Clostridial Proliferation and Intestinal Instability

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The intention of this paper is to review the syndromes associated with clostridial overgrowth and proliferation as observed in Northern Europe particularly. Several reviews have already been published and salient points worthy of further study will be picked out.

The syndromes are:
- Necrotic enteritis
- Cholangiohepatitis
- Dysbacteriosis

All of these syndromes have been increasing in incidence in both the UK and in Holland (Fabri, 2000) and this has been associated with changes in the use of antibiotic growth promoters (AGPs).

**Necrotic Enteritis.**

This disease is well understood and is caused by Clostridium perfringens type A. The organism replicates readily in the intestinal tract where it produces alpha toxins which are believed to be responsible for the mucosal necrosis (Ficken et al, 1997). The chick is born with a sterile intestinal tract and for the first two weeks of life the oxygen content of the intestinal flora prevents the multiplication of clostridia which require anaerobic conditions (Mortimer, 2000). After the first two weeks clostridia begin to proliferate and clinical disease results if excessive multiplication takes place. An important characteristic of the disease is that subclinical disease which does not result in mortality but is associated with poor performance will be present and the disease can be graded according to severity (Prescott, 1979) and a subclinical form of NE described by Kaldhusdal et al (1992) has been characterised by focal ulceration in duodenum and jejunum.

Risk factors for the disease have been well described in literature, and these are:
- Antibiotic growth promoter removal (Vissiennon, 2000)
- Long anticoccidial withdrawal periods (Elwinger, 1992)
- Concurrent coccidial infections with scores of 2.8 or more (Shane et al, 1985)
Feed and litter contaminated with clostridial spores have been incriminated as sources of infection (Wicker et al, 1977; Ficken et al, 1997)

Diet is believed to be an important determinant of the disease. Manipulation of the diet can affect the numbers of Cperfringens in the intestinal tract (Branton et al, 1997). High levels of wheat and barley (Branton et al, 1987; Riddel et al, 1992; Kaldhusdal et al, 1992; Kaldhusdal, et al 1996) have been associated with NE in experimental and epidemiological studies. Diets high in barley (vs. corn) have also been associated with subclinical NE infection associated with decreased growth rate (Kaldhusdal et al, 1992). Oxidised fats and mycotoxins have also been incriminated (Ferket, 1996)

The viscosity of the intestinal contents will affect clostridial proliferation in the proximal intestinal tract, and the inclusion of enzymes to offset the viscosity of wheat and barley is an important control factor (Smit, 1996)

Despite the identification of the different predisposing factors, no study has been performed that correctly calculates the relative risk of the different factors and no study on the relative importance of different environmental management strategies has been undertaken. Damage to the intestinal mucosa is also considered a predisposing factor in this disease (Truscott et al, 1977). Litter high in fiber content (Truscott et al, 1977) has been associated with NE, and concurrent coccidia infection (Shane et al, 1985; Baba et al, 1991) has been shown experimentally to predispose to the disease.

**Cholangiohepatitis**

Cholangiohepatitis can cause excessive downgrading of between 3% and 5% of all chickens slaughtered, with severe economic consequences. But often, no disease has been reported on-farm. Clostridium perfringens is usually isolated in association with the disease, and antibiotic therapy in the young bird will control its development. Histologically the following is observed: a fibrosing hepatitis, proliferation of the bile ducts, multiple granulomas and compressed hepatocytes (due to proliferation of biliary ductules).

Published work is sparse. However, some work in Canada (Onderka et al, 1990) has experimentally reproduced the condition by either tying off the bile ducts or injecting Clostridium perfringens into the bile duct. The condition reproduced is virtually identical to that which is seen on the processing line. It therefore appears that the clostridia are again the culprit, either blocking the bile ducts or migrating into the liver to then upset liver function.

The assumption is made that the risk factors for the disease are the same as that for necrotic enteritis, but again this has not been confirmed with epidemiological studies.

**Dysbacteriosis**

The dysbacteriosis complex can be defined as the presence of a qualitatively and/or quantitatively abnormal flora in the small intestine and this causes a clinical disorder and/or malabsorption. It is generally seen after 21 days of age and is associated with wet faeces and a reduction in feed intake (Fabri, 2000). In Holland it is well-documented
that the incidence of dysbacteriosis has increased since 1995 from 0.3% to 5.6% in 1999. The absence of AGPs, animal protein and animal fat appear to predispose farms to the disease. Certainly the incidence of dysbacteriosis in the UK since the removal of AGPs has increased significantly. Other predisposing factors may include non-specific stress, mycotoxins and systemic disease.

Bacterial overgrowth has been demonstrated in the intestines of birds affected with dysbacteriosis by the application of Terminal Restriction Fragment Length Polymorphism (T-RFLP) (Panneman et al, 2000), a method of monitoring intestinal microflora by DNA analysis. This method has demonstrated that the proximal intestine of normal birds has very low levels of bacteria, whereas birds affected with dysbacteriosis have substantially higher bacterial numbers. Clostridium spp. have been shown to contribute to this overgrowth. It has also been shown that affected birds, post-treatment with Tylan, had a similar bacterial population to normal healthy birds (Mortimer 2000).

**Conclusion**

Clostridial proliferation and intestinal instability are major threats to sustainable low-cost broiler production – particularly in countries where large amounts of wheat are used. The removal of AGPs from part or all of the production cycle further compounds this threat, as does the imposition of long anticoccidial withdrawal periods. This emphasises the need to develop quantitative risk factors for the different syndromes along with medication regimes that maintain bird performance and intestinal stability most effectively.

**References**


**Biography**

George Tice, BVSc, MMedVet, MRCVS, is a Marketing Manager for Elanco Animal Health, United Kingdom. He began his career in animal health in 1989 as a veterinary surgeon in Bristol, England. After three years of work in large animal practice, Dr. Tice entered academia, serving as a member of the veterinary faculty at the Medical University of South Africa. In 1994 he joined Elanco as a veterinary adviser where he currently supports Elanco’s poultry team.