Poultry Diseases Influenced by Gastrointestinal Health

Traditional Treatments and Innovative Solutions

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INTRODUCTION TO GUT HEALTH

Feed costs represent a large percentage of the operational expenses associated with animal production. It is for this reason that producers should pay close attention to the efficient utilization (absorption) of feed. From a strict point of view the gastrointestinal lumen, from mouth to anus, corresponds to the external environment and the intestinal mucosa represents the barrier that separates the animal from the environment. Besides its absorptive capacities the intestine must provide adequate protection against pathogenic bacteria. Considering the billions of bacteria that populate the intestinal tract this is not a trivial task.

The maintenance of a healthy gastrointestinal tract insures that nutrients are absorbed at an optimum rate and that bacteria are kept in adequate numbers and confined to their natural niches. Whenever the integrity of the intestinal mucosa is compromised, nutrient absorption decreases. In addition, part of the effectively absorbed nutrients are directed to repair the damaged area and to support the immune system which starts working relentlessly until the intestinal insult is eliminated. In case of prolonged activity, inflammatory processes indeed drain plenty of energy which is otherwise stored as body tissue.

For the mentioned reasons it is wise to use all the available means to ensure that our flock counts with the optimal conditions to achieve the best possible feed conversion. This book focuses on gastrointestinal diseases of poultry and on poultry diseases that do not have an intestinal origin but that are somehow influenced by intestinal heath. In countless occasions, improvement in flock management has a huge beneficial impact on several of the conditions that will be covered in this book, and thus technical advice from poultry veterinarians and our team of poultry specialists is given.

The number of countries that are currently banning antibiotics for non-therapeutic purposes in animal husbandry is increasing; new tools are emerging to control or to ameliorate poultry diseases using an environmentally friendly approach. Among the new available tools organic acids, phytogenics, and especially probiotics will be covered in this book. In addition, conventional treatments for poultry diseases are also listed.

We hope that this guide will increment your knowledge of poultry diseases and poultry management, and that at the end of the rearing cycle you may see this reflected on your pay check.

A. Gino Lorenzoni DVM, MSc, PhD.

SECTION I

BASIC INTRODUCTION TO THE ANATOMY AND PHYSIOLOGY OF THE DIGESTIVE SYSTEM

Basic Introduction to the Anatomy of the Digestive System

The beak is the first anatomical structure of the gastrointestinal system. Unlike mammals birds do not have a clear anatomical distinction between the pharynx and the mouth and the complex formed between these structures is called oropharynx. In contrast to mammals birds do not have soft palate and the palatine cleft or choana, a longitudinal fissure in the palate, connects the oral and nasal cavities (Figure 1).

Figure 1. The roof of the mouth cavity of a juvenile broiler is shown in this picture. Note the presence of the longitudinal fissure (choana).

There are several salivary glands in the roof of the mouth – maxillary, palatine, and sphenopterygoid glands - and in the floor of the mouth – mandibular, lingual, and cricoarytenoid glands. The bucal gland is located in the cheeks (M. Denbow, 2000). Saliva helps to lubricate feed and also contains enzymes (amylase) in some species (not present in chickens or turkeys) that may exert some digestive effect when the feed is stored in the crop (Figure 2) (M. Denbow, 2000). The chicken's tongue is arrow-shaped and helps to propel feed into a sphincter-less esophagus which is thin-walled and divided into cervical and thoracic regions. The cervical region of the esophagus dilates and opens into the crop which is an expansible structure that allows storing of swallowed feed. Mucus glands are located within the mucosa of the esophagus and crop lubricating feed. After a variable storage period in the crop, feed continues through the thoracic portion of the esophagus reaching the stomach.

SECTION II

IMMUNE FUNCTIONS OF THE GASTROINTESTINAL TRACT

Immune Functions of the Gastrointestinal Tract

The gastrointestinal tract is the place of residence and transit of pathogenic and non-pathogenic microorganisms. Due to its extensive surface, the gastrointestinal tract is also a major portal of entry for many pathogens and thus it must be carefully monitored by the immune system. The enteric immune system must also differentiate pathogens from commensal bacteria and food antigens. Failure to accomplish this would result in generalized inflammation and probably massive tissue destruction. Actually, unbalanced immune responses against normal microflora and food antigens seem to cause intestinal disorders like the Crohn's disease in humans. The ability of the mucosal immune system to mount immune responses exclusively against pathogens has fascinated researchers for years. Structurally, pathogen and commensal bacteria share similar molecules. Obviously, one of the factors that limit the immune response and inflammation with commensal bacteria is their lack of invasiveness. However, there are many other factors that could contribute to a moderate response or to a lack of response of the intestine. It appears that the response of the epithelium depends on a complex equilibrium that we are just starting to understand. For instance, at the beginning of the century it was postulated that normal enterocytes were not able to respond to bacterial lipopolysaccharides (LPS) due to a lack of the appropriate cellular receptor (TLR4; toll-like receptor 4) on the apical side of the epithelium (Naik et al., 2001). Contemporaneous research in a different institute showed that there are TLR on the apical membrane of enterocytes but there are also different lines of intestinal cells with different expression of TLR4 and thus variable degrees of response to LPS. Furthermore, after LPS recognition the TLR were shown to traffic to cytoplasmic compartments of the enterocytes suggesting that these cells may play a role assessing the balance of intestinal bacterial populations more than responding to individual signals (Cario et al., 2000, 2002; Suzuki et al., 2003). TLR4 is just one example of the complexity of the mucosal immune system. Interactions between microbiological molecules and the mucosal immune system are highly coordinated by complex communications among the different components of the immune system. In the following paragraphs the main components of the avian immune system in connection to the gastrointestinal system will by described.

Primary Lymphoid Tissue

In birds there are two main areas defined as primary places for development of B and T lymphocytes: the bursa of Fabricius and the thymus, respectively. The thymus is composed by seven lobes at each side of the jugular veins (Figure 10). In a strict sense the thymus receives information from other immune tissues (primary and secondary) scattered throughout the body via blood vessels. However, since it is not anatomically connected with the digestive system it will not be further covered in this book.

SECTION III

MODULATING THE GASTROINTESTINAL ECOSYSTEM AVAILABLE TOOLS

Antibiotics

Antibiotics administrated in sub therapeutic doses have been used for decades as growth promoters (AGP; Antimicrobial used as Growth Promoter) and in many countries this is still an acceptable practice to improve animal production. The mechanisms for the observed improvement in productive parameters (body weight gain and feed conversion) have not been completely elucidated. However, it is suspected that an overall reduction in bacterial load within the intestine is responsible for increased availability of nutrients to the animal. Additionally, a decrease in pathogenic bacteria and their metabolites theoretically could contribute to reduce subclinical lesions on the intestinal mucosa. Less epithelial damage can be indeed an efficient way to save energy since the healing process involves the use of resources to repair the damaged cells. Furthermore, a damaged intestine will mount inflammatory and immune responses to promote the healing of injured tissues and to avoid the entrance of pathogenic organisms into the animal's tissues. In fact, it has been estimated that in broilers undergoing severe experimental inflammation (caused by toxins derived from Gram-negative bacteria) 41 % of the observed growth depression is explained by reactions derived from immune responses and the associated inflammation (Jiang et al., 2009).

The current decrease in popularity of the use of antibiotics to promote growth rate in animal husbandry has been derived from the banning of AGP in several countries. Actually, Sweden banned the use of AGP in 1986 and starting in 2006 the use of AGP was banned in the complete European Union (Benchaar et al., 2009). The banning of AGP is due to the suspected role they have in the development of antibiotic resistance in pathogenic bacteria. Actually, epidemiologic studies in severe cases of gastrointestinal diseases in humans have traced the antibiotic resistant bacteria to commercial animal farms. It is also suspected that humans have a role in the development of antibiotic resistant bacteria. This is derived from the high concentration of antibiotic residues present within the hospitals sewer's system (Kümmerer, 2004).

Antibiotics present in sub-lethal concentrations may effectively exert genetic pressure on bacterial communities favoring those able to effectively resist antibiotic challenges. The genetic pressure favors resistant bacteria to develop in this antibiotic-rich environment that has plenty of space and nutrients available for replication of the survivors (Kümmerer, 2004). The efficacy of antibiotic banning has also been a topic of discussion. In fact, the level of antibiotic used in animal industry has not decreased because a dramatic increase in therapeutic use of antibiotics has been observed. Consequently, even if antibiotics are used in therapeutic concentrations they may eventually reach crops and water sources eventually reaching the appropriate concentration to start exerting genetic pressure in bacterial ecosystems. Despite of the real cause for the emergence of antibiotic resistant strains, the fact is that these bacteria are currently in the environment causing serious problems to humans and animals (Burkholder et al., 2007). In 2004, several water samples were taken in Rio Grande do Norte, Brazil. Within those samples 64 isolates of *E. coli* were isolated from which 36 % were resistant to more than one antibiotic (Cardonha et al., 2004).

Yeast fragments

In the past yeast products have been experimentally tested to reduce the incidence of *Salmonella* in broilers with positive results. In one study, 1 and 100 ppm of *Saccharomyces boulardii* were included in broiler feed. Birds were inoculated by oral gavage with *Salmonella* and 21 days later the *Salmonella* content was measured in the ceca. Yeast extract reduced the incidence of *Salmonella* positive cecal samples in a dose dependent manner. However, the same treatment did not have effects in the reduction of *Campylobacter* colonization in broilers (Line et al., 1998).

In vitro studies demonstrated that D-Mannose is effective protecting chicken epithelia against *S. typhimurium* (Droleskey et al., 1994). Spring et al. (2000) demonstrated that 5 out of the 7 *E. coli* strains and 7 out of the 10 strains of *S. typhimurium* tested agglutinated in the presence of yeast cells and yeast fragments. In contrast, *S. choleraesuis, S. pullorum,* and *Campylobacter* did not agglutinate in the presence of *Saccharomyces cerevisiae in vitro*.

Ganner et al. (2009) used a qualitative microplate-based test to determine the capability of a product containing cell wall fractions to bind *E. coli* and *S. typhimurium*. They reported a binding capacity of 10^4 CFU/mg *in vitro*. When the product was added in broiler diets at 1 kg/ton of feed, weight gain was improved from 1.381 to 1.570 g and mortality was reduced from 5.33 to 2.62 % in the control vs. the treated group, respectively. Treatment with the cell walls of *Saccharomyces cerevisiae* also increased villi length, probably explaining some of the differences obtained in body weight. A possible reduction in bacterial toxins was offered to explain the increased villi length (Zhang et al., 2005). In addition, limited research incorporating *Saccharomyces cerevisiae* into broiler diets suggests that this yeast could ameliorate negative effects of grains contaminated with aflatoxin B_1 (çelýk et al., 2003).

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SECTION IV

DISEASES IN POULTRY DIRECTLY INFLUENCED BY GUT HEALTH

rapidly. In the sub-acute form of necrotic enteritis the clinical signs are severe depression, diarrhea, dehydration, decrease in feed consumption, ruffled feathers, and reluctance to exercise. Necrotic enteritis can also be present in a sub-clinical form in which sub-optimal production can be the sole sign of the disease.

POST MORTEM FINDINGS:

During postmortem examinations the small intestine is usually distended with gas. Intestinal lesions are more prevalent in the jejunum and ileum; however, lesions usually extend to the adjacent regions of the small intestine and could even compromise the large intestine (Long et al., 1974). Advanced macroscopic lesions consist of patches of diphtheritic membrane lining the intestinal mucosa (Figures 14, 15, and 16). The diphtheritic membrane is composed by degenerated epithelial cells, red blood cells, heterophils, macrophages, lymphocytes, fibrin, and bacteria.

Figure 14. Experimental induction of necrotic enteritis in broilers. Small intestine with extensive necrosis. Courtesy of Guillermo Tellez, Poultry Health Laboratory, Center of Excellence for Poultry Science, University of Arkansas, USA.

Microscopically the beginning of the condition is characterized by local destruction of the enterocytes at the apices of the villi. Sloughing of the epithelium is visible and it is

SECTION V

NON-SPECIFIC ENTERITIS IN POULTRY

houses thoroughly. Preferentially let the house rest for several days before receiving new chicks. If possible leave the heater on during the resting period as the causative agents are known to be heat sensitive.

Once the disease is in progress, inclusion of bacitracin methylene disalicylate (BMD) at 220 ppm or virginiamycin at 22 ppm, improves the performance of affected turkeys. In case of an outbreak temperature should never be reduced below recommendation (Zavala and Sellers, 2005).

COMPLEMENTARY CONTROL VIA GUT HEALTH PROMOTION:

Nutritional and management recommendations have been summarized by Cervantes (2003). Supplemental vitamin E in the diet might help providing additional antioxidant effects. 100 I.U. of vitamin E/kg of diet plus adequate administration of Se in the diet (0.3 ppm) ameliorated clinical signs of chicks affected with the malabsorption syndrome (Colnago et al., 1982). Vitamin A should be kept as low as possible. Vitamin A supplementation caused further body weight reduction in affected birds (Veltmann et al., 1985). Feeding a complex ration with different sources of protein seemed to ameliorate the body weight depression in affected animals (Angel et al., 1990). Since intestinal inflammation and bacterial metabolites seem to be at least a part of the problem, addition of probiotics in the diet may help to ameliorate the body weight reduction.

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Figure 25. Examples of good quality turkey feces (left and right). Note the defined shape of these feces.

Figure 26. Left picture: example of watery feces of turkeys; right picture showing watery feces with the presence of gas and undigested particles.

SECTION VI

DISEASES IN POULTRY INDIRECTLY INFLUENCED BY GUT HEALTH

Ascites

ETIOLOGY:

Ascites or pulmonary hypertension syndrome (PHS) in poultry was first identified in countries where birds are raised at elevated altitude like Mexico. Ascites is caused by the inability of the pulmonary vasculature to cope with increasing oxygen demands of metabolically challenged birds. Whenever the oxygen demands are increased (cold, extreme heat, disease, rapid growth rate, elevated feed consumption, etc.) or whenever the ability of the lungs to make an effective gas exchange is compromised (low oxygen tension in high altitude, inflammation derived from respiratory disease, inflammation derived from poor air quality, etc.) the right ventricle must propel additional blood through the lungs in an attempt to compensate for the increased oxygen demands. If blood velocity is increased above a certain point, erythrocytes do not have time to conduct a full gas exchange process in the pulmonary capillaries and under-oxygenated blood will be propelled into the main circulation (diffusion limitation) (Julian., 1993; Wideman et al., 2007). When diffusion limitation ensues arterial blood is not fully saturated with oxygen which establishes the beginning of a positive feedback over the left ventricle that increases the pressure in the pulmonary circulation even further. After the stimuli over the right ventricle have persisted long enough its muscular walls dilate generating a poor sealing of the monocuspid right atrio-ventricular valve resulting in blood regurgitation towards the cava vein. Increased vein pressure is transmitted to the hepatic sinusoids which normally work under very low pressure. Pressure stress leads to histo-pathological degeneration of the sinusoid capillaries which results in hepatic cirrhosis, plasma leaking through the degenerated blood vessels and accumulation of the ascitic fluid in the abdominal cavity (Julian et al., 1987; Wideman et al., 2007).

Unlike mammals, birds have unique physiological limitations that turn them prone to develop PHS. We will review only the most important ones in this chapter.

Birds do not have diaphragm to aid with the respiratory movements. In addition, birds have a relatively rigid rib cage that limits lung expansion during the respiratory cycle.

Birds have relatively rigid pulmonary blood vessels. As a consequence, birds are prone to increase their pulmonary arterial pressure and blood velocity when a higher blood flow is propelled through the pulmonary circulation. Within this context, the balance between vasodilators and vasoconstrictors has a profound impact in the development of pulmonary hypertension. Inflammation derived from continuous inhalation of airborne toxins and ammonia leads to the priming of immune cells that reside inside the airways. It has been reported that heterophils (the avian counterpart of mammalian neutrophils) increase in number after a respiratory insult. Interestingly, birds raised in pristine environments were tolerant to respiratory challenges of 1 mg of intra tracheal LPS while birds raised under commercial environments developed pulmonary hypertension 20 minutes after the respiratory challenge (Lorenzoni and Wideman, 2008).

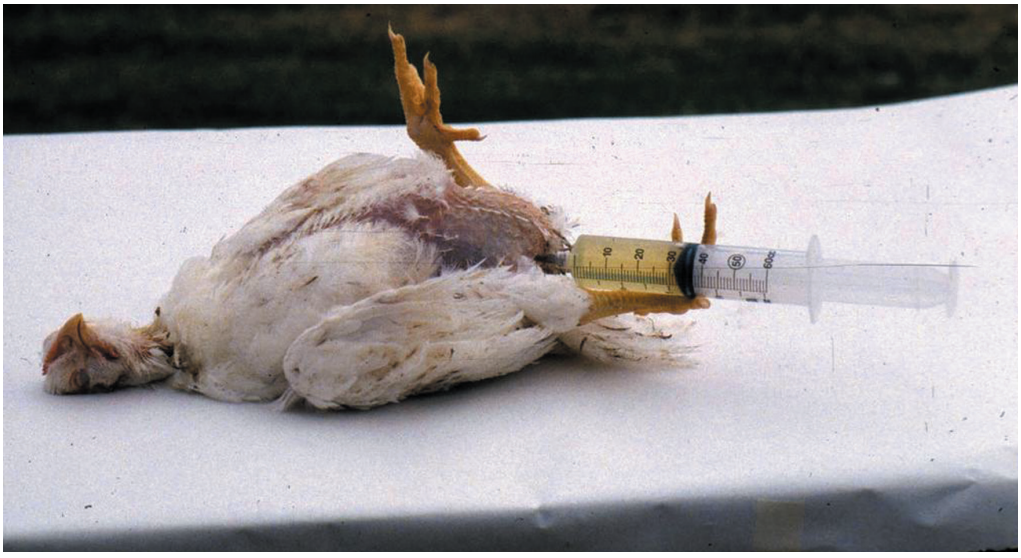

Figure 32. Yellowish ascitic fluid removed from the abdominal cavity of a dead broiler with a 60 mL syringe. Courtesy of Dr. Robert Wideman, Poultry Science Department, University of Arkansas, USA.

Figure 33. The two broilers at the left show typical clinical signs related to ascites: cyanotic combs and wattles and reluctance to exercise compared to the bird at the right side of the picture (notice the bright red color of its comb and wattles). The bird in the middle of the picture was captured gasping in clear respiratory distress. Courtesy of Dr. Robert Wideman, Poultry Science Department, University of Arkansas, USA.

SECTION VII

DISEASES IN HUMANS CAUSED BY BACTERIAL INFECTIONS VECTORED BY POULTRY

Poultry as a Vector for Campylobacteriosis

Campylobacter is a Gram-negative bacterium that is present in the gastrointestinal tract of birds and it is known to affect humans. Isolates of *Campylobacter jejuni* obtained from human patients have been used to produce experimental diarrhea in young chickens. Oral gavage with *C. jejuni* (9×10^7) induced diarrhea in 88 % of 3 day old chickens. Actually, the authors determined that diarrhea could be consistently induced in young chicks using as little as 90 CFU (Ruiz-Palacios et al., 1981). 2 to 3 day old chickens challenged with human derived strains of *C. jejuni* consistently developed diarrhea and the inoculated number of *Campylobacter* was amplified by 3 to 4 logs throughout the intestine. In addition, systemic infection was suggested after isolation of *Campylobacter* from spleen, liver, and blood withdrawn from the heart (Sanyal et al., 1984). However, *Campylobacter* is not currently considered as an important cause of intestinal disorders in poultry and thus this topic will be no further covered in this book. The importance of *Campylobacter* in the poultry industry is largely related to human campylobacteriosis and the role of poultry as a vector and reservoir for this zoonosis. Consequently, human campylobacteriosis will be briefly discussed in this chapter to illustrate the relevance of controlling this pathogen in poultry.

C. jejuni is often considered as one of the most important causes of human food borne disease in developed countries with an estimate of 2.5 million cases of human campylobacteriosis in the USA per year (1020 cases/100000 people/year). Approximately, 100 people die due to *Campylobacter* infections in the USA every year being most cases reported in infants, elderly, or immuno-compromised patients (Mead et al., 1999).

Poultry species are considered to be an important vector for human campylobacteriosis. For example, 83 % of broiler chickens sampled in a live poultry market in New York City carried *C. jejuni* in their intestines (Grant et al., 1980). Using retrospective epidemiological studies, chicken meat manipulation and chicken meat consumption (especially raw or undercooked) were strongly related with increased risk of developing *Campylobacter*-associated diarrhea in humans (Harris et al., 1986). Campylobacteriosis seems to follow different patterns in developed and developing countries. In developing countries campylobacteriosis is reported primarily in young individuals but in developed countries this disease is reported in all age groups (Coker et al., 2002).

Human campylobacteriosis ranges from mild enteritis with watery diarrhea to severe inflammatory diarrhea with abdominal pain, vomiting, and dehydration (Coker et al., 2002). A remarkably feature of human campylobacteriosis is that it can trigger the Guillain-Barré syndrome which is a neurological debilitating immune mediated disease. The Guillain-Barré syndrome occurs in 1 out of 1000 cases of campylobacteriosis taking place approximately 12 weeks after the enteric form of the disease (Altekruse and Tollefson, 2003). Guillain-Barré specifically affects the peripheral nervous system inducing leg weakness and ascendant paralysis. In most cases people recover after several months but close to 8 % of the affected people remain unable to walk after one year and 2 % remain bedridden. *Campylobacter* is not the only trigger for the development of the Guillain-Barré syndrome, but it is related to approximately 40 % of the reported cases. In addition, cases of the Guillain-Barré syndrome

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