

COCCIDIOSIS INCIDENCE, CONTROL AND EPIDEMIOLOGY PUT IN FOCUS

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Hany M Elsheikha explains how greater cooperation between the veterinary and scientific communities could yield results against a difficult-to-spot-early disease

COCCIDIOSIS is an intestinal disease caused by coccidian protozoa of the genus *Eimeria*.

These protozoa are invasive pathogens that colonise the mucosal surface of the intestine, causing major economic losses in farm animals.

Biology

Coccidia have a direct, yet complex life cycle (from ingestion of the oocysts, to passage from the host in the faeces) that can be completed in roughly 18 to 21 days in cattle and sheep.

Infection is spread through the faecal-oral route, with the ingestion of infectious-stage mature oocysts. Direct transmission through the contamination of barns and/or pasture appears to be the principal mode of infection. The organism reproduces in the host's intestine, and thousands of oocysts are shed into the environment through the faeces.

Under proper temperature, moisture and oxygen conditions, the oocysts mature within three to seven days and become capable of infecting the animal. Each mature oocyst contains eight sporozoites, each of which is capable of entering a cell in the animal's intestine after the oocyst is ingested ([Figure 1](#)).

Clinical disease

Approximately 70 per cent of the life cycle is spent in the small intestine, where the parasite burrows in and out of intestinal cells, causing diffuse damage to the intestinal mucosal surface, and a reduction in the height of the villi and microvilli.

The normal consequences include a loss of the absorptive surface area throughout the small intestine and a reduced capacity to absorb required nutrients. There is impaired glucose, water, and sodium absorption in the small intestine, and disaccharidase activities are also reduced, resulting in impaired digestion. An increase in intestinal motility has also been reported in infected animals.

In growing ruminants, coccidiosis has a substantial negative effect on animal performance and productivity, leading to a reduced rate of gain in feeder calves without a reduction in feed intake, suggesting malabsorptive and/ or maldigestive disorder. Infected animals may also have lower carcass weight and an increased time to slaughter. Many cattle are affected and experience weight loss or decreased weight gains without showing obvious illness (sub-clinically infected). These cattle account for the majority of the economic losses (Fitzgerald, 1980). Sub-clinical infections are costly because calves will achieve lower-than-expected weight gains and, generally, not perform as expected.

Cattle and sheep experiencing severe bouts of coccidiosis may never perform as well as noninfected pen-mates. Because of the damage inflicted on the intestinal tissue, the digestive process and overall homeostasis can become severely affected, even with the absence of clinical disease, with adverse effects on animal welfare and performance. *Coccidia* not only damage the host's intestinal tissues and result in reduced absorption capacity, but also:

- cause blood and tissue loss, for which there is a cost of substitution;
- allow secondary pathogenic organisms and toxins to enter; and
- induce immunosuppression and make the host more susceptible to other diseases.

Clinical signs don't appear until late in the *Eimeria* life cycle (approximately day 18), when damage from the disease has already occurred. Infections with the highly pathogenic species, *E. bovis* and *E. zuernii* in calves and *E. ovinoidalis*, *E. bakuensis* and *E. ahsata* in sheep, can lead to moderate-to-severe diarrhoea (sometimes bloody and may contain shreds of mucous), tenesmus, weakness, rough coat, loss of appetite, slight fever, debility, listlessness, abdominal pain, dehydration, weight loss, growth retardation, impairment in weight gain, decreased feed efficiency in feedlot cattle, increased susceptibility in neonates to other infections (especially in endemic areas) and mortality in severe cases. Even though clinical coccidiosis is frequently not diagnosed in adult animals, it may be the most common cause of diarrhoea in calves and lambs.

Incidence

Coccidiosis is geographically widespread worldwide. Although coccidiosis cases can occur sporadically, clinical outbreaks have been reported from several European countries (Svensson et al, 1994; Marshall et al, 1998; Snoep and Potters, 2004).

The prevalence of *Eimeria* infection in cattle is generally high and can reach 100 per cent in calves (Fox, 1985). A study by Stewart et al (2008) on the prevalence of coccidiosis in England and Wales, reported an overall prevalence of seven per cent (88 of 1,253 samples). The prevalence and infection pressure of the various more or less pathogenic species may vary considerably between farms, regions, season and age groups. Although coccidiosis affects animals in all age groups, the number of reported cases is highest among calves and lambs aged one to three months.

The prevalence and/or incidence of clinical coccidiosis in the UK is not fully known and, although there have been some studies on coccidiosis, research has been very limited and there is no up-to-date data available on the dynamics of coccidian pathogens. Season is not a consistently reported risk factor for *Eimeria* species, but trends in the prevalence with season have been reported. According to the Veterinary Investigation Diagnosis Analysis (VIDA, 2006) report, the number of diagnoses increased during the spring and early summer in sheep, and late spring, summer and autumn in cattle ([Figure 2](#)). The high prevalence of coccidiosis in lambs during spring and early summer might not be related to the season, but rather to the increasing number of young lambs with the most susceptible ages during this lambing season.

In cattle, the incidence is less seasonal, but its apparent increase during the grazing season may reflect a greater incidence of sample submissions to differentiate from other gastrointestinal parasites.

In the UK, cases of cattle coccidiosis are more prevalent in winter, relating to peak calving in autumn and, therefore, at-risk groups (calves one to four months old) are most susceptible in the winter period. In other cases, an increase has been reported to occur in pastured calves within two weeks of spring turnout (Dauguschies and Najdrowski, 2005).

Yearly coccidiosis trends in cattle and sheep showed non-stability over the past few years (1999-2006). As shown in [Figure 3](#), the yearly incidence peaked in 1999; declined through 2000-2001; peaked up again in 2002; then gradually declined to 2005; followed by a peak at 2006. This pattern was consistent among cattle and sheep. Coccidiosis is not listed as a notifiable disease in section 88 of the Animal Health Act 1981, thus, all available data is based on the submissions made to the Veterinary Laboratories Agency (VLA), Regional Laboratories (RLs) and Scottish Agricultural College (SAC) disease surveillance centres (DSCs) in Great Britain. The inherited bias in the VIDA data surveillance scheme among regions can affect the capability to detect cases, making interpretation of coccidiosis trends difficult.

Parasite epidemiology

Most infections in ruminants have been described as asymptomatic, and older cattle infrequently show clinical signs.

Disease only occurs if animals are subjected to heavy infections, or if their resistance is decreased. It is, therefore, important to differentiate between infection and disease, as the mere presence of *Coccidia* does not invariably lead to the development of clinical outbreaks. Indeed, trials have demonstrated that low levels of challenge can be beneficial by stimulating protective immune responses in the host.

Most cattle are infected at an early age and carry some *Coccidia* through adulthood. Continuous exposure to low numbers of oocysts, which is often the case under field conditions, results in endemic stability. After continuous exposure to the organism, cattle develop some degree of immunity. However, immunity to *Coccidia* is species-specific: cattle exposed to only *E bovis* and develop immunity will only be immune to *E bovis*. *Coccidia* species are also host-specific (in effect, species affecting cattle act on cattle, not pigs or chickens).

Cattle carrying *Eimeria* are a source of infection for other cattle. In cases of true coccidiosis outbreaks, diarrhoea will be accompanied by excretion of considerable numbers of pathogenic *Coccidia*, particularly *E bovis* and/or *E zuernii* and, occasionally, *E alabamensis*. Cattle, especially young calves, have been recognised as a significant source of oocysts because of the high prevalence of infection, the high numbers of oocysts shed within their faeces, and the large volume of manure generated by confined beef and dairy cow operations. Because of the high reproductive potential of *Eimeria* species, infected animals may shed millions of oocysts daily during patency, thus contaminating their environment with the pathogen.

The primary concern in coccidiosis outbreaks is the potential to spread the disease to other susceptible animals in the herd. Susceptible animals exposed to moderate numbers of oocysts will seldom develop signs of disease. However, because of their lack of resistance, they will produce millions of oocysts in their faeces. These oocysts will then contaminate the feed and water supplies in sufficient numbers to fatally expose susceptible animals. Animals at increased risk of infection include:

- animals with close contact with infected animals;
- animals that ingest contaminated food or water;
- animals housed in proximity or confined in small areas, such as yards or small paddocks; and
- young ages or weaners.

Coccidia are ubiquitous and unlikely to be destroyed in nature, because the oocysts have a protective carbohydrate wall that makes them resistant to environmental destruction and provides protection against a wide range of chemical disinfectants. Oocysts sporulate within a few days at ambient temperatures, and maintain infectivity for months – and more than a year in a 4°C environment. Consequently, the infection pressure rapidly increases in infected calves' surroundings. High infection pressure increases the individual risk to acquire clinical coccidiosis, and factors that impose stress on the calves, such as weaning, weather, transport, frequent regrouping, inadequate feeding or other infectious diseases, may exacerbate the condition. Therefore, it is not feasible to manage the condition by treating only the external environment.

For successful and economical control of coccidiosis, detailed knowledge about the *Eimeria* species involved, and the risk factors for infection in livestock, will allow for the logical development of oocyst shedding management strategies. The role of the environment in shaping the parasite epidemiology, such as climatic changes and increased demand for livestock production, cannot be disregarded.

As we continue to make progress in understanding the behaviour and epidemiology of coccidiosis, many new opportunities will evolve to improve the prevention of this disease.

Controlling and preventing coccidiosis

Prevention is still the primary and most effective strategy of controlling coccidiosis. Modern, high-density confinement operations are ideal for the survival and reproduction of Coccidia and other parasites because they concentrate both the host and parasite populations, leading to clinical infections. Both clinical and sub-clinical coccidiosis result in a decline in herd condition; if left untreated, mortality can occur. Even animals that recover from severe infections may suffer permanent production losses.

As shown in [Figure 4](#), control of coccidiosis can be best accomplished by:

- exact diagnosis of the *Eimeria* species involved;
 - applying proper sanitation and good animal husbandry practices;
 - using chemotherapeutics; and
 - controlling the numbers of oocysts ingested by the animals, while effective immunity builds through the use of coccidiostats, especially when exposure is imminent.
- Diagnosis of coccidiosis can be difficult and should be undertaken by an experienced professional. A preliminary diagnosis made only on clinical signs can be misleading because

clinical signs of coccidiosis are easily confused with other causes.

Additionally, large numbers of non or low-pathogenicity coccidian may be present, which could lead to misidentification. Misdiagnosis can result in overlooking other diseases and contributing to overall health problems.

Proper diagnosis of coccidiosis includes history, clinical signs, faecal oocyst examinations (count and speciation), and postmortem examination of dead animals.

- Good management practices are important when establishing coccidiosis control programmes. Recognising that animal manure is a major contributor of *Coccidia* oocysts in the environment, strategies for management and instigation of preventive measures should focus on safe and prompt disposal of animal waste, as well as the protection of water supplies. Measures that reduce transmission between animals should be encouraged.

First, livestock industries should be constantly reminded of the threat coccidiosis poses to animals.

Second, limit animal transmission and oocyst load via limiting the number of animals enclosed in the same facilities (reduced stocking density), and minimising contact or isolating young animals from adults. Maintaining a short calving period may assure reduced opportunities for the parasite to spread within a herd. However, under UK conditions, a shorter calving period is likely to lead to an increase in production intensity (increased group size with stocking density) of the most susceptible animals, as in most systems, most young calves will be housed for at least four months.

Thirdly, flies can carry oocysts to animals' water and food sources. Physical barriers, such as insect screens and other environmental control strategies, should be placed in areas where animal manure is densely accumulated.

The key point in reducing the risk of coccidiosis is to limit exposure to coccidian oocysts via keeping the level of contamination down by:

- raising feed and water troughs high enough off the ground to avoid faecal contamination;
- keeping pens clean, well-drained and supplied with ample dry bedding;
- using well-drained pastures;
- minimising faecal contamination of hair coats and fleece;
- adopting an “all-in/all-out” method of calf movement from pen to pen, with full clean out;
- avoiding overgrazing, as animals forced to graze down to the roots of plants may eat large

numbers of parasites;

- turning out to clean pastures (those not grazed by calves in the same or the previous year);
- isolating heavily parasitised animals from the rest of the herd;
- applying strict sanitation conditions; and
- instigating regular movement to concentrate feeding points and mineral licks for animals at pasture, and keeping a distance between these points and wet areas.

While controlling coccidiosis, the above steps will have the added benefit of preventing many other diseases.

With regards to reducing stress on the animals, coccidiosis is more frequently associated with intensive husbandry systems and high stocking densities. Infection control should consider changes in the management factors that contribute to stress development by:

- avoiding overcrowding, nutritional disorders, weaning and other stresses;
- avoiding grazing young and older lambs on the same pasture;
- grouping calves by size;
- isolating ill animals;
- ensuring an adequate intake of colostrum to assist newborns in coping with infection; and
- correcting the pens' ventilation and housing conditions.

- With regards to treatment, the overall objective of any control programme is to reduce the economic impact of the disease. Although infected animals may recover without treatment, due to acquired resistance to the disease, treatment with anticoccidial drugs should be administered when the earliest clinical signs arise, because they may abrogate the further development of oocysts and reduce the disease's severity, as well as decrease mortality (Fox, 1985).

A major challenge in treating clinical coccidiosis is that signs of the disease do not appear until the life cycle is almost complete. By this time, most of the damage to the intestine has already been done and, therefore, therapy is only of limited value.

The main targets of therapeutic drugs are the sexual (gamont) stages. Therefore, the difficulty in treating coccidiosis is that, by the time signs appear, parasites have already passed through the

stage in which anticoccidial drugs are most effective.

Batch rearing lambs in groups of similar ages allows for targeted treatment of susceptible age groups. Also, it is usually more economical to treat the whole cattle pen, rather than relying on individual calf treatment. To prevent losses due to coccidiosis, exposed calves should be treated pro/metaphylactically, rather than therapeutically (Mundt et al, 2005).

The cost of therapy after a clinical outbreak of cattle coccidiosis is considerably higher than the cost of early treatment of infection to prevent the disease. Furthermore, treatment of severely diseased animals is not very rewarding, since clinical signs result from the final stage of the parasite cycling in the host.

In fact, later treatment may not be as effective, since the gamont stages of the *Coccidia* life cycle are generally not as sensitive to coccidiostats as the earlier asexual stages. Therefore, a strategic plan of preventive treatment, based on a previous history of the disease in a particular environment and other management considerations, could offer the best way to reduce losses due to cattle coccidiosis.

Once an accurate diagnosis is made, a number of drugs with anticoccidial activity can be used for the therapy of clinically affected animals. In the UK, current licensed medications used to treat coccidiosis in cattle and sheep include sulphadimidine, decoquinate and diclazuril.

Sulphonamide drugs also act against certain bacteria that may help suppress secondary infections, and may partially explain the benefit of sulphonamide treatment in coccidiosis outbreaks.

Antibiotic therapy is recommended to prevent secondary gastrointestinal bacterial infections. Intestinal protectants that coat damaged intestinal mucosa may assist recovery from severe enteritis.

In an outbreak, the clinically affected animals should be isolated, drenched with electrolyte mixtures and given supportive parenteral fluid therapy (as necessary) to reduce dehydration and maintain haemostasis.

The owner must be made aware that drugs administered in feed or water may not be consumed by sick animals. Animals with coccidiosis and nervous signs should be brought indoors, kept well-bedded and warm, and given fluid therapy orally and parenterally.

However, the case fatality rate may be high, despite intensive supportive therapy.

- Regarding the use of coccidiostats, there is no vaccine for coccidiosis. The idea of using coccidiostats is based on the notion that most coccidiostats do not kill, but interfere with the reproductive potential of *Coccidia*, allowing enough antigenic stimulation of the immune system to

increase resistance while the animal is protected by the drug. When the coccidiostat is removed from the diet, resistance to disease will be maintained as long as exposure to the *Coccidia* continues.

Routine prophylactic medication of feed, saltprotein blocks or water usually prevents severe outbreaks. Indeed, using feeds that contain coccidiostats prior to the outbreak of disease may be essential, especially in crowded conditions.

A clinical research trial found that coccidiosis prevention, using decoquinatate in weaned calves that are minimally exposed to *Coccidia*, may have important production benefits through improved feed efficiency and gain rate.

To be most effective, coccidiostats should be given early in the coccidian life cycle and before massive infections overwhelm the animal.

In some cases, producers start prophylactic use of coccidiostats in pregnant females several weeks prior to calving or lambing, and continue until about 60 days postpartum. Calves and lambs are started on coccidiostats in creep rations and continue until after the stress of weaning has subsided.

In cases where new animals are introduced into a herd, the producer should treat the incoming animals with the control dose of a coccidiostat for at least 28 days to prevent coccidiosis prior to mixing animals. This should reduce the incidence of stressrelated coccidiosis in the incoming animals and reduce contamination of existing facilities.

The ideal coccidiostat suppresses the full development of the *Coccidia*'s life cycle, allows immunity to develop and does not interfere with production performance. A number of labelled coccidiostats can control or prevent coccidiosis.

Decoquinatate is a feed additive effectively used as a preventive treatment in confined cattle and sheep. It can also be used as treatment to reduce the effects of an acute outbreak.

Clinically affected cattle or sheep should be treated with sulphadimidine, and coexistent cattle should then receive decoquinatate to prevent further oocyst cycling.

Toltrazuril is effective at preventing coccidiosis in cattle. Diclazuril is also used to prevent coccidiosis in sheep and as an effective aid to control the disease in cattle. Continued use of coccidiostats will, over time, lessen the number of oocysts passed into the environment for as long as that coccidiostat is effective.

However, the continued use of the coccidiostat against a population of parasites for extended periods will eventually lead to resistance to the coccidiostat.

Experience with poultry showed the continuous application of anticoccidial drugs led to the development of *Eimeria* species' anticoccidial resistance to virtually all drugs (Stephan et al, 1997).

Therefore, preventive measures should rely not only on drug application, but include evaluation and, if necessary and possible, improvement of management and hygiene.

Outlook

Although they have a direct life cycle, *Eimeria* species are very complex organisms, from their biochemistry to the mechanisms of virulence.

Pathogenesis areas that should be pursued include defining soluble factors, further elucidating the contact-dependent relationship between the intestinal epithelium and *Eimeria*, and defining how the parasite can establish itself in a normally inhospitable and changing environment. It will also be important to further define the parasite's impact on host physiology and the role of the animal immune response in coccidiosis to develop targeted intervention strategies. With continued collaboration and cooperation within the scientific community, we may one day understand the pathogenesis of *Eimeria* species well enough to develop a safe, effective and cost-effective vaccine.

Concluding remarks

- Coccidiosis is a significant cattle and sheep disease that mostly affects neonates, with serious health and economic burdens.
- Transmission occurs from animal-to-animal, by water ingestion and food contaminated with oocysts that were excreted in the faeces of infected animals.
- Mechanical transmission by flies is also documented.
- Feedlot conditions provide the ideal circumstances for an outbreak.
- Preventive measures are by far the most effective approach in controlling this parasite.
- Treatment and prevention are most effective when initiated early.
- Mass treatment or open medication is usually effective.
- Implementation of measures to decrease the spread of the parasitic oocysts in the environment is critical.

- The role of the veterinarian in coccidiosis diagnosis, treatment and counselling is relevant for disease management and prevention.
- Continued research is needed to increase our knowledge of the parasite's epidemiology, evolutionary biology, virulence and molecular diversity.
- Understanding the biological behaviour of *Coccidia* will be critical if intervention and control strategies are to be effective.

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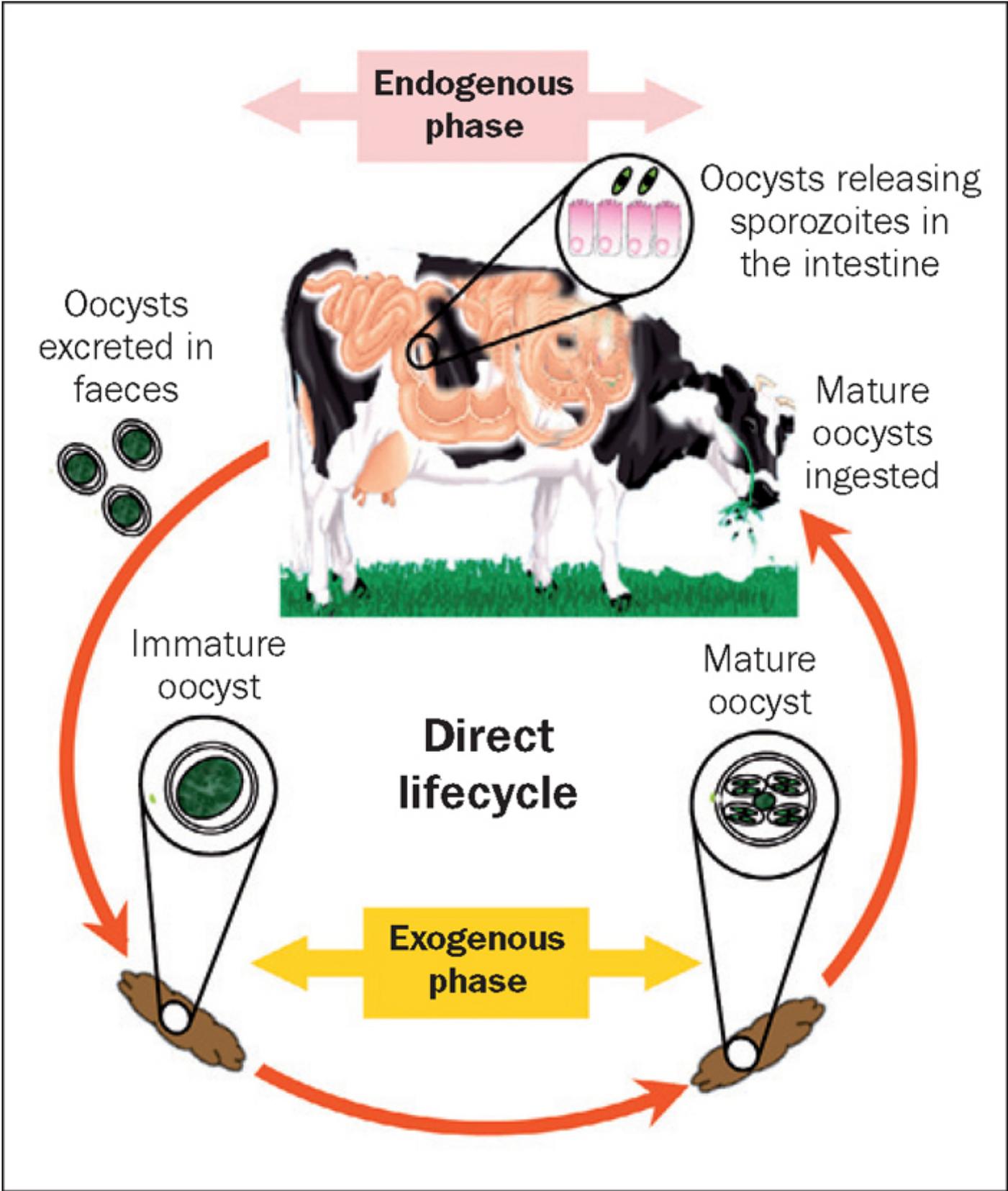


Figure 1. The general life cycle of *Coccidia*.

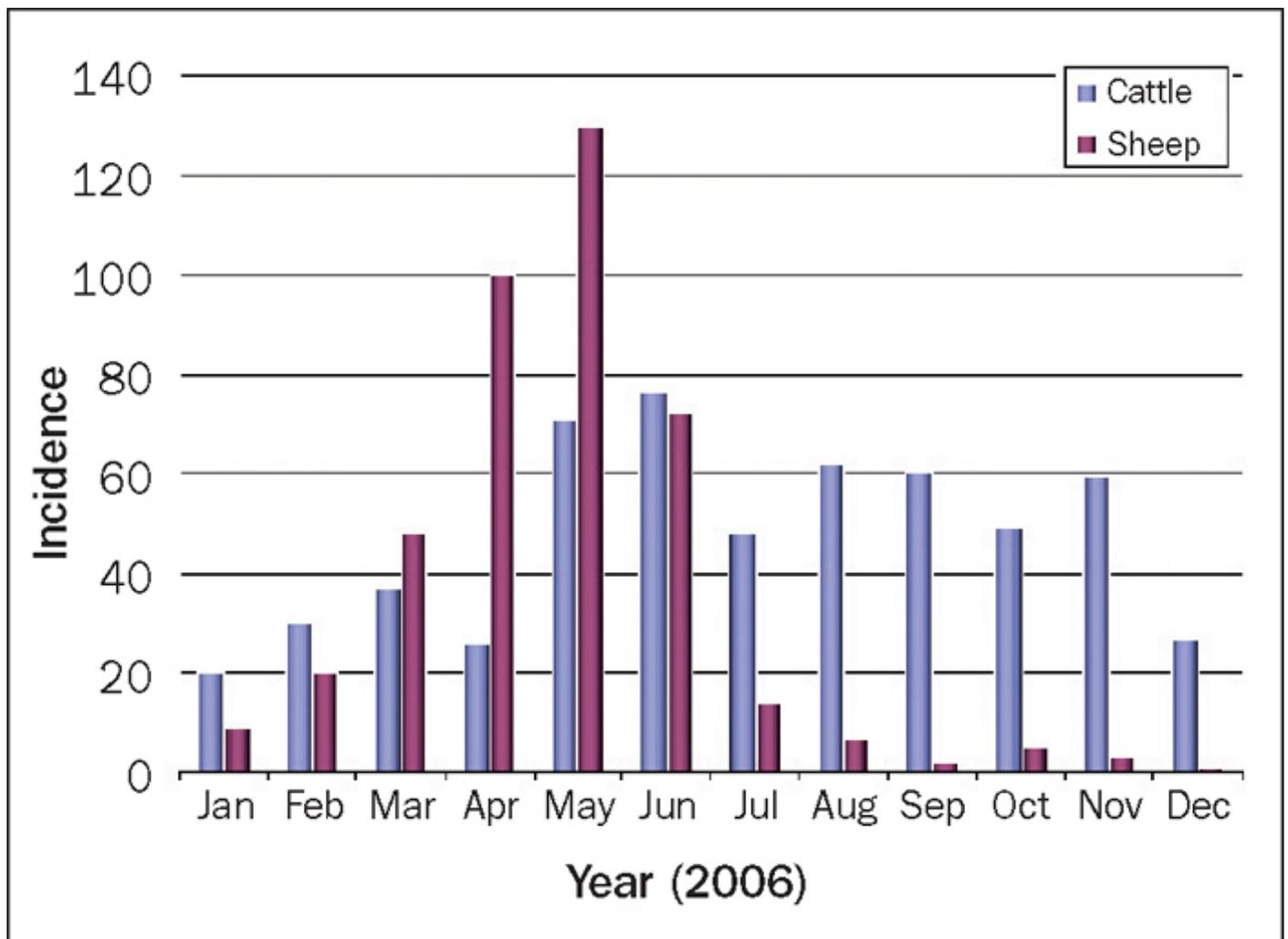


Figure 2. Incidence of coccidiosis in cattle and sheep in the UK as a percentage of diagnosable submissions. Based on data in VIDA, Table 2 – Cattle, and Table 4 – Sheep: 2006 monthly trend.

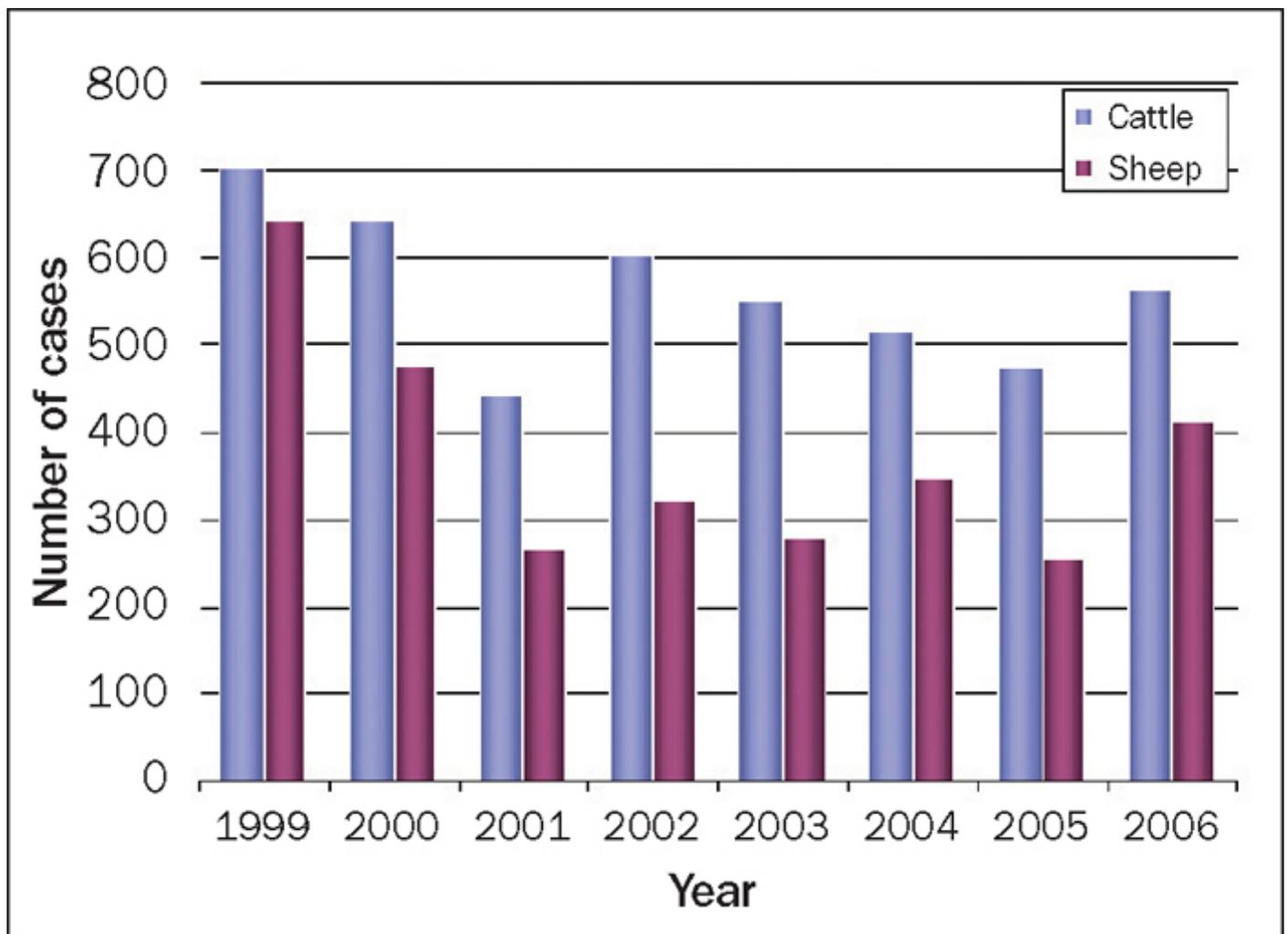


Figure 3. Incidence of coccidiosis in cattle and sheep in the UK as a percentage of diagnosable submissions. Based on data in VIDA Table 3 – cattle, and Table 5 – sheep: 1999-2006 yearly trend.

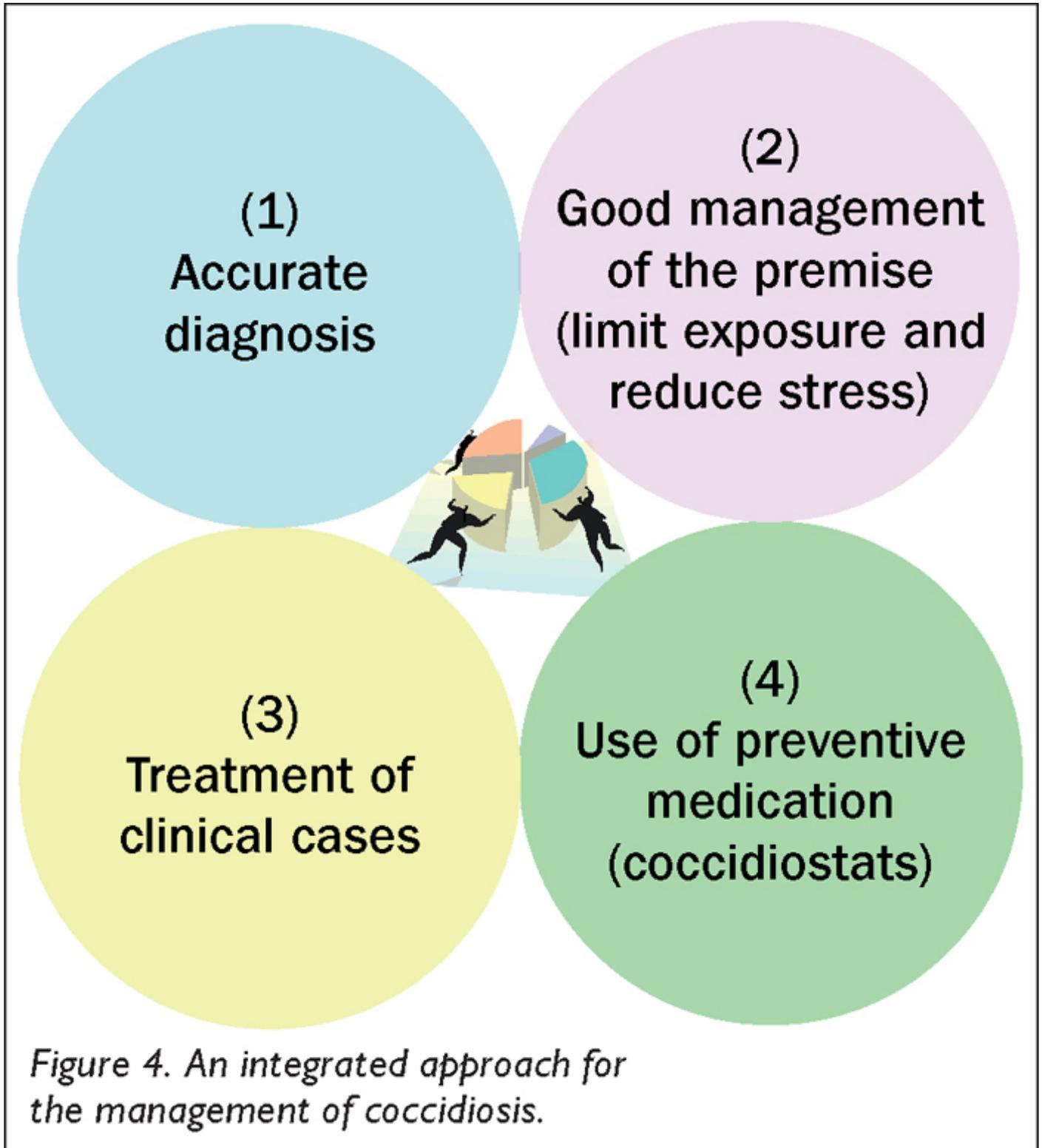


Figure 4. An integrated approach for the management of coccidiosis.