# Overview of coccidiosis

**By Peter D Constable BVSc (Hons) MS PhD DACVIM, College of Veterinary Medicine at the University of Illinois explores coccidiosis, its diagnosis, treatment and prevention**

Coccidiosis is usually an acute invasion and destruction of intestinal mucosa by protozoa of the genera Eimeria or Isospora. Clinical signs include diarrhoea, fever, inappetence, weight loss, emaciation, and in extreme cases, death. However, many infections are subclinical. Coccidiosis is an economically important disease of cattle, sheep, goats, pigs, poultry and rabbits, in which the liver as well as the intestine can be affected. In dogs, cats, and horses, coccidiosis is less often diagnosed but can result in clinical illness.

## AETIOLOGY AND EPIDEMIOLOGY

Eimeria and Isospora typically require only one host in which to complete their life cycles. Some species of Isospora have facultative intermediate (paratenic or transfer) hosts, and a new genus name, cystoisospora, has been proposed for these species of Isospora. Coccidia are host-specific, and there is no cross-immunity between species of coccidia.

Coccidiosis is seen universally, most commonly in young animals housed or confined in small areas contaminated with oocysts. Coccidia are opportunistic pathogens; if pathogenic, their virulence may be influenced by various stressors. Therefore, clinical coccidiosis is most prevalent under conditions of poor nutrition, poor sanitation, or overcrowding, or after the stresses of weaning, shipping, sudden changes of feed, or severe weather.

In general, for most species of farm animals, the infection rate is high, and rate of clinical disease is low (5%-10%), although up to 80% of animals in a high-risk group may show clinical signs. Most animals acquire Eimeria or Isospora infections of varying severity when aged between one month and one year. Older animals usually are resistant to clinical disease but may have sporadic inapparent infections. Clinically healthy, mature animals can be sources of infection to young, susceptible animals. The critical thing to remember also is that infected animals can ingest small numbers and, ultimately, shed millions of oocysts. This is why we see the disease progressing over the months after turnout. So, it is a disease usually seen where large numbers of calves are housed and there is a big build-up of these coccidian oocysts in the environment. It can affect animals at pasture as well. It is common to see creep-fed lambs/calves being exposed to this disease. Anywhere there is a build-up of faeces in the environment, there is a risk of coccidiosis. Therefore, creep feeders should be moved regularly, and water sources should not be leaking in order to prevent favourable conditions for spread.

## PATHOGENESIS

Infection results from ingestion of infective oocysts. Oocysts enter the environment in the faeces of an infected host, but oocysts of Eimeria and Isospora are unsporulated and, therefore, not infective when passed in the faeces. Under favourable conditions of oxygen, humidity, and temperature, oocysts sporulate and become infective in several days. During sporulation, the amorphous protoplasm develops into small bodies (sporozoites) within secondary cysts (sporocysts) in the oocyst. In Eimeria spp., the sporulated oocyst has four sporocysts, each containing two sporozoites; in Isospora spp., the sporulated oocyst has two sporocysts, each containing four sporozoites.

When the sporulated oocyst is ingested by a susceptible animal, the sporozoites escape from the oocyst, invade the intestinal mucosa or epithelial cells in other locations, and

develop intracellularly into multinucleate schizonts (also called meronts). Each nucleus develops into an infective body called a merozoite; merozoites enter new cells and repeat the process. After a variable number of asexual generations, merozoites develop into either macrogametocytes (females) or microgametocytes (males). These produce a single macrogamete or a number of microgametes in a host cell. After being fertilized by a microgamete, the macrogamete develops into an oocyst. The oocysts have resistant walls and are discharged unsporulated in the faeces. Oocysts do not survive well at temperatures below -30°C or above 40°C; within this temperature range, oocysts may survive ≥1 year.
Of the numerous species of Eimeria or Isospora that can infect a particular host, not all are pathogenic. Concurrent infections with two or more species, some of which may not normally be considered pathogenic, also influence clinical disease. Within pathogenic species, strains may vary in virulence.

## CLINICAL FINDINGS

Clinical signs of coccidiosis are due to destruction of the intestinal epithelium and, frequently, the underlying connective tissue of the mucosa. This may be accompanied by haemorrhage into the lumen of the intestine, catarrhal inflammation, and diarrhoea. Signs may include discharge of blood or tissue, tenesmus, and dehydration. Serum protein and electrolyte concentrations (typically hyponatremia) may be appreciably altered, but changes in haemoglobin or packed cell volume (PCV) are seen only in severely affected animals.

**Figure 1: Normal intestinal epithelium.**

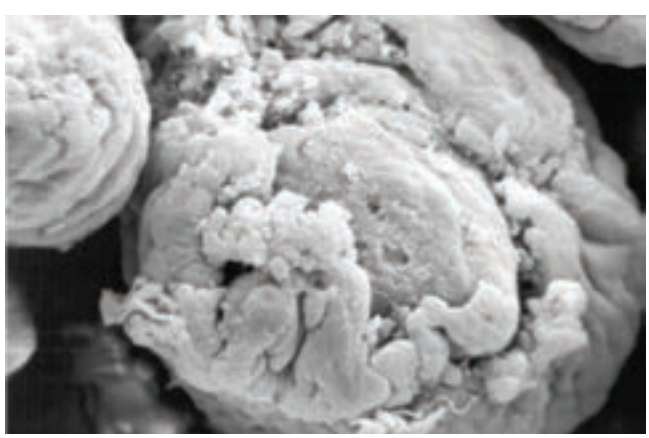

**Figure 2: Five days post infection.**

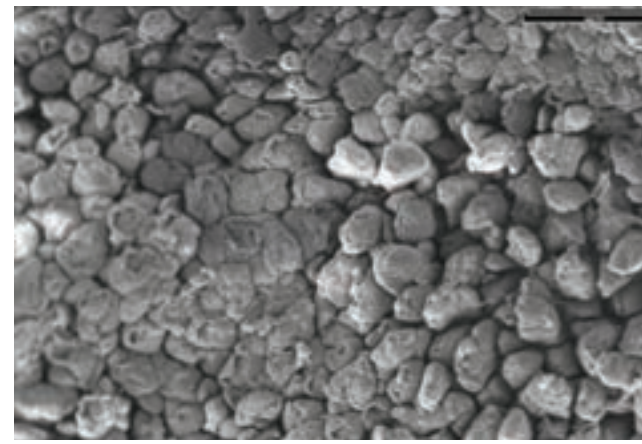

**Figure 3: Five days post infection.**

## DIAGNOSIS

Oocysts can be identified in faeces by salt or sugar flotation methods. Finding appreciable numbers of oocysts of pathogenic species in the faeces is diagnostic (>100,000 oocysts/g of faeces in severe outbreaks), but because diarrhoea may precede the heavy output of oocysts by one to two days, and may continue after the oocyst discharge has returned to low levels, it is not always possible to find oocysts in a single faecal sample; multiple faecal examinations of one animal or single faecal examinations of animals housed in the same environment may be required. The number of oocysts present in faeces is influenced by the genetically determined reproductive potential of the species, the number of infective oocysts ingested, stage of the infection, age and immune status of the animal, prior exposure, consistency of the faecal sample (free water content), and method of examination. Therefore, the results of faecal examinations must be related to clinical signs and intestinal lesions (gross and microscopic). Furthermore, the species must be determined to be pathogenic in that host. The finding of numerous oocysts of a non-pathogenic species concurrent with diarrhoea does not constitute a diagnosis of clinical coccidiosis.

## TREATMENT

The life cycles of Eimeria and Isospora are self-limiting and end spontaneously within a few weeks unless reinfection occurs. Prompt medication may slow or inhibit development of stages resulting from reinfection and, thus, can shorten the length of illness, reduce discharge of oocysts, alleviate haemorrhage and diarrhoea, and lessen the likelihood of secondary infections and death. Sick animals should be isolated and treated individually whenever possible to ensure delivery of therapeutic drug levels and to prevent exposure of other animals. However, the efficacy of treatment for clinical coccidiosis has not been demonstrated for any drug, although it is widely accepted that treatment is effective against reinfection and should therefore facilitate recovery.
Most coccidiostats have a depressant effect on the early, first-stage schizonts and are therefore more appropriately used for control instead of treatment. Soluble sulphonamides are commonly administered orally to calves with clinical coccidiosis.

## PREVENTION

Prevention is based on limiting the intake of sporulated oocysts by young animals so that an infection is established to induce immunity but not clinical signs. Good feeding practices and good management, including sanitation, contribute to this goal. Neonates should receive colostrum. Young, susceptible animals should be kept in clean, dry quarters. Feeding and watering devices should be clean and must be protected from faecal contamination; this usually means feed is placed in troughs above the ground and positioned so that it is difficult for faecal contamination of feed to occur. Stresses (eg. weaning, sudden changes in feed, and transport) should be minimised. Preventive administration of coccidiostats is recommended when animals under various management regimens can be predictably expected to develop coccidiosis.

As coccidia oocysts are robust and long-lived, calf buildings should be maintained as hygienically as possible; this should include the use of appropriate disinfectants, eg. amine-based (Kenocox) or chlorocresol (Interkokask). It should be noted that many of the disinfectants commonly used on farms are ineffective. Feed and water troughs should be clean and raised from the ground in order to limit faecal contamination and reduce the risk of exposure to heavy levels of challenge. Bedding should be kept dry and there must be good falls on floors to avoid pooling of water or dampness of bedding. Plumbing should be maintained to avoid leakage at drinking bowls/troughs. Anywhere there is a build-up of faeces in the environment, there is a risk of coccidiosis. This is why creep feeders should be moved regularly at pasture.

## TREATMENT AND CONTROL

Diarrhoea usually develops at the end of the parasitic life cycle, which means that severe damage to the intestines has already occurred and specific treatment at this stage is usually unrewarding. It is therefore recommended to use drugs prophylactically, i.e. during the risk period, to prevent the development of clinical disease. There are three narrow-spectrum anticoccidials registered for use in cattle in Ireland:

- Decoquinate (60.6g/kg premix for medicated feeding stuff). Feed for 28 days over the risk period. Withdrawal period 0 days;
- Diclazuril (2.5mg/ml oral suspension). Treat 14 days after entering high risk environment. Withdrawal period 0 days; and
- Toltrazuril (50mg/ml oral suspension). Treat during the prepatent period. Do not use in calves weighing more than 80 kg or in fattening units. Withdrawal period 63 days.

These products are primarily indicated for the prevention of coccidiosis by administering them strategically in anticipation of disease. This requires either local knowledge of the history of outbreaks on a particular farm in order to time the treatment appropriately or should be based on the knowledge of the age groups most at risk allied to the occurrence of trigger factors such as weaning, adverse weather or turn out to pasture. Various sulphonamides have also been used in the treatment of coccidiosis. They have limited efficacy against coccidia but may help to suppress secondary infections which may at least partially explain the apparent benefits of sulphonamide treatment in coccidiosis outbreaks.

*Additional information contained within this article was sourced from Animal Health Ireland: Bovine Coccidiosis – The facts. https://online.flippingbook.com/view/372320/6/*

# READER QUESTIONS AND ANSWERS

1. **WHEN DO MOST CALVES DEVELOP CLINICAL SIGNS OF COCCIDIOSIS**
   - **A.** Less than one month of age
   - **B.** Between one month and one year of age
   - **C.** Over one year of age

2. **ANTI-COCCIDIAL PRODUCTS ARE USED PRIMARILY**
   - **A.** For treatment of disease
   - **B.** For prevention of disease

3. **WHICH OF THE FOLLOWING ARE TRUE**
   - **A.** All farm disinfectants are effective against coccidia.
   - **B.** Coccidia oocysts are long lived both in the calf house and outdoors at pasture.
   - **C.** Feed troughs should be moved regularly to prevent faeces and oocyst build up in the area.

ANSWERS: 1:B; 2:B; 3:B&C.