

CONCERNING POULTRY

STRATEGIES FOR PREVENTING AND TREATING COCCIDIOSIS AND NECROTIC ENTERITIS

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Coccidiosis and necrotic enteritis are important diseases that affect intestinal health in poultry worldwide. Although both diseases have a different pathology, they act synergistically because the development of necrotic enteritis is highly dependent on the intestinal damage caused by coccidiosis. Anti-coccidial drugs and conventional antimicrobial agents can be used to control the pathogenic pressure in a flock. However, searching for alternative management and dietary strategies to control both diseases is a main topic on the agenda of the poultry industry. One of the dietary treatments could be the addition of butyrate, an energy source for epithelial cells that reinforces the intestinal barrier function. In this article we describe the background on the development of coccidiosis and necrotic enteritis in poultry and discuss dietary strategies to control these intestinal infections.

AVIAN COCCIDIOSIS

Avian coccidiosis occurs worldwide and the species affected are mostly chickens, although other species can also be affected like turkeys, quail and pheasants. The disease is caused by a parasitic protozoan of the genus *Eimeria*. Already at 7 days of

Gut health

Digestive problems in chicks are identified by one or more features:

- The animals move more slowly, sit down together, their feathers look ruffled.
- Abnormal manure: too loose, voluminous manure or sticky droppings with undigested food.
- Water / feed ratio increases: the feeders do not get empty while the water consumption remains the same.

Source: www.diereninformatie.be

age chickens can be infected. The effects are watery and/or bloody droppings, poor weight gain and feed conversion, anaemia, depression, drop in egg production in layers.

The main concern from coccidiosis is that disease is not related to a single *Eimeria* spp. Infections result from a mixture of *Eimeria* species that invade different parts of the intestine, as shown in **Figure 1**.

The intracellular parasite invades and destroys the epithelial cells of the host, causing severe damage to the intestinal wall. Nine different *Eimeria* species are known in poultry, only five to seven of which are associated with diseases in commercial flocks. Whereas an *E. praecox* infection is generally considered to produce little pathology, an infection with *E. acervulina* and *E. mitis* can result in mild enteritis followed by fluid loss and the malabsorption of nutrients.

In more severe cases, inflammation of the intestinal wall with local bleeding (haemorrhages) and sloughing of epithelia (*E. brunetti*, *E. maxima*) or complete villar destruction resulting in extensive haemorrhage and death (*E. necatrix*, *E. tenella*) is seen in infected chickens.

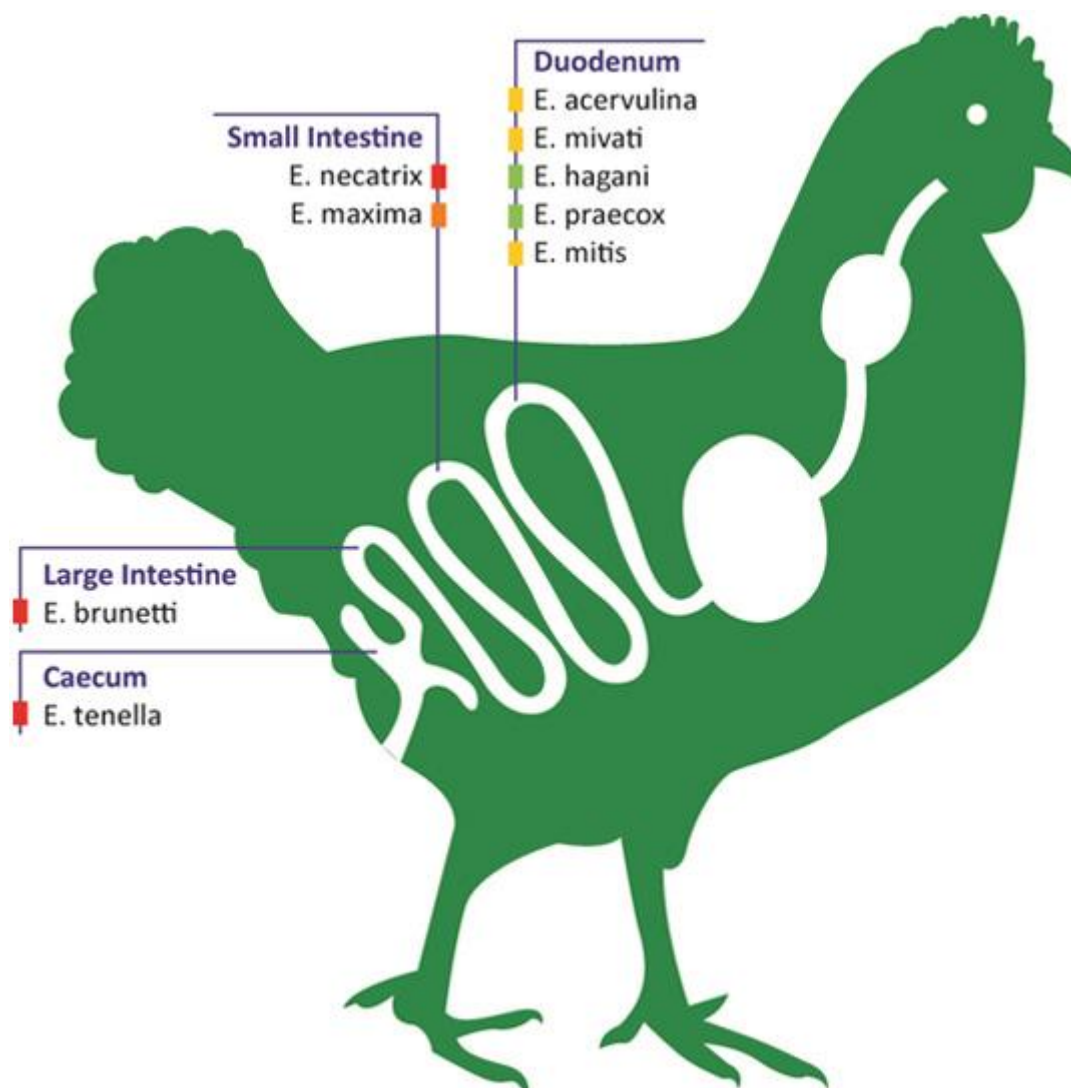


Figure 1 - Nine different species of the parasitic protozoan *Eimeria* are to infect different regions of the avian digestive tract, causing the disease coccidiosis. Colour of the square show the pathogenicity: Green, least; yellow, less; orange, moderate-highly; red, highly.

Most of the highly pathogenic species invade the lower parts of the digestive tract. The life cycle of *Eimeria* is relatively short, from four to six days, and consists of two developmental stages; exogenous (in faecal matter) and endogenous (in host digestive tract).

The exogenous stage starts after the release of unsporulated (non-infective) oocysts in the faeces. Sporulation of the oocyst occurs in the faeces and is encouraged by the right temperature, humidity and access to oxygen in the litter.

The endogenous stage starts after ingestion of the infective oocysts by the chicken. (Step 3, figure 2) In the micro-environment of the gizzard, sporozoites are released from the oocyst. Further down the digestive tract, the sporozoites invade and destroy epithelial cells and start the highly efficient reproduction cycle. This involves several rounds of asexual reproduction, followed by sexual differentiation, fertilisation and the shedding of unsporulated oocysts.

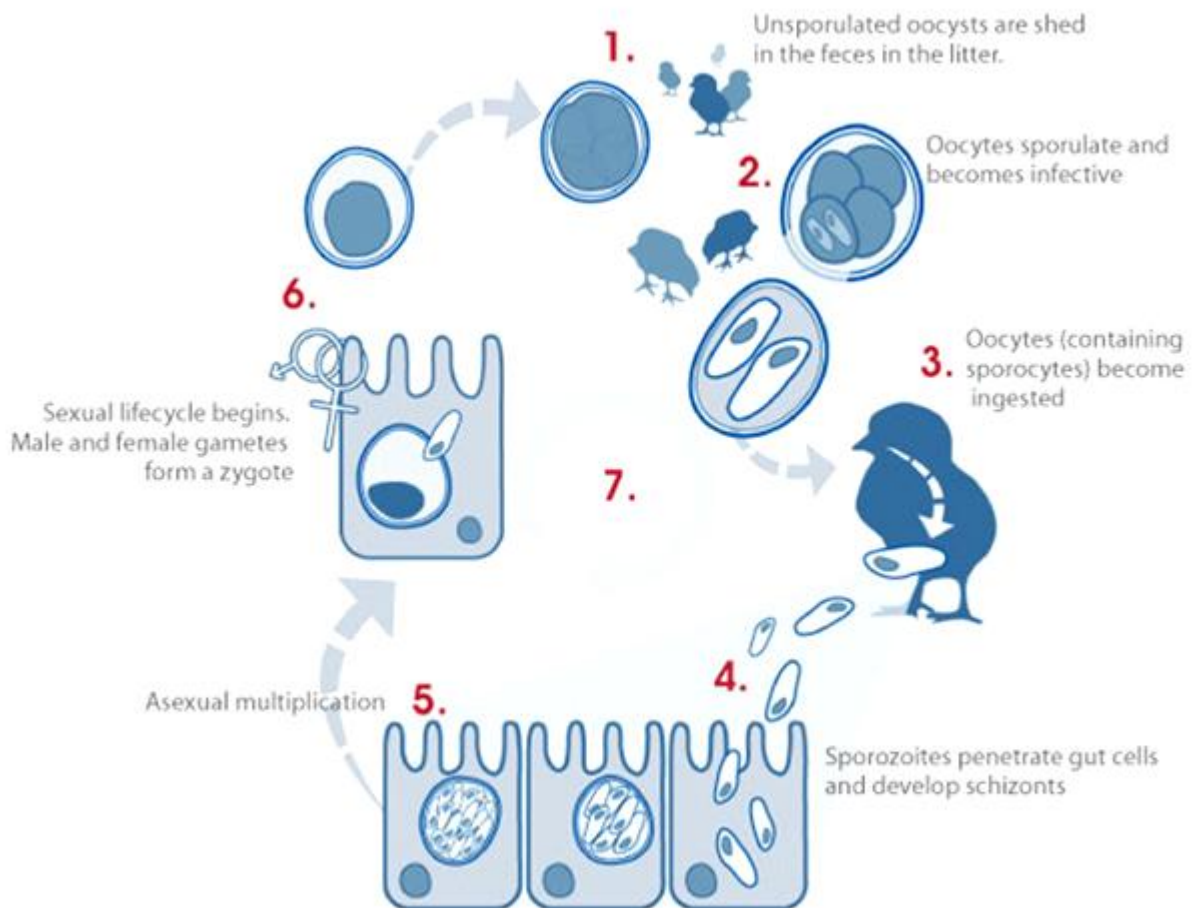


Figure 2: Eimeria life cycle shows the infection of birds by fecal matter and the reproduction of the protozoa in the intestinal cells, resulting in a damages on the intestinal wall (source www.immunocox.com).

Vaccinations and dietary treatments

The oocysts are eventually excreted in the manure. The litter in the stable is thus a source of infection to other chickens that ingest the oocysts via their beak, after which they undergo the reproductive cycle of Eimeria in their intestinal tract. The ideal conditions for sporulation (germination) of the oocysts consist of warmth and high humidity (wet litter, leaking nipple drinkers or overflowing water bowls) and oxygen. It is therefore of great importance to keep the litter dry enough to eliminate the humidity factor in the development of new oocysts. Source: www.diereninformatie.be

Good husbandry helps to reduce the risk of transmitting the coccidiosis-causing parasites. Additional treatments are essential, especially when the parasites cause much harm to the digestive tract of the bird. The best treatment of coccidiosis depends on contamination. Consult your vet for advice for any specific situation.



Left: Baycox® Coccidiocide Solution, one of the drugs for the treatment and control of coccidiosis in chickens, prescribed by a vet after fecal float test. It is administered as a water medication. Baycox Coccidiocide contains Toltrazuril, an active constituent which destroys all intracellular stages of the pathogen's life cycle in the intestine, without impairing the chicken's ability to acquire lifelong natural immunity against coccidia.

Use of vaccines

Besides the number of ingested oocysts, the severity of coccidiosis highly depends on their being immunological memory for the pathogen. As early as 1923, Johnson published the first articles showing that resistance to a dose of oocysts was not age-dependent, but relied on earlier exposure to the parasite. Today, we still make use of this knowledge

through the application of vaccines.



**A vaccine injection used to immunize chicks against coccidiosis.
Photo: Keith Weller/ U.S. Department of Agriculture (USDA).**

Dietary treatments

From a dietary point of view, different strategies can be used to fight coccidiosis. Some products act anti-microbially against *Eimeria*-specific species, e.g. essential oils and herbal extracts. Other products beneficially modulate the immune status of the chicken, whereas prebiotics and probiotics improve microflora in the digestive tract to reduce the chance of secondary infections (e.g. *C. perfringens*). Damaged intestinal tissue benefits from additional antioxidants to reduce the oxidative stress caused by damaged cells. Other products improve intestinal protection and mucosal healing, e.g. betaine, butyrate or specific amino acids.

Necrotic enteritis

This pathogen causes severe intestinal inflammation, which may result in necrosis in the intestinal tissue.

Clostridium bacteria can be present in the feed and bedding and in the intestinal tract of the chicks. Infected birds secrete watery / diarrhetic droppings. The droppings often contain orange coloured intestinal mucus. After the first clinical signs a chick may die within hours.

Source:

www.diereninformatie.be

NECROTIC ENTERITIS LESIONS

Coccidiosis in poultry often pre-exists or occurs concurrently with field outbreaks of necrotic enteritis. The causative agent of necrotic enteritis is *Clostridium perfringens*. Necrotic enteritis has long been controlled by the use of antimicrobial growth promoters (AGP). In 2006, the ban on the use of AGP's came into force and necrotic enteritis re-emerged in poultry farming. Therapeutic antimicrobial agents and anti-coccidial drugs, which not only exert an effect against *Eimeria* spp but also against *C. perfringens*, are currently used to control necrotic enteritis. To achieve the new targets for reducing the use of antibiotics in livestock farming and keeping animals healthy, research for preventive alternatives is being done for many years. Understanding the pathology and predisposing factors of necrotic enteritis is helpful in searching for preventive alternatives.

Necrotic enteritis usually occurs three to four weeks after hatching. The necrotic lesions are mainly restricted to the small intestine and the infection can result in an acute clinical disease or is present in a subclinical condition. In clinical cases, there is increased flock mortality during the last weeks of rearing, often without premonitory signs. The disease is acute, with death occurring within one to two hours, and mortality rates can rise up to 50%.

The pictures below show the difference between Coccidiosis and Necrotic enteritis.

Below, left: Coccidiosis, presence of blood in the droppings.

Right: Necrotic enteritis, presence of orange-coloured intestinal mucosa.



Chronical damage to the intestinal mucosa leads to poor digestion and absorption of nutrients, resulting in reduced weight gain and increased feed conversion. In subclinical conditions, feed intake can be reduced by 35% during the infectious



period. Although clinical outbreaks of necrotic enteritis may cause high levels of mortality, the subclinical form often stays undetected in the flock. The true impact of necrotic enteritis is not from birds that die from infection, but rather those that suffer from disease and survive the subclinical form.

Available nutrients allow proliferation

Clostridium perfringens is commonly found in the normal healthy avian gut microbiota. The virulence of a strain depends on its nature. Strains of *C. perfringens* are classified into five different types (A to E) on the basis of the production of four major toxins. *Clostridium* type A is associated with necrotic enteritis in chickens, although this type is also found in the digestive tract of healthy poultry. As necrotic enteritis is a multifactorial disease, more predisposing factors are needed for its development. High levels of (animal) protein or poorly digestible protein sources are associated with a higher

risk of necrotic enteritis, as undigested protein is a growth substrate for pathogenic bacteria such as *C. perfringens*. Wheat, rye, oats and barley are more often associated with necrotic enteritis, due to higher levels of indigestible, water soluble, non-starch polysaccharides, whereas maize is not. Besides the choice of raw materials, particle size also seems to influence gut health. Feed containing many small and some large-sized particles is more predisposed to necrotic enteritis than feed containing uniform particles. Changes in the feeding regime, other diseases and increased stocking density increase stress in the flock and suppress the immunological status of the chickens, making them more sensitive to necrotic enteritis infection. The intestinal damage caused by coccidiosis is one of the main factors for the development of Necrotic enteritis. *C. perfringens* benefits from cellular components that are released when the intestinal cells are damaged. The mucosal damage caused by *Eimeria* provides a favourable environment for *C. perfringens* to proliferate.

To date, there is no single strategy to combat *C. perfringens*-associated necrotic enteritis. The combination of good hygiene management in poultry houses, the use of vaccination (against *C. perfringens* and coccidiosis) and dietary interventions can, to some extent, be an alternative to antibiotics to maintain production and control necrotic enteritis. Low-protein diets or the use of highly digestible protein sources in combination with enzymes to break down the indigestible structural components in the diet will reduce the opportunity for *C. perfringens* to develop in the gut. Also, the use of additives to control coccidiosis, as earlier mentioned in this article, is a helpful tool to reduce the incidence of necrotic enteritis.

Antimicrobial-resistant bacteria are a major threat to public health and therefore, the use of antibiotics in livestock should be kept to a minimum and only used for treating sick animals. Feed additives are seen as part of the solution.

Multifarious effect of butyrate

The constant immunological challenge presented by *Eimeria* species, concurrently with *C. perfringens*, makes the barrier defence function of the gut wall extremely important. Butyrate is a short-chain fatty acid that is naturally

produced in the digestive tract by the fermentation of fibres. The fatty acid is considered the most important energy source for intestinal cells and has multiple beneficial effects on vital intestinal functions. Research and practical field experience confirm that butyrate alone or in combination with other dietary strategies reduces the incidence of necrotic enteritis.

In the small intestine, butyrate promotes villi development, gut morphology and function. Further on in the digestive tract, butyrate represents the preferred energy source for the cells in de large intestine and is a major precursor for lipid synthesis. These lipids are used for incorporation into the cell membranes. By supporting the cell membrane structure, butyrate contributes to the maintenance of the barrier and transporter functions in the gut. Researchers observed an important role of butyrate in intestinal wound-healing owing to its positive effect on tight junctions (connections between gut cells) and gut integrity. At low concentrations, butyrate reinforces the intestinal defence barriers by increasing the release of protective mucins in the mucus layer and the release of antimicrobial peptides. These peptides, also called host defence peptides (HDPs), possess a broad spectrum of antimicrobial activity against bacteria, protozoa, enveloped viruses and fungi. HDPs bind to the microbial membrane and cause membrane disruption, which results in microbial death.

Below: Healthy Brahma chicks. Photo: Bobo Athes.



The Belgian research group of Van Immerseel shows another antimicrobial efficacy of butyrate, whereby the fatty acid reduces the ability of pathogenic bacteria to adhere to the gut cell wall. Finally, butyrate acts as an anti-inflammatory agent. Tempering the inflammatory status of the chicken is a useful tool to overcome feed intake reduction and thereby reduce the breakdown of muscle tissue during necrotic infection. Uncoated butyrate is directly absorbed in the first part of the small intestine and does not reach the lower parts of the

digestive tract. Proper coating of dietary butyrate is essential for a targeted release of the fatty acid over the whole digestive tract. By testing the micro-encapsulated butyrate product Excential Butycoat it is shown that all butyrate passes the stomach and is gradually released in the intestinal tract.

Conclusion

Since the ban on in-feed antibiotics, necrotic enteritis caused by *C. perfringens* has re-emerged in avian flocks with a severe economic impact. As the occurrence of drug-resistant strains is a significant problem for public health and long-term control strategies against necrotic enteritis, solutions to control necrotic enteritis must be found in alternatives to conventional medication. Nutrition strongly influences the incidence of necrotic enteritis and the other pre-existing pathogenic infection coccidiosis. Proper micro-encapsulated butyrate in the diet ensures a targeted release of butyrate throughout the digestive tract, thus supporting intestinal cells and reinforcing the mucosal barrier function damaged by *Eimeria* species and *C. perfringens*.



Note: Orffa is a direct supplier of feed ingredients to feed mills and supplement producers. Excential Butycoat is also available for the use in ornamental, game and sport birds. The product can be part of a feed or a supplement, please contact your feed supplier for more information.

<http://www.orffa.com/products/excential-butycoat/>

Photo: Archives Aviculture Europe



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