Anecdotally, cryptosporidiosis seems to be becoming more commonly seen as a cause of diarrhoea in calves. However substantiating this with hard fact is difficult. The VIDA figures (which are based on samples submitted to the SAC and the VLA) show that the actual number of diagnoses has fallen over the last decade with 1746 diagnoses of cryptosporidiosis in 1995 and only 1232 in 2006. Some of this reduction may be due to the fall in submissions to the veterinary laboratories over this period. However if this is taken into account there appears to have been no real change in the percentage of digestive disease in cattle caused by cryptosporidiosis over the period between 1995 and 2006. The number of cryptosporidiosis diagnoses as a percentage of all submissions for digestive diseases has varied between 4.5 and 6% over that period. Nevertheless, the relative importance of cryptosporidiosis compared to the other major causes of calf scour, coccidiosis, E. coli K99, coronavirus and rotavirus does appear to have increased over that period (Fig. 1). In both 2005 and 2006, cryptosporidiosis was the most commonly diagnosed cause of diarrhoea in calves.

The VIDA data show a significant seasonality in the increase in isolations in the period from March to May (Fig. 2).

This seasonal trend is also seen for all other major causes of calf diarrhoea. Some of the seasonality is simply the result of there being more calves during this period, but even when this is taken into account, late winter/early spring is still the peak risk period for cryptosporidiosis. It is the build-up of infection in the environment during the winter housing period which is the underlying cause, highlighting once again the importance of hygiene in controlling disease (Fig. 3).

*Cryptosporidium parvum* is an apicomplexan protozoan parasite. This subphylum, sporozoa or Apicomplexa contain an ‘apical complex’, a structure which is thought to assist in penetration of the host cell.
The lifecycle (Diag. 1) is completed within a single host and it is in this respect that it resembles the *Eimeria* species, the cause of coccidiosis. However the pathophysiology of the two species are very different, thus it is probably best not to link cryptosporidiosis and coccidiosis too closely.

The infective dose of cryptosporidia is very low; an inoculum of five oocysts has resulted in infection. This low dose contrasts greatly with the parasite’s ability to produce huge numbers of infective oocysts; over the 7-10 day duration of a typical infection infected individuals may shed $10^{10}$ oocysts, with faecal oocysts counts in the order of a million per gram (Fig. 4).

**LIFE-CYCLE**

Oocysts excreted in the faeces by an infected host are directly infective to a new host. These thick-walled oocysts are environmentally resistant, require no maturation as they have already sporulated and therefore are immediately infective in contrast with other enteric coccidian. *Cryptosporidia* oocysts have a diameter around 5 mm, much smaller than the pathogenic coccidian (e.g. *Eimeria bovis* oocysts are 28 x 20 mm).

Four sporozoites are liberated from the oocysts upon reaching the ileum, whereupon they invade the intestinal epithelial cells. It has been shown that there is an increased predilection for cells in mitosis due to the dependence of the organism on host cell metabolites. Unusually the sporozoites develop intracellularly but extracytoplasmically into Type 1 meronts, which in turn produce 6-8 merozoites. These invade other epithelial cells and proliferate rapidly through several cycles of asexual reproduction. With the production of type 2 meronts, containing 4 merozoites, gametocytes (macrogametes and microgametes) are generated during the sexual stage completing the lifecycle by the production of oocysts. Not all the oocysts are shed in the faeces. 20% of the oocysts produced are thin-walled oocysts, which encyst in the gut lumen to release sporozoites, which directly reinfect the host. This autoinfection is an important part of the lifecycle and is one of the main differences from coccidiosis.

**CLINICAL SIGNS**

Clinical signs usually coincide with oocyst shedding, which generally begins 4-5 days post infection. Clinical disease is therefore most commonly seen in calves between one and two weeks of age. Calves are depressed, mildly febrile and anorexic. Gastrointestinal discomfort may lead to colic. A profuse watery green diarrhoea with occasional mucus and blood results. The diarrhoea is both malabsorptive and secretory. Malabsorption results from the villous atrophy, villous fusion, crypt hyperplasia, disruption of microvilli and infiltration of inflammatory cells. The result is a loss of surface area, enzymes and impaired nutrient and electrolyte transport. The secretion of a cholera-like toxin contributes to the secretory diarrhoea together with the loss of the epithelial tight junction.

**DIAGNOSIS**

Stained faecal samples are examined for oocysts. A modified Ziehl-Neelsen (acid fast) or phenol-auramine are the more commonly used stains. Recently, the use of fluorescent-antibody tests (FAT) has been investigated. So far FAT has been most commonly used in lambs, but it is likely to become the standard diagnostic test for cattle in the future. Cow side tests are available but the accuracy of the tests is not known (Bio-X Diagnostics) Fig 5.

Simply identifying oocysts is not sufficient for a diagnosis of cryptosporidiosis. The organism is found on most farms and its presence in faeces does not mean that the animal has cryptosporidiosis. This is the same for most causes of diarrhoea in calves. For
example, in one study 15% and 37% of animals excreting cryptosporidia and rotavirus respectively had diarrhoea. However 75% of those calves with both infections had diarrhoea, so clearly the combination of the two pathogens was more likely to produce clinical disease. Most outbreaks of diarrhoea in calves >1 week old involve multiple pathogens.

The likelihood of a calf infected with enteric pathogens developing diarrhoea is dependent on the infective dose, the number of different pathogens and the extent and the location of where those pathogens exert their pathogenic effect. Thus to understand and control diarrhoea, one must have a good understanding of the individual pathogens and their mode of action. Infection with one agent may not in itself cause sufficient damage to cause scouring.

This is also important for control and prevention. Controlling one pathogen effectively may prevent clinical disease even when two or more pathogens are involved. For example, controlling rotavirus with pre-calving vaccine and good colostral management may lead to the farm falling below ‘the clinical scouring threshold’, even though cryptosporidia was an important cause of disease.

TREATMENT

There is currently no effective treatment for cryptosporidiosis but the development of halofuginone lactate has significantly helped its control. Halofuginone is a synthetic quinazolinone derivative, which has cryptosporodistatic effect. It acts against the extracellular stages of the lifecycle, i.e. the sporozoites and merozoites.

Halofuginone is licensed as an oral treatment for the prevention of diarrhoea caused by Cryptosporidium parvum in newborn calves on farms with a history of cryptosporidiosis and reduction of diarrhoea due to diagnosed C. parvum, Halacur (Intervet Schering-Plough). For prevention of diarrhoea, treatment should start within 24-48 hours of birth; for reduction of clinical diarrhoea treatment should commence 24 hours before the anticipated start of diarrhoea. Treatment should continue for seven consecutive days.

A trial involving 68 treated calves and 80 placebo calves showed a significant reduction of 44% in C. parvum oocyst shedding and diarrhoea in the treated calves, compared to the placebo calves, 24 hours after the end of a seven day course of treatment (Lefay, 2001). The treatment also delayed and reduced the peak of oocyst excretion. This occurred at day 7 for most of the placebo calves and day 14 for the treated calves. The peak of oocyst excretion in the treated calves did not coincide with significant levels of diarrhoea. Indeed only 4% had signs of diarrhoea despite 78% of calves excreting oocysts. There appeared to be no significant effect on bodyweight gains and weight at weaning between treatment and placebo groups (Jarvie, 2005) (Fig. 6).

Decoquinate (a coccidiostat) has been suggested as a treatment for cryptosporidiosis. However evidence of efficacy in calves is not convincing. Seventy-five experimentally challenged calves were treated daily for up to 28 days with 2 mg/kg of decoquinate after challenge with C. parvum oocysts. Compared to controls there was no effect of the treatment on oocyst shedding or clinical signs (Moore, 2003). One field study compared the efficacy of halofuginone and decoquinate in the treatment of cryptosporidiosis. Halofuginone, unlike decoquinate, significantly reduced the excretion of oocysts on day 7 (Lellemand, 2006).

PREVENTION

Adherence to good hygienic practice is the best method of control. It is important to ensure all calving pens are clean, with the use of copious amounts of bedding changed between calvings. The calving cow should be clean to prevent any contamination from dirty flanks and udders. However, usually such measures are practically difficult to achieve. Any scouring calves should be isolated and action taken to limit spread by changing clothing and separating any feeding utensils.

Oocysts are extremely resistant to disinfection. Ammonia-based disinfectants are the only effective disinfectants available on farm for use. Temperatures in excess of 6000 degrees will kill oocysts, so high temperature pressure washers can be used to clean buildings between batches of calves.
Mixing purchased calves and homebred stock greatly increases disease risk, so if practicable, purchased calves should be kept separate for at least 7 days.

Effluents, pen leaches and faecal soiling of watercourses represent further sources of contamination. The low concentrations of chlorine used to disinfect water supplies are ineffective at destroying cryptosporidia oocysts. Filtration and UV irradiation are both effective defences against contaminated water supplies.

**VACCINATION**

Immunisation of late gestation Holstein cows with a recombinant protein of *C. parvum* will induce antibody production in colostrum-protected calves against oral challenge with oocysts. Twelve calves were challenged orally with $10^7$ oocysts at 12h of age. Immune or control colostrum was administered at 2h, 12h and 24h of age. All of the calves, which were given pooled control colostrum developed severe diarrhoea. None of the calves given immune colostrum developed diarrhoea and they produced significantly less oocysts as compared to the control calves (Perrymon, 99).

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