Approaches to helping farmers reduce losses from coccidiosis

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Coccidiosis in ruminants is a disease caused by protozoan parasites of the genus Eimeria, which develop and reproduce within the lining cells of the gastrointestinal tract (Figure 1).

Figure 1. Sexual stages of coccidia (gamonts) within intestinal cells of the large intestine.

In the wild, many animals harbour Coccidia without causing disease, but the situation may change with starvation, debilitation or stressful events that affect the immune system. In livestock, disease outbreaks due to coccidiosis are essentially a consequence of intensification. As with all vertebrate hosts, domesticated ruminants each have their own Coccidia species.

With a few exceptions, Coccidia are host-specific. Therefore, sheep Coccidia only infect sheep and not goats or vice versa, cattle Coccidia only infect cattle, and poultry Coccidia only infect poultry. Even in poultry, chickens and turkeys have their own species of Coccidia, as do game birds (pheasants, partridge and grouse) and cross-infections in gallinaceous bird species does not generally occur, with possibly a few rare exceptions.

This is in contrast to the situation with some avian nematodes (for example, gapeworms and ascarids) and other genera of protozoa (for example, Histomonas, Spironucleus and trichomonads) where cross-infections do occur. As a consequence of host-specificity, a common myth among livestock owners that poultry manure spread on to land may cause Coccidia infection in grazing
livestock is completely unfounded.

**Life cycle and disease transmission**

![Unsporulated oocysts from sheep faeces.](image)

Coccidia have a life cycle in which the different stages develop within the host’s intestinal cells by asexual (merogony) and sexual reproduction (gametogony) – eventually producing unsporulated oocysts (Figure 2).

Oocysts, like worm eggs, are passed out in faeces and need to undergo further development (sporulation) before they become infective to the next host. Infected animals produce billions of oocysts that contaminate the environment, which, under intensive production systems, becomes heavily contaminated and a constant source of infection to subsequent crops of animals.

Clinical coccidiosis is more usually seen in young animals, although older animals can also be infected following periods of stress (Daugschies and Najdrowski, 2005). Another interesting feature is lambs and calves can be infected by a number of different coccidia species, but not all species that occur in each host are pathogenic or cause disease. However, some species are very pathogenic and capable of causing significant levels of infection, leading to clinical disease and even mortality in livestock (Taylor et al, 2016).

During the first few months of life, the majority of calves or lambs will probably have been infected with Coccidia and may show signs of disease – depending on a number of factors, in particular, the level of infection they encounter.

Animals that reach adulthood are highly resistant to the pathogenic effects of these parasites as they develop a strong immunity, but will continue to harbour small numbers of Coccidia throughout
their lives. Occasionally, acute coccidiosis occurs in adult animals with impaired immunity or in those that have been subjected to stress (Taylor, 1995).

Clinical signs

Figure 3. Affected lambs showing faecal staining of the perineum.

Clinical signs of coccidiosis include diarrhoea, often characterised by faecal staining of the perineum and hindlegs (Figure 3), and failure to thrive. Affected animals eventually lose their appetite, become weak and unthrifty. In acute coccidiosis, infected animals show profuse watery diarrhoea, often containing streaks of blood (Taylor, 2010). If left untreated, these animals may continue to scour and eventually die of dehydration.

Diagnosis

The diagnosis of coccidiosis is not always straightforward and should be based on clinical signs, farm history and supported, where possible, by postmortem and laboratory findings. Simply finding or counting Coccidia present in faeces (Figure 4) is not sufficient and the species present should also be examined for the presence of known pathogenic species. Speciation is a specialist skill few laboratories are capable of undertaking.

Economic effects
Figure 4. Large numbers of oocysts in faeces do not necessarily indicate disease. These are non-pathogenic Coccidia species from sheep with two worm eggs also present (yellow arrows). The animal was clinically normal.

The economic losses from coccidiosis to the poultry industry are huge, with an estimated loss worldwide of more than US$3 billion (£2.28 billion) annually (Williams, 1999). It is difficult to estimate the economic losses due to coccidiosis in sheep and cattle.

Losses caused by subclinical coccidiosis are high and result from marked decreases in growth rate, liveweight gain and poor food use. In the US, for example, coccidiosis is reportedly one of the five most economically important diseases of its cattle industry and is estimated to cost US$100 million (£76 million) or more annually.

Estimates on the losses due to the effects of subclinical coccidiosis in dairy cattle in the UK vary between £24 and £59 per animal. In lambs up to 10% may die due to coccidiosis infection and survivors may show varying degrees of weight loss.

**Epidemiology**

In livestock, colostrum generally provides passive immunity during their first few weeks of life, although this can vary depending on the level and standard of farm husbandry. Thereafter, susceptibility to infection increases until the animals acquire resistance through repeated exposure to the oocysts (Taylor, 2000).

Low levels of challenge can actually be beneficial by stimulating protective immune responses in the host. Ensuring the newborn animal suckles, and gets its colostrum soon after birth, is extremely important in protecting against coccidiosis and, indeed, many other diseases.
Resistance to Coccidia infections, however, can be reduced by adverse conditions that affect the immune system, such as changes in environment or diet, prolonged travel, extremes of temperature and weather conditions, or severe concurrent infections with other viral, bacterial and parasitic diseases. In this respect, the management and husbandry of young livestock during the first few months of life plays a key role in both the incidence of coccidiosis infection and the prevention of disease.

How can farmers help prevent coccidiosis?

Figure 5. Coccidiosis is more common in twin lambs – especially where stocking rates are high.

Animals particularly at risk from coccidiosis are young calves, lambs kept indoors on damp bedding, or those on contaminated heavily stocked pasture – especially during cold, wet weather (Taylor and Catchpole, 1994). Twin lambs on permanent pasture are particularly at risk as they are unlikely to drink as much colostrum as singles (Figure 5).

The incidence of disease can be reduced by simple measures, such as providing dry bedding and avoidance of overcrowding and stress. Other measures that can be taken include reducing stocking densities, batch rearing of animals and avoidance of mixing different age groups. It also helps provide plenty of clean bedding in birthing and rearing pens, and to keep young animals off heavily contaminated pastures when most susceptible. Good feeding of the dams prior to parturition and creep feeding of their progeny will also help boost resistance to coccidiosis (Panel 1).

Panel 1. Coccidiosis control measures

- Promote good hygiene and management
Hygiene plays a major part in the control of coccidiosis and, to achieve effective control, good management and hygiene are vital. Regularly moving food and water troughs, and raising or covering them to prevent faecal contamination, can help reduce the level of infection. It is good practice to clean and disinfect all buildings between groups of animals or provide clean pasture for animals turned out on to grass.

Steam cleaning or pressure washing helps remove faecal debris and it is important to use a disinfectant that claims activity against Coccidia oocysts, as not all disinfectants will kill oocysts. Those that are effective contain either ammonia-based disinfectants or chlorophenol (chloro-m-cresole).

**Treatment and control**

Treatment of clinical cases of coccidiosis is possible, but recovery depends on the degree of damage to the gastrointestinal tract, so prognosis should be guarded. Recovered animals often fail to catch up with their cohorts. Any affected animals should be medicated and moved to a cleaner environment or uncontaminated pasture as soon as possible. Supportive therapy may be required, depending on the clinical presentation. Affected animals showing signs of dehydration should be given fluid therapy, and astringents/anti-diarrhoeals may be indicated if there is profuse diarrhoea.

Several anticoccidials are available for use in cattle and sheep, and can be given to prevent and control coccidiosis outbreaks. Decoquinate is given as a creep feed for 28 days (Taylor and Bartram, 2012) or, alternatively, products containing diclazuril or toltrazuril can be given as a single oral drench prior to anticipated disease outbreaks.

The timing of these strategic treatment interventions should be based on clinical judgement and farm history. As coccidiosis is usually associated with a previous stressful situation, the timing can be based around these events. Disease in calves and lambs often occurs from three to four weeks of age and follows one of the risk factors identified above. Under these circumstances, the
treatment should be given about two weeks after the identified stress factor (Taylor et al, 2011; Phillipe et al, 2014).

Practically, treating batches of lambs of susceptible age can be targeted at those around two weeks post-turnout on to heavily contaminated pastures, or at the very first sign of disease. In calves, where coccidiosis is most common between three weeks and six months of age, batches should be treated two weeks following a change in management procedure, such as feed change or move to new accommodation.

The timing of treatment is important because the parasite’s life cycle takes about 18 to 21 days to complete, so treating around 14 days “post-stress” breaks its life cycle at a point where sufficient damaging numbers have not developed, but the parasite exposure allows immunity to develop. In situations where contamination levels vary, subsequent or delayed treatments may be indicated based on knowledge of the farm management system and identified disease risks.

References