Subclinical coccidiosis: continuing major challenge to cattle industry

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ADAM MARTIN examines how this parasitic disease persists in being a problem for vets and cattle farmers

Most veterinary surgeons who perform a significant amount of work on cattle will, unfortunately, be all too familiar with coccidiosis in calves.

Classically, affected calves present with a bloody scour that is often associated with tenesmus. However, as with many diseases, this textbook description represents a minority of cases.

The disease causes significant economic loss: worldwide it has been conservatively estimated to cost cattle farmers more than £400m annually. The costs of treatment and fatalities are believed to represent a fraction (seven to 20 per cent) of the costs associated with clinical disease. However, it is believed that the economic losses associated with subclinical coccidiosis in cattle may be up to 20 times greater than those associated with clinical disease.

Life cycle

The *Eimeria* species that parasitise cattle have a three-stage indirect life cycle. Infection occurs by the faeco-oral route. After ingestion of the parasite in the form of an infectious (sporulated) oocyst, digestive enzymes begin to break down the parasite's protective coat and activate the sporozoites it contains as it is transported to the lower duodenum and jejunum, where development begins.

After entering intestinal cells the parasite undergoes asexual fission (merogony), which produces merozoites. As the merozoites mature, the cell in which they are developing ruptures, releasing the meronts into the gastrointestinal lumen once again. Typically, two cycles of merogony occur before the parasite enters the next distinct stage of development, gamogony, which, once again, occurs inside the cells of the intestine.

Gamogony, as the name implies, constitutes the sexual development stage in the parasite's life cycle. Typically, this stage in the life cycle occurs in the lower ileum, caecum or colon of the host. As merozoites (produced from the final stage of merogony) enter the cells they mature into macrogametes (analogous to mammalian ova) and microgametes (analogous to mammalian spermatozoa). Microgametes are flagellated and, after release from the host's intestinal cells, migrate towards, and enter, a cell containing a macrogamete. Fusion between the two gametes occurs, resulting in the production of a zygote. As the zygote is formed, an impermeable cyst wall forms around the cell, now known as an oocyst. This cyst wall has important implications in the epidemiology of infections.

The oocysts are shed from the host cell into the lumen of the gastrointestinal tract, where they pass from the animal in its faeces. Having being excreted, sporogony, the final stage of development, occurs. This can be thought of as the ripening of the oocyst – the period in which the immature parasite develops into its infective form. In total, completion of the life cycle takes between 14 and 21 days, depending on the species involved and the environmental ambient temperature.

Epidemiology

At least 13 different *Eimeria* species have been identified as infecting cattle. However, only two, *E bovis* and *E zuernii*, are regarded as primary causes of life-threatening coccidiosis. Two other species are referred to as moderately pathogenic: *E auburnensis* and *E alabamensis*. There is little evidence that the remaining species are capable of producing clinical disease, although their role in subclinical disease is difficult to define.

The parasites are ubiquitous and have been shown to be present on all farms. *Eimeria* are capable of surviving exogenously for more than a year in a wide variety of environmental conditions. This fact, combined with the current distribution of the parasite, make eradication a distant, if not unobtainable dream. Consequently, it is reasonable to assume that all calves will be exposed to the parasite at some point in their lives.

Despite this, the prevalence of infection varies considerably between farms. Recently published data from a trial set up to monitor *Escherichia coli* 0157 found calves on 36 out of 57 farms to be infected with *Eimeria*. In total, seven per cent of the faecal samples contained coccidial oocysts. Prevalence was shown to vary between two and 48 per cent in suckler herds in Germany, while another study in Holland showed oocyst excretion to occur in 10 to 100 per cent of calves on individual farms. Clinical disease is typically seen in calves less than six months of age.

Interestingly, the prevalence of *Eimeria* infection in yearlings and calves is similar, at 46 per cent and 43 per cent respectively, although infection rates tail off rapidly after 12 months of age.

It is currently thought that acquired immunity is responsible for the decreasing levels of both infection and clinical disease in older animals, rather than it being the result of age-related changes to the digestive system.

On well-managed cattle farms, the coccidial challenge is maintained at a low level over a long period of time, allowing calves the time to develop immunity without their immune systems being overwhelmed. This means animals that are not clinically affected by disease will often shed oocysts in their faeces. Indeed, adult animals can act as reservoirs of infection by shedding large numbers of oocysts over a long period of time without being clinically diseased. During this period, dynamic equilibrium is reached between the parasite, the host and the environment.

Pathogenesis

The obligate intracellular stages of the parasite's life cycle necessitate the colonisation of the epithelium and *lamina propria* of the small intestine. Merogony and gamogony both occur intracellularly and these processes damage the epithelial and underlying cells – resulting in death of the cells and, potentially, the sloughing of significant proportions of the intestinal mucosa. This can result in intestinal ulceration, villous atrophy, gland and crypt dilatation and tissue necrosis.

This pathology affects the ability of the calf to digest and absorb ingesta efficiently and, if the compensatory mechanisms of the low gastro-intestinal tract are overwhelmed, clinical disease will result. The compensatory mechanisms are considerable. Consequently, animals with relatively high coccidial counts may not show the classic sign of disease that is scour. However, it would be incorrect to assume that, just because an animal was not scouring, its digestive efficiency and the general wellbeing of the calf were not compromised if the level of infection is considerable.

A number of studies have compared the growth rates of groups of calves treated prophylactically for coccidiosis to untreated control groups. Results between the studies have been variable but some studies have reported improved daily liveweight gains of 250g. In addition to reduced feed conversion efficiency, infected calves suffer from a reduction in voluntary feed intake. Reduced feed intakes have been reported to last more than 10 weeks in experimentally infected animals.

One problem with the disease is that it generally affects groups and, as such, individuals that are failing to thrive cannot readily be identified. Therefore, if calves are not regularly weighed, considerable losses could go undetected as the whole group looks the same. Even when poor growth rates are detected, many producers will blame the feed rather than ruling out any underlying disease problem. After all, why show the calves to a vet if they are not showing any signs of disease?

It is important that calf health and performance is periodically incorporated into regular herd visits if opportunities to improve health, and market the practice, are not to be missed. It is also important that, when addressing the calves, the person in charge of calf husbandry is present. Commonly, problems will be overlooked because the vet speaks to the individual responsible for the cows and not the calves. Do you know who feeds the calves every day? When did you last discuss the performance of the calves with that person?

Discussion

Given that all animals are likely to encounter coccidia at some time in their life, there is some discussion about what level of infection should be regarded as subclinical. Furthermore, experience in the poultry industry has shown that resistance can rapidly emerge when anticoccidials are used routinely to maximise growth rates. As vaccination remains a distant prospect, the practitioner must balance the need to achieve maximal growth rates with the responsible use of medicines.

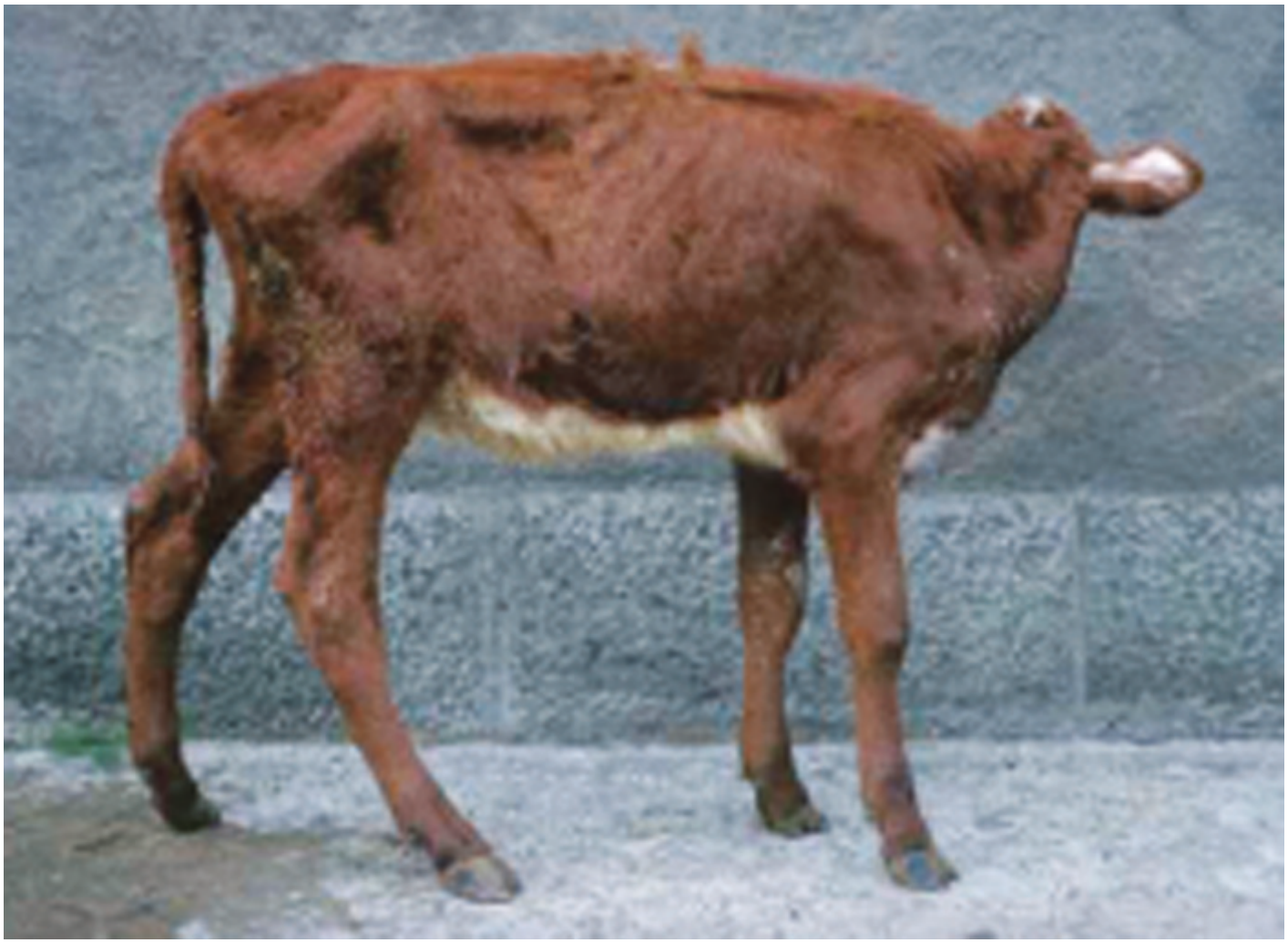
Husbandry changes, such as reduced stocking densities and improved hygiene, enable the majority of farms to overcome problems with coccidia. However, these are time-consuming methods, which may be met with resistance by producers. While it is reasonable to use medication in the short term to control a problem, more sustainable solutions should be sought in the longer term.

Anecdotally, problems with coccidiosis seem to appear at a time when calves are stressed. The type of stress is not necessarily important – it could be housing, feed, climate or disease related. It is likely that the stress causes a degree of immunosuppression, which alters the dynamic equilibrium between the host's immune system, the parasite and the environment sufficiently to cause disease. Consequently, control of immunosuppressant pathogens, such as bovine viral diarrhoea virus at the herd level, should also form part of the management strategy to control coccidiosis.

Conclusion

Unfortunately, as farms get bigger, stocking densities and margins get tighter and, as such, coccidiosis, in both the clinical and subclinical forms, is likely to continue to represent a major challenge to the cattle industry.

Good husbandry practices should reduce the subclinical losses caused by the disease to an acceptable level. However, this will require considerable effort on the part of both farmer and veterinary surgeon. Signing a prescription for prophylactic medication, to be placed in feed, is the short-term, easy solution, but if this solution is overused, we may have a far larger problem in years to come as resistance to anticoccidials appears.



Calves with coccidiosis often lose condition.

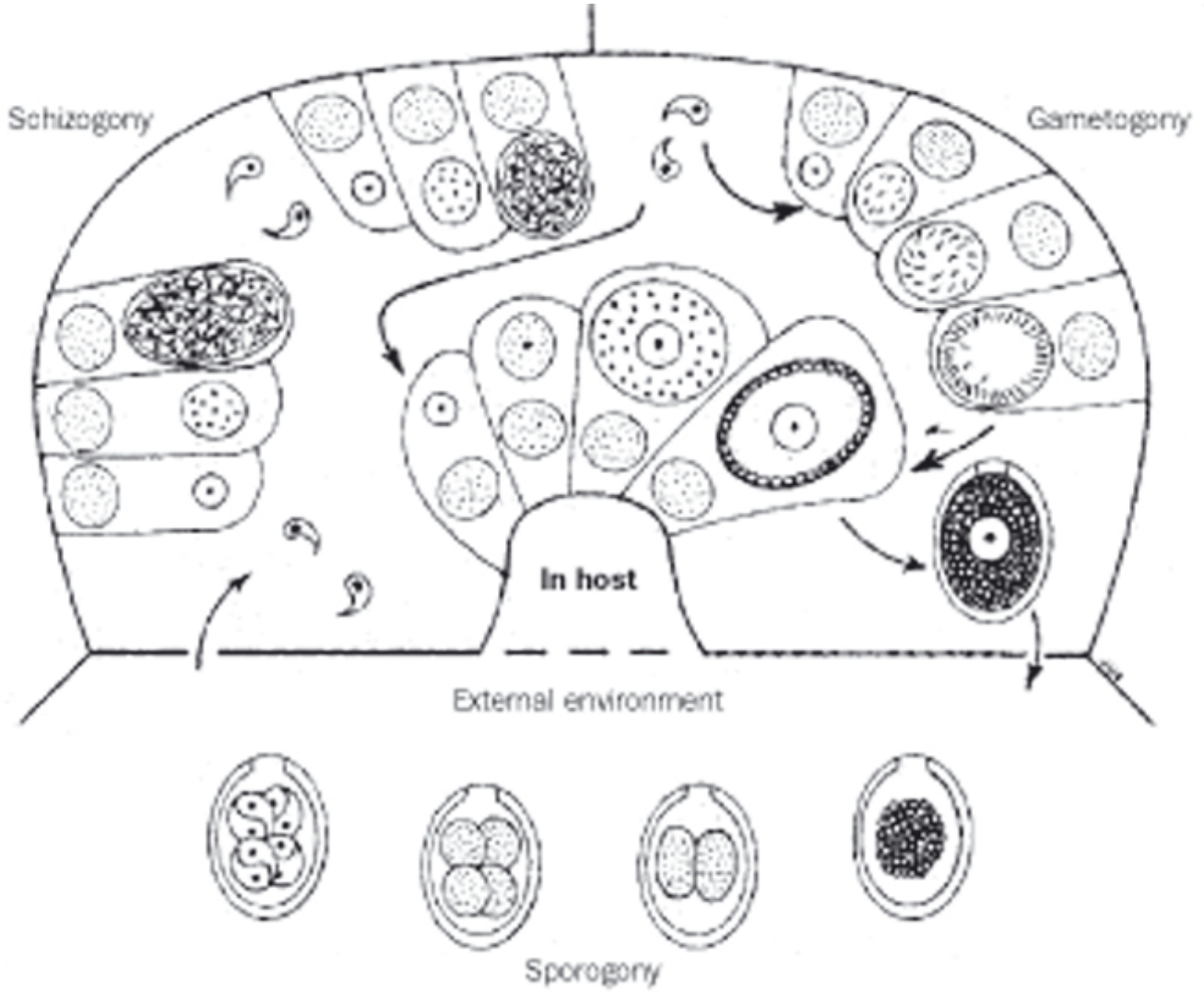


Diagram of a typical coccidian life cycle (adapted from Todd, 1975).

The prevention of coccidiosis starts at the calf's birth by ensuring it receives adequate colostrum.

A group of calves, subclinically affected with coccidiosis, will often fail to thrive.