Clostridial enteritis – a continuing challenge for the poultry sector

by Dr Stefaan De Smet and Dr Tom Verleyen, Kemin Agrifoods Europe, Kemin Europa NV, Toekomstlaan 42, B-2200 Herentals, Belgium.

Growth retardation around the third week of age associated with impaired intestinal health is a major problem in broiler production, fully recognised by farmers, veterinarians and nutritionists. This problem occurs so frequently that it is no longer considered a disease by some veterinarians. Clostridial enteritis is prevalent in the broiler industry worldwide and the pathogen involved is Clostridium perfringens.

Clinical signs

Clostridium perfringens infections in poultry may show up as an acute clinical infection or by a subclinical infection. The acute form of the disease leads to an increased mortality of the broiler flock, which can account for high losses of up to 1% per day, reaching mortality rates up to 50%.

Clinical signs include depression, ruffled feathers, diarrhea and lesions in the small intestine are evident macroscopically.

A typical example of necropsies in the gastrointestinal tract due a Clostridium infection is shown in the photograph below.

The clinical form of necrotic enteritis is easily detected and luckily occurs quite seldom in flocks and can be treated.

In the subclinical form of the disease, damage to the intestinal mucosa caused by Clostridium perfringens leads to decreased digestion and absorption of nutrients, reduced weight gain and higher feed conversion.

The subclinical form of necrotic enteritis is the most important as it occurs predominately and has most significant economic impact due to impaired growth rate and feed conversion.

Typical signs seen by poultry producers are specific growth retardation around the 23rd day of age (Fig. 1).

Litter quality changes and becomes more wet, leading to moisture levels above 40% and often undigested feed particles are found in the litter.

The consequences of poor litter quality are obvious, as it leads to increased issues of foot pad lesions, hock lesions and breast blisters resulting in higher levels of rejections at the processing plant.

The causative bacteria

Clostridium perfringens is a commensal organism in the intestinal tract of poultry, colonising in the early phase of life of the animals. It is a Gram positive anaerobic spore forming bacterium, able to produce various toxins and enzymes responsible for the associated lesions.

Clostridium can be classified in five types (A, B, C, D and E), with type A being the most predominant cause of Clostridium infections in poultry. For a long time it was accepted that the alfa toxin is responsible, but new research indicates that Net B is related to the causative form of necrotic enteritis.

Chickens generally take up Clostridium perfringens from the environment, such as feed, water and soil. Inoculation of animals with Clostridium perfringens does not lead per se to the development of necrotic enteritis. One or several predisposing factors may be required to elicit the clinical signs and lesions of necrotic enteritis.

Occurrence

Studies showed that the subclinical form of necrotic enteritis is a worldwide problem with an average of 80% of the flocks having had Clostridium diagnosed (see Fig. 2). A follow-up study in 2005 indicated an increased incidence of Clostridial enteritis in all regions of the world. Recent European surveys confirmed the severity and the widespread nature of the problem.

Impact on performance

Clostridium perfringens associated necrotic enteritis may appear with variable degrees of severity. An overview of the impact of

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Clostridium on broiler performance is summarised in Table 1. Birds acutely infected with Clostridium perfringens will show high mortality rates up to 30% of the flock.

The clinical form of Clostridium perfringens is easily seen and can quickly be treated through medication.

However, as the disease occurs at subclinical level, where mortality is not substantially increased but with clear signs of intestinal disorders, then it becomes more difficult to quantify the impact.

At subclinical level Clostridium perfringens is known as a serious profit killer, leading the FCR to increase with 6-9 points and final body weight to reduce between 3-5%. As subclinical necrotic enteritis is not always detected in the broiler flock there is a serious risk that it can pass unnoticed and affect broiler production economy.

Annual losses to producers in the USA and Canada due to subclinical necrotic enteritis are estimated to be $5 per bird.

Predisposing factors
Several factors can lead to a disrupted gut health. These include an infectious challenge, dietary factors that disturb the gut health balance or a reduced immunity.

Infectious causes:
The most important known predisposing factor is intestinal damage caused by coccidial pathogens. Intestinal damage by Eimeria results in initial damage of the gastrointestinal lining, which is further used by Clostridium perfringens for additional proliferation. Coccidiosis is often seen to occur just prior to or during a necrotic enteritis outbreak. Inflammation of the gastrointestinal tract or a disruption of the gastrointestinal balance due to an infection often leads to the development of Clostridium perfringens.

Nutritional factors:
Dietary factors are also very important in order to control the actions of Clostridium. Diets with high levels of indigestible, water-soluble non-starch polysaccharides (coming from rye and wheat for example) are known to increase the viscosity of the intestinal content which encourages the development of necrotic enteritis. Also, diets rich in high levels of proteins, such as fish meal give an excellent amino acid source to Clostridium which is known as a predisposing factor. As Clostridium lacks the ability to produce 13 out of the 20 essential amino acids its growth is therefore enhanced in an environment rich with proteins.

<table>
<thead>
<tr>
<th>Mortality</th>
<th>Body weight gain</th>
<th>FCR units</th>
</tr>
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<tbody>
<tr>
<td>Clinical</td>
<td>Up to 30% of flock</td>
<td>+ &gt;0.7</td>
</tr>
<tr>
<td></td>
<td>3 to 7% of flock</td>
<td>+ 0.05-0.1</td>
</tr>
<tr>
<td></td>
<td>+1% versus control</td>
<td>-5%</td>
</tr>
<tr>
<td>Subclinical</td>
<td>+ 0.8% versus control</td>
<td>-4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+ 0.04-0.08</td>
</tr>
</tbody>
</table>

Table 1. Impact of Clostridial enteritis on broiler performance.

Typical wet litter due to Clostridial enteritis (picture courtesy Dr R. Chanthirasekaran).
Besides these factors, several other nutritional factors influence necrotic enteritis. Basically all diets with an imbalanced nutritional content can predispose necrotic enteritis.

Diets with a low energy to protein ratio lead to a higher feed consumption. This results in an excessive protein intake and thereby increases the nitrogen level in the digesta and excreta.

Similarly, poorly digested proteins in the lower gastrointestinal tract act as a substrate for the microflora. In order for efficient excretion to take place the animals need to take in larger quantities of water so the litter tends to become wetter with a higher level of nitrogen.

This allows the opportunity for pathogenic bacteria such as Clostridium perfringens to proliferate in the litter which is likely to exacerbate the problems.

- **Reduced immunity:** Any factor that stresses the broiler chicken’s gastrointestinal tract is a risk for Clostridium perfringens proliferation. There is evidence to suggest that alterations in feeding regimes cause stress in the gastrointestinal tract.

**Preventive management**

There are clear indications that Clostridial enteritis is under diagnosed and treated too late in the disease onset.

When a watery intestinal content and wet litter are observed clostridium has already had the chance to proliferate. For a long time the traditional method of managing Clostridial enteritis has been through the use of antibiotic growth promoters (AGPs).

Preventive actions, through products, such as Kemin’s CloSTAT, with selected activity against Clostridium perfringens before the first symptoms are observed, are a valuable solution to maintain a healthy gut flora.

CloSTAT contains a unique strain of Bacillus subtilis PB6 that has been isolated and selected from the gut of healthy chick-