Management and treatment of coccidiosis in sheep and goats

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In this article I hope to outline the facts behind approaches to dealing with coccidia, their effect on two host species, and to place these in a practical/ clinical context.

Sheep and lambs that are well spread out on dry grazing.

Coccidia are protozoans (very small single-celled organisms) and span a variety of species. They tend to be species-specific pathogens, where they cause disease and can only survive by replication in specific hosts. There are, of course, exceptions to this rule and this can make taxonomy and identification very difficult. The most common species found in sheep and goats are listed in **Tables 1** and **2**.

All protozoon parasites belong to the phylum Protista and are then contained in a number of sub-phyla, such as Sarcodina, Mastigophora and Microspora. The largest subphylum is the

Apicomplexa, which includes most of the protozoon parasites of large animal interest.

Life cycle

The life cycle of the coccidia tends to be similar in that it establishes in the lining of the host's gastrointestinal tract and then undergoes two merogeny stages – generating giant first generation meronts and then smaller second generation meronts. During gametogeny, the fusion of macro and microgametocytes leads to zygote formation and the unsporulated oocysts are excreted in the faeces. This leads to gradual erosion of the intestinal epithelium.

TABLE 1. Coccidia species recognised in sheep in the UK (major pathogenic species in bold)

Species	Site of infection
Eimeria crandallis	Ileum and caecum, colon
Eimeria ovinoidalis	Ileum and caecum, colon
Eimeria ahsata	Small intestine
Eimeria bakuensis	Small intestine
Eimeria faurei	Small and large intestine
Eimeria granulosa	Unknown
Eimeria intricata	Small intestine
Eimeria marsica	Unknown
Eimeria pallida	Unknown
Eimeria parva	Small intestine
Eimeria weybridgensis	Small intestine

Table 1. Coccidia species recognised in sheep in the UK (major pathogenic species in bold).

These sporulate over a period dependent on climatic conditions (taking longer in cold weather). The sporulated oocysts release sporocysts, containing sporozoites, on ingestion and infect another host of the same host species. This process generates levels of infection that gradually increase until the naïve new ingester is faced with an overwhelming burden and succumbs to the pathological effects of that infection.

If the exposure is gradual, immunity develops, thus the mixing of susceptible animals plays a large part in the impact this disease has at farmyard level. The development of a resistant immunity needs to be encouraged, as a low level of exposure to coccidia is inevitable and, if the animals are not immune, the replication cycle resumes until clinical signs of disease are seen. The immunity generated in goats may vary according to differing exposure levels and different management regimes.

Adult sheep and goats can intermittently shed coccidia on to pasture – thus the threat of disease is effectively present whatever preventive measures are taken.

The disease picture is similar in sheep and goats. The most common form of coccidiosis is a profuse watery scour – often termed a black scour in lambs, due to the extensive damage done to the intestinal lining and accompanying haemorrhage.

Typically, young animals are affected between four to eight weeks old, but this depends on the husbandry system and the precise parasite the animal is infected with.

Diagnosis

TABLE 2. Coccidia species recognised in goats in the UK (major pathogenic species in bold)

Species	Site of infection
Eimeria arloingi	Small intestine (polyp formation and not highly pathogenic)
Eimeria caprina	Small and large intestine
Eimeria christenseni	Small intestine
Eimeria hirci	Unknown
Eimeria ninakohlyakimovae	Small and large intestine
Eimeria alijevi	Small and large intestine
Eimeria aspheronica	Unknown
Eimeria caprovina	Unknown
Eimeria jolchijevi	Unknown

Table 2. Coccidia species recognised in goats in the UK (major pathogenic species in bold).

The clinical signs of scour may or may not be accompanied by large numbers of oocysts shed in the faeces. This makes clinical diagnosis more difficult and, where opportunities exist, postmortem examination of freshly dead carcases will yield more meaningful results.

Faecal oocyst counts are of limited value as, without knowing the species, the oocysts may well be from a non-pathogenic species commonly found in the gut and that cause no disease. Specialist laboratories are needed to speciate the oocysts. It is likely there is an overdiagnosis of coccidiosis as a cause of disease, as high faecal oocyst counts alone do not confirm a diagnosis.

Differential or concomitant disease issues, such as nematodiriasis, teladorsagiosis or cobalt deficiency, should all be considered. Combined infection with *Nematodirus battus* is not uncommon, and the two parasites act to potentiate the effect of disease and work together to annihilate the anatomy of the intestine, as described above.

Symptoms arise from damage to the villi (those finger-like projections of the gastrointestinal tract wall that increase the surface area for absorption of nutrients), and the loss of these means animals that have had the disease and recover will never perform as well as those that have

remained uninfected or disease-resistant.

Coccidiosis is a disease of intensification (or of intensive husbandry), where the increasing severity of disease is linked to the degree of contamination present. The effective recirculation of the parasites through successive cycles of ingestion and elimination allow the initially naïve lamb to develop an immunity where it then becomes a significant risk to subsequent naïve lambs, which face progressively higher burdens of challenge.

Feeding in constant locations and with no creep facilities may increase risks.

There is no cross infection between sheep and goats and, although the life cycles are generally similar, predilection sites and clinical signs both vary.

Symptoms can range from a simple loss of faecal pellet form through to a bloody diarrhoea with loss of appetite, abdominal pain and dehydration – commonly seen as clinical indicators.

Risk factors that increase the chance of developing disease include: heavily stocked pasture (particularly where different age groups of lambs are mixed together); cold, wet weather; and the prolificacy of the flock (larger lamb numbers and multiples increase risk).

In goatherds, risk factors and differentials are similar, and hygiene and housing are important management factors in risk control (**Table 3**).

Prevention

The aetiology and pathogenesis of the disease described above leads to a realisation of common preventive practice. This should include avoiding overstocking and mixing batches of lambs, making sure the ewes' nutrition ensures a good milk supply to the lambs and that intercurrent

disease is dealt with in a timely fashion. Trying to manage exposure to coccidia as a batch and not following on with naïve lambs into the same pasture.

Good levels of hygiene are also important, notwithstanding the need to encourage exposure to coccidia, which will inevitably be shed by the ewes on to the pasture as all treatments suppress rather than eliminate infection.

Treatment

TABLE 3. Differential diagnosis of coccidiosis	
Nematodirosis	Salmonellosis
Teladorsagiosis	Dietary imbalance (high protein content)
Cobalt deficiency	Dietary inadequacy (disease in the ewe)
Copper deficiency	

Table 3. Differential diagnosis of coccidiosis.

Treatment is necessary in disease outbreaks. Preventive therapies can lower the risk of developing the full-blown disease, thus reducing longterm production losses seen following an outbreak. However, the widespread use of in-feed coccidiostats may not be as effective as hoped and, in some instances, serves only to move the challenge of disease to older groups of lambs as the suppressive effect also delays the development of natural immunity.

The careful management of exposure is an essential part of controlling the damage coccidiosis can wreak. Treatment regimes and opportunities are summarised in **Table 4**.

Treatment options for coccidiosis should include dehydration relief through using oral electrolyte solutions and antiinflammatory support, as well as a consideration for pain relief, which is a problem area for minor species, as small ruminants are presently classified.

TABLE 4. Treatment regimes and opportunities					
Product	**Use**	**Availability**	**Route of administration**	**Dose rate**	**Withdrawal period**
Decoquinate (Deccox)	Preventive/ treatment over main risk period	POM-V	In feed: creep feed or blocks	1mg/kg bodyweight	Meat: 0 days
Diclazuril (Vecoxan)	Preventive at time of risk	POM-VPS	Oral drench	1mg/kg bodyweight	Meat: 0 days
Toltrazuril (Baycox)	Preventive dosage prior to onset of clinical signs	POM-V	Oral drench	20mg/kg bodyweight	Meat and offal: 42 days

Table 4. Treatment regimes and opportunities.

Treatment is possible, but recovery depends on the degree of damage to the gastrointestinal tract, and young animals often fail to catch up with their cohort. Preventive therapy is based on predicting a treatment timing that suppresses damage to the gut, while allowing the natural immunity to be

stimulated.

Preventive treatments need to be based on a clinical risk assessment and be monitored for their effect. They may also need repetition where the timing or the level of challenge has not been successfully managed.

History of previous experience with the disease and the alteration of peak risk with different management challenges each year mean this disease threat is one that needs to be continually reassessed.

Lambs showing the after effects of clinical coccidiosis. Note the degree of abdominal distension that can also feature with milk-fed lambs.

Treatment is most effective if the risk can be properly assessed. It has been shown diclazuril appears to have a direct effect on several stages of the parasite’s life cycle (in particular, the large, first-generation meronts) and that maximum benefits can be obtained if treatment is provided early in the infection – before damage to the intestine occurs.

In effect, treatment would be most beneficial if provided at about two to three weeks after an anticipated risk or period of challenge. Under differing management systems, for example, early weaning at eight days old, treatment with diclazuril, two to three weeks later, significantly reduced the levels of oocyst excretion and resulted in improved health status and weight gains when compared with untreated controls.

Common practice under the cascade would be to administer kids a double dose of diclazuril, as it has a wide safety margin, but to keep the same dose as for lambs with toltrazuril, due to its persistence in the gut.

Practically, treating batches of lambs of susceptible age can be targeted at those 10 to 14 days

after turnout on to heavily contaminated pastures or at the very first sign of disease. Where contamination is light, time to recycle the coccidia must be factored into the preventive regime – this depends on knowledge of the individual farm and its management.

The temptation to treat coccidiosis prevention as a routine batch needs to be resisted; reports of suspected resistance to the available pharmaceuticals are often based on poor timing, or on an expectation that the effect of treatment lasts over a longer period. This does not happen, and so the age at treatment, the length of exposure to infection and the level of exposure, as well as the persistence of the active ingredient in the gut, all need to be factored into the timing of the treatment used.

Historically, sulphonamides were among the first treatments used. These are active against first and second stage schizonts and have a dosedependent action that is coccidiostatic at lower doses and coccidiocidal at higher doses. These have now largely been replaced by treatments specific to the coccidian species involved in the disease, but remain in clinical use for acute outbreaks and individual treatments – usually in a potentiated formulation.

It has been suggested the use of treatments to the dams around the time of parturition may aid coccidiosis control in the young. However, in the author’s experience, all this appears to do is delay the peak exposure, as there is still intermittent shedding of oocysts with a resultant delay in increasing pasture contamination as the coccidian population gradually increases.

Outside with plenty of space reduces disease risk.

Deep litter and large numbers increase the risk of exposure in indoor situations.

Hollowed crouched stance of a lamb with severe abdominal pain.

Potential concerns also exist with treating lambs at pasture, due to the metabolites passed in the faeces, which can persist and may have a negative environmental effect. However, this is only likely to be a potential problem in intensively produced indoor lambs, where the faeces remain undiluted by any non-treated animals, and should not be spread on the same piece of ground more frequently than once every three years. The longer persistence of this product in the gut provides a wider timespan for effective dosing, and when used in targeted treatments, is unlikely to cause problems.Coccidiosis remains a difficult, but essential, disease to control, but it needs to be approached by the whole team dealing with the animals and not viewed as a problem that can be solved by reliance on pharmaceutical support alone.

As with all disease in sheep and goats, time ta ken to revisit recorded data on farm and to plan preventive processes will be particularly useful in the consideration of coccidiosis prevention in lambs and kids. This planning depends on the input of the informed clinician.

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