OF the multitude of protozoan species known to modern science, the vast majority live in happy coexistence with mammals.

Indeed, many play an essential symbiotic role for animals, most notably in the process of digestion in ruminants. However, for some of these single-cell organisms, this relationship has moved from one of symbiosis to that of parasitism.

For humankind, this protozoal parasitism has given rise to several of the world’s most intractable diseases: malaria, leishmaniasis and sleeping sickness.

To the veterinary surgeon, bacterial and viral pathogens tend to be associated with the most acute disease and higher mortality rates. Protozoal diseases of farm livestock also have a capacity to be both financially damaging and difficult to manage.

Two such conditions affecting young ruminants are cryptosporidiosis and coccidiosis. While the former is of greater significance for calves and the latter for lambs, both can cause significant losses in either species.

Cryptosporidiosis
• Presentation

Cryptosporidiosis is caused by infection with the single-cell organisms of the genus Cryptosporidium. Recent advances in typing have enabled better characterisation of mammalian infections with Cryptosporidia. Although as many as 16 species of Cryptosporidium and twice as many genotypic variants have been identified, only a handful of species are pathogens of domestic animals.

Of these, C parvum (which was previously called C parvum genotype two) is by far the most important. It is known to be capable of causing significant diarrhoea in lambs, kids, calves and neonatal camelids, as well as a range of other mammalian species, including humans.

C bovis has also been recognised as a potential pathogen of animals. However, in a recent study carried out in Northern Ireland, of the 224 diarrhoeic dairy calves diagnosed with cryptosporidiosis, eight calves were infected by C bovis while C parvum was found in 213.

C hominis (previously known as C parvum genotype one) is the main species associated with human infection, although - as befits an organism with catholic tastes - it can also be found in animals from time to time.

The mass infection of humans by one of these genotypes - usually the result of contamination in water sources - occasionally leads to the appearance of Cryptosporidia in newspaper headlines. The most exceptional case of zoonotic infection was recorded in Milwaukee in 1993, when half the city’s population was exposed to oocysts following the contamination of drinking water supplies. Around 400,000 people developed signs of gastroenteritis and as many as 100 died as a result.

Like the Eimeria species of Coccidia, C parvum has a direct life cycle, with infection occurring by the faecal-oral route. The oocysts of C parvum are highly resistant to environmental conditions, and also to most commonly used farmyard disinfectants at recommended concentrations. Therefore, contamination may persist within the farm environment for long periods, particularly in standing water. Oocysts are also well suited to mechanical transmission on farm equipment or clothing and footwear, so strict biosecurity in the face of an outbreak is essential.

The initial source of these oocysts for early lambs and calves is likely to be their dams, which show evidence of a periparturient rise in excretion equivalent to that recognised in some species of helminths. Indeed, one study of dairy calves found that a risk factor in whether they went on to develop cryptosporidiosis was the length of time they were left with their mothers.

Unlike Coccidia, the oocysts of C parvum are capable of sporulation within the host gut and, as a result, are immediately infectious after being passed in the faeces. This means that as calving or lambing proceeds, the burden of infection within an environment tends to rise, as more and more neonates pass through the shedding phase of clinical or sub-clinical infection.
With no species barrier and highly persistent oocysts, *C parvum* infections may pass between lambs and calves on the same farm. Outbreaks of cryptosporidiosis can be diagnosed in calves housed in a particular shed, only for the condition to be diagnosed again when the same shed is used for lambing.

The infectious dose of *Cryptosporidium* oocysts in neonatal ruminants is very small. The oocysts hatch in the small intestine and multiplication occurs within the brush border of the enterocytes, rather than in the cytoplasm of the cell itself. This asexual multiplication step is enhanced by the production of thin-walled schizonts that immediately release eight merozoites within the intestine. These merozoites infect further enterocytes, leading to the creation of a further generation of merozoites.

The sexual phase of the life cycle results in the production of thick-walled oocysts that are excreted in the faeces of infected neonates in huge numbers. Levels of infectivity can quickly increase around young animals, so the risks of serious cryptosporidiosis outbreaks tend to rise as lambing or calving progresses.

This situation can be made worse in locations where environmental hygiene is not treated as a priority. Indeed, a recent study in north-west England showed that skimping on straw in the calving shed was a significant risk factor in the development of the disease.

In the past couple of years, cryptosporidiosis has overtaken rotavirus infection to become the most frequently identified enteropathogen in cases of calf scour in Britain. Many cases show evidence of a mixed protozoal and viral infection when outbreaks of diarrhoea in calves are investigated.

Clinical cryptosporidiosis generally affects calves during the first month of life, with the peak incidence between one and two weeks of age. In lambs, disease commonly appears in the first week to 10 days of life. For both species the infection is often asymptomatic, requiring concurrent infection or another stressor to initiate clinical disease.

The relative importance of the condition in lambs and calves can be seen in data from Britain’s veterinary investigation services ( ). While the infection is isolated from more cases of calf scour than any other, for lambs the condition tends to have lower incidence and significance. Of the 177 lambs necropsied by the Scottish Agricultural College Veterinary Services during the first four months of this year, cryptosporidiosis was diagnosed in three per cent.

Although mortality rates due to cryptosporidiosis alone are low, morbidity may be high - sometimes approaching 100 per cent in intensive situations, such as artificial rearing of orphan lambs. As a result, its principal impact is the impairment of intestinal function and a resultant lowering of animals’ performance.

Work published this year shows how *C parvum* infection affects a range of normal intestinal
functions in dairy calves. These are mostly related to the permeability and absorptive capacity of affected areas of gut. Intestinal dysfunction was seen for up to two weeks postinfection, with full gut recovery usually occurring by week three. More importantly, however, the growth of infected calves was seen to be adversely affected, even after the gut environment had returned to normal.

In clinical cases of cryptosporidiosis, the faeces tend to be pale and fluid. On occasion, this may include flecks of blood or even a more severe melaena. The condition also appears to cause significant abdominal pain, which leads, in turn, to anorexia and weight loss. In the worst cases diarrhoea is severe and acute, with the affected young appearing depressed and obviously dehydrated.

Although most neonates will clinically recover after a week to 10 days, relapses are common and affected calves and lambs may suffer a significant delay to their weight gain. Therefore, the infection can cause substantial losses to the farmer through poor production in the early weeks of life. By the time calves and lambs reach one month of age, innate resistance to the protozoa has generally developed.

Since cryptosporidiosis is so rarely fatal, diagnosis usually follows the examination of samples of diarrhoea, combined with the appropriate clinical history and presenting signs. The identification, using a microscope, of large numbers of *C. parvum* oocysts in stained samples of faeces is the clincher in most cases.

The capacity of infections to be subclinical means the presence of oocysts in faeces does not necessarily indicate disease. However, one study of 919 calves found that animals shedding *C. parvum* oocysts were five times more likely to be diarrhoeic than those with none.

• **Management**

Halofuginone lactate is licensed for prophylaxis and treatment of cryptosporidiosis in calves. Although this oral treatment is not licensed for use in lambs in the UK, it is used for this purpose in continental Europe.

In neonatal calves, treatment and prevention requires a seven-day course, with a particular emphasis on accurate calculation of the treatment dose. This is because of a particularly tight safety margin that means toxic effects may occur at twice the recommended dose, particularly in dehydrated animals.

Many authors have emphasised the significance of environmental hygiene in the control of cryptosporidiosis. The selection of a cleaning method for housing is particularly important.

The only class of disinfectant capable of inactivating *C. parvum* oocysts are ammonia-derived products; some are marketed specifically for this purpose.
Oocysts are also effectively killed by the cycle of freezing and thawing, during composting of organic material and by temperatures greater than 50ºC. For this reason, disinfection of buildings using steam cleaners following the housing of neonatal calves or lambs has much merit.

**Coccidiosis**

- **Presentation**

Just at the age when lambs and calves develop a protective immunity to cryptosporidiosis, they become more susceptible to coccidiosis, the other major protozoal disease of early life.

While *C parvum* is a single species with little host specificity and relatively minor genotypic variation, many Coccidial species are found within sheep and cattle. In the UK, 11 species of *Eimeria* are recognised to affect lambs and some 13 species have been found in calves. Thankfully, most of these are not significant pathogens.

*E crandallis* and *E ovinoidalis* are the two Coccidian species regarded as clinically serious for lambs. Both cause most of their damage in the ileum and, to a lesser extent, the caecum and colon. Infection produces local oedema, haemorrhage and villus atrophy - in the process reducing fluid absorption and causing diarrhoea.

The received wisdom is that diarrhoea containing blood suggests *E ovinoidalis* infection, while a grey and mucoid presentation indicates that *E crandallis* is the more likely diagnosis.

*E bakuensis* can cause quite obvious nodular lesions along the intestinal mucosa of lambs, but these are not thought to be of great clinical significance. For calves, clinical coccidiosis is associated with heavy infection with *E bovis* and/or *E zuernii*.

Coccidiosis is a complex problem because interactions with other parasites or pathogens, along with the animal’s nutritional status and other stressors, can exacerbate clinical disease. The important Coccidial species have a long prepatent period in lambs of 12-28 days, while in calves the prepatent period of *E bovis* is 17-22 days and *E zuernii* is 15-19 days. This means that in both species, clinical disease may be present before oocysts are passed in faeces in significant numbers. Conversely, high oocyst counts may be found in faeces as a result of infections with non-pathogenic *Eimeria* species.

Coccidiosis is recognised as one of the most important diseases of lambs and calves during the first two or three months of life. Almost all young animals become infected with Coccidia. Whether this initial infection then leads to clinical disease depends on a number of factors including husbandry, management and concurrent disease. What is universally the case is that the risk of clinical coccidiosis is greatest in unhygienic and overstocked conditions.
During the first few weeks of life, adequate intake of colostrum provides a safe period during which exposure to oocysts will produce a protective immunity without the development of clinical signs. Thus, outbreaks of coccidiosis often reflect inadequacies in the initial colostrum intake or in the subsequent nutrition.

Likewise, the stress of concurrent enteric pathogens, such as cryptosporidiosis and/or rotavirus, or other infections such as orf, can predispose the juvenile to clinical coccidiosis. Problems affecting the dam, such as lameness or mastitis, can also act as predisposing factors.

In lambs at grass, an important synergistic effect occurs with the gut worm *Nematodirus battus*. While infection with either the Coccidia or the helminth may be subclinical in a particular lamb, when the two infections are combined the effects can cause severe disease or death. In addition, such combined infections may occur while both infections are in the prepatent phase, meaning that diagnosis is dependent on the results of postmortem examination, rather than faecal analysis.

Clinical coccidiosis is most often seen in lambs aged four to eight weeks, although it can occur at other ages (1).

If lambs are not exposed to infection during the period of colostral protection they do not develop immunity and are susceptible to severe clinical disease later. This could occur if, for example, ewes receive prophylactic anti/protozoal treatment, or if lambs are not turned out to pasture until they are three to four weeks of age.

The peak disease incidence for calves occurs between one and three months of age. Very few cases of coccidiosis are reported in older calves or adults, although heavy infections may occasionally be associated with haemorrhagic scour, anorexia and dehydration in older animals. An unusual and poorly understood form of the disease in calves is described as “nervous” coccidiosis. Affected animals initially show typical enteric signs and depression, before developing convulsions and dying.

The diagnosis of coccidiosis can prove problematic. The presence in the faeces of huge numbers of oocysts may not be significant if the species represented is non-pathogenic.

In contrast, acute or even fatal disease may occur before significant numbers of oocysts of pathogenic species appear in the faeces. Diagnosis should, therefore, be based on the history and typical clinical signs. However, this must be allied to a faecal oocyst count and confirmation of a significant proportion of pathogenic species, or on the basis of postmortem findings and the examination of gut mucosal scrapes.

- **Management**

Control of coccidiosis is aimed at exposing calves and lambs to sufficient Coccidial oocysts to
engender protective immunity, but not enough to trigger clinical disease.

Indoors, high stocking densities should be avoided and levels of hygiene maintained through good drainage and the provision of adequate bedding. An “all in all out” arrangement is ideal for dairy calves, giving an opportunity for steam cleaning or treatment with a suitable disinfectant between batches.

Outdoors, the aim is to avoid the build up of infectivity in areas of faecal contamination, such as around feeders. Regularly moving such equipment may help.

The inclusion of prophylactic decoquinate in ewes’ feed can reduce the number of oocysts excreted by them in the periparturient period. However, it has been suggested that this may reduce the protective effects of colostral antibodies to lambs to a certain extent.

Decoquinate can also be added to lamb and calf feed and fed continuously for at least 28 days during the at-risk period to prevent and treat coccidiosis. Again, this is not without some potential problems: when the treatment period ends, lambs may again be at risk of infection, although this can be mitigated to some extent if they are immediately placed on clean grazing.

Diclazuril is given orally and is licensed for the treatment and prevention of coccidiosis in lambs, and as an aid to control of the disease in calves. It is particularly effective when used for indoor systems, where the best response is achieved if a whole group is treated, rather than only clinically affected individuals. The drug acts on multiple stages of the life cycle in the gut, leading to the development of rapid immunity from antigen release. This means the diarrhoea, in some lambs, becomes more severe for a few days after treatment.

The only other treatment with a specific indication for coccidiosis is licensed for use in dairy calves. Toltrazuril may now be used as a prophylactic treatment to prevent clinical signs and reduce oocysts production, on premises where *E bovis* and/or *E zuernii* have been confirmed in the past. To get the most from this oral treatment, all at-risk calves should be dosed during the prepatent period of infection and steps should be taken to improve hygiene and reduce exposure to infection.

Oral rehydration therapy and supportive measures are recommended in the worst affected animals. If required, intravenous fluids will benefit severely affected calves and may be justifiable in high-value lambs.
Figure 1. Incidents of cryptosporidiosis as a percentage of diagnosable submissions, 1999-2006.

Source: VIDA.
Figure 2. *Eimeria crandallis* and *E ovinoidalis* infection produces local oedema, haemorrhage and villus atrophy - in the process reducing fluid absorption and causing diarrhoea.
Figure 3. Incidents of coccidiosis as a percentage of diagnosable submissions, 1999-2006.

Source: VIDA.
Figure 4. Clinical coccidiosis is usually seen in lambs aged four to eight weeks, although it can occur at other ages. The peak incidence for calves occurs between one and three months of age.