after hatching. After this time, the jejunal area continues to increase more rapidly than that of the duodenum and ileum. With the growth of the villus the number of enterocytes per villus also increases (Geyra *et al.*, 2001).

A rapidly growing broiler devotes about 12% of newly synthesised protein to the digestive tract. An increase in cell proliferation will reduce the age and maturity of the goblet cells, which might affect the quality of mucins they produce. As a consequence, the absorption of nutrients may be reduced (Hampson, 1986). In addition, a fast turnover of these cells will increase the energy requirement for maintenance of the digestive tract. Changes in intestinal morphology as described above can lead to poor nutrient absorption, increased secretion in the gut, diarrhoea, reduced disease resistance and impaired overall performance (Nabuurs *et al.*, 1993). It follows that stressors present in the digesta can

Table 3. Effects of MOS, monensin and Zn-bacitracin on mortality due to necrotic enteritis, NE lesion scores, body weight (BW) and feed conversion ratio (FCR) at d 21 and morphological development of the jejunum (villus/crypt ratio at d 16)

Treatment	NE (%)	NE score	21-d BW (g)	21-d FCR (g/g)	Villus/crypt ratio
Unchallenged control	0.7	0.00 ^b	716 ^{bc}	1.403 ^b	11.5 ^a
Challenged control	13.3	0.33 ^{ab}	607 ^d	1.540 ^a	5.3 ^c
Monensin + Zn-bacitracin	0.0	0.00 ^b	774 ^{ab}	1.338 ^b	11.7 ^a
MOS	14.7	0.17 ^{ab}	621 ^{cd}	1.609 ^a	8.9 ^b
MOS + monensin	1.6	0.67 ^a	721 ^{bc}	1.375 ^b	11.4 ^a
MOS + monensin + Zn-bacitracin	0.0	0.17 ^{ab}	837 ^a	1.343 ^b	11.9 ^a

¹Means of 6 replicates for treatments.

^{a-d}Means within a column not sharing a common superscript differ at $P < 0.05$.

lead relatively quickly to changes in the intestinal mucosa because of the close proximity of the mucosal surface and the intestinal contents. Changes in intestinal morphology, such as shorter villi and deeper crypts, have been associated with the presence of toxins. A shortening of the villi decreases the surface area for nutrient absorption. The crypt can be regarded as the villus factory and a large crypt indicates rapid tissue turnover and a high demand for new tissue. Demand for energy and protein for gut maintenance is high compared with other organs. Cook and Bird (1973) reported a shorter villus and a deeper crypt when the counts of pathogenic bacteria increase in the GIT, which result in fewer absorptive and more secretory cells (Schneeman, 1982).

Savage *et al.* (1997) observed that dietary inclusion of MOS increased the duodenal and jejunal goblet cell numbers, elevated the villus height and reduced crypt depth in poults. A recent experiment (Ao *et al.*, 2008) clearly shows a link between gut morphology, disease resistance and performance in broiler chickens challenged with *Clostridium perfringens*. As shown in Table 3, poor growth, depressed feed conversion ratio (FCR) and a high mortality rate due to necrotic enteritis (NE) coincide with a low villus to crypt ratio in broiler chickens challenged with *C. perfringens*.

CONCLUSION

Health and nutrition are interdependent. The interaction between the two occurs largely in the gut. Thus, the term 'gut health' is a very broad topic that requires a multi-disciplinary approach involving gut physiology, endocrinology, microbiology, immunology and nutrition. If gut microflora is taken as an example, the science determining the roles of micro-organisms in health and nutrition is still in its infancy despite tremendous development in molecular techniques for characterisation of micro-organisms. Questions, such as what constitutes an ideal

flora and how organisms interact amongst each other and with the host, will continue to intrigue scientists for many years to come.

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