Coccidiosis and cryptosporidiosis: control and management options

Author : Sara Pedersen

Categories : Farm animal, Vets

Date : April 18, 2016

Coccidiosis and cryptosporidiosis have many things in common. Both are diseases caused by protozoal parasites that can lead to enteric disease in young farm animals through parasitisation of the intestinal lining.

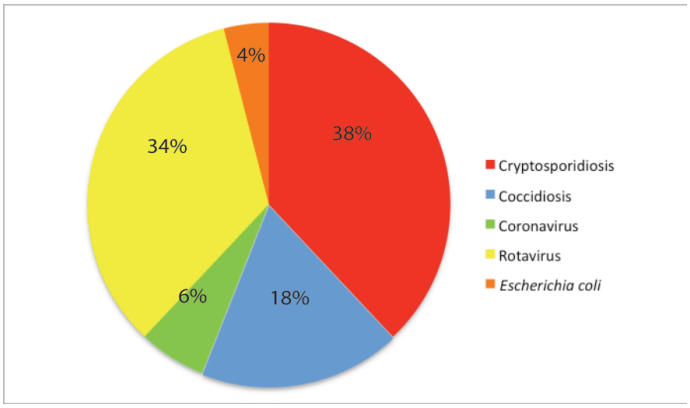

Figure 1. Pathogens causing enteric disease in neonatal calves.

Following exposure to either parasite, the outcome can vary, from asymptomatic infection to severe disease and mortality. However, more commonly, morbidity is high and mortality relatively low.

Once established in the farm environment, both parasites can be challenging to overcome, therefore reducing infection risk through specific management strategies is key.

Species involved

While many species of coccidia and cryptosporidia exist, only certain ones are pathogenic.

In the case of coccidiosis, the *Eimeria* species that can cause disease are host-specific with no cross-infection. In sheep, this includes *E ovinoidalis* and *E crandallis*, considered the most pathogenic, while *E bovis*, *E zuernii* and *E alabamensis* cause clinical disease in cattle. In comparison, only one pathogenic species is recognised as causing cryptosporidiosis in young animals – *Cryptosporidium parvum*.

Unlike *Eimeria*, *C parvum* is not host-specific, but is capable of infecting a wide range of animals, including cattle, sheep and deer. It is also zoonotic and can infect humans, causing severe diarrhoea and, therefore, must be discussed with staff when diagnosed on a farm.

While coccidiosis has been a commonly recognised cause of young ruminant enteric disease for several decades, the prevalence and awareness of cryptosporidiosis has become increasingly widespread and, in the past decade, has consistently been the main cause of enteritis in calves (APHA, 2012).

In 2012, *C parvum* was the major cause of enteric disease in neonatal calves, accounting for 38% of viable laboratory submissions, with coccidiosis accounting for a further 18% (**Figure 1**; APHA, 2013). Therefore, controlling these protozoal parasites remains a key challenge in reducing neonatal enteric disease.

Sources of infection

Figure 2a. Adult animals can act as reservoirs of infection for coccidiosis and cryptosporidiosis, with birth a major risk period for exposure to infection.

Figure 2b. Adult animals can act as reservoirs of infection for coccidiosis and cryptosporidiosis, with birth a major risk period for exposure to infection.

In cryptosporidiosis and coccidiosis, the infective stage is the oocyst, which is shed in the faeces of an infected animal. Crucially, in the case of cryptosporidiosis, the oocyst is immediately infective, with fewer than 100 oocysts (sometimes as low as 10) required to establish infection.However, in the case of coccidiosis, the oocyst is not infective until it has undergone sporulation. Specific conditions are required for this to occur, including temperature, oxygen levels and humidity. For example, the *Eimeria* species affecting sheep take one day to five days to sporulate at the optimum temperature of 20°C. Therefore, unlike cryptosporidiosis, climate and weather is a key component when considering disease risk factors.

The thick-walled oocysts of *C parvum* and *Eimeria* species are highly resistant in the environment and can persist for several months, allowing infection to pass from one group of animals to the next or, in the case of coccidiosis, from one year to the next. Reducing environmental infection pressure is a key component of managing both of these diseases, yet this is challenging due to oocysts being resistant to many of the commonly used disinfectants at the recommended concentrations.

Many different disease transmission routes exist – directly between shedding individuals via faecal-oral contact, or indirectly; for example, via environmental contamination.

Adult animals can shed both coccidia and C parvum oocysts, thus infecting the lambing or calving areas (**Figure 2**). Transmission can also occur directly through exposure at the point of birth or through suckling dirty teats. Contamination of feeding equipment, transportation or personnel are

also risk factors for transmission.

In theory, transmission of *C parvum* could also occur via contaminated water sources and other animals, such as sheep or deer, which can become infected, albeit at a lower risk. Due to *C parvum* being not host-specific, unlike *Eimeria* species, mixed farms can be at a greater risk of infection if there is a crossover between the housing of cattle and sheep. Lambs tend to have a higher prevalence of *C parvum*, with subclinical infections more common in comparison to calves; however, they can still contribute to environmental contamination (Wells et al, 2015).

Outcome of infection

Clinical cryptosporidiosis is most commonly seen in young calves at one week to three weeks of age and in lambs at three days to seven days of age. In comparison, coccidiosis tends to occur at around four weeks to eight weeks of age in calves and lambs. However, disease can be seen outside of these times, particularly in immunocompromised animals.

As with most diseases, in both cases, the outcome of infection is multifactorial and covers a wide spectrum of disease, from animals showing no signs of infection to those with severe clinical disease.

Coccidiosis

Our understanding of the disease process in coccidiosis is more extensive than that of cryptosporidiosis (**Figure 3**).

Infections predominantly involve pathogenic and non-pathogenic species. When a naïve animal ingests low numbers of pathogenic occysts, the infection usually does not result in clinical disease as damaged intestinal cells are quickly replenished. However, if large numbers are ingested the damage is more widespread and clinical signs result.

Damage to the intestinal lining is the result of the sporozoites released from the oocyst invading the intestinal cells and subsequently multiplying (schizony) and rupturing the cells, releasing merozoites that infect further cells.

One coccidial oocyst can result in up to 50 million intestinal cells being destroyed, resulting in a reduction in the height of the epithelial cells and also a decrease in the extent of the brush border of the intestine (Mundt et al, 2005; **Figures 4** and **5**).

As a result, there is a reduction in the surface area available for absorption and a decreased feed efficiency (Taylor et al, 2007), leading to diarrhoea, weight loss and ill-thrift.

Although clinical and subclinical coccidiosis occur, the latter is more common, with 95% of cases in

calves (Laven, 2003). However, regardless of the type of disease, even once animals are treated, longer-lasting effects occur, with reports of reduced feed intake for a month or more after infection.

In dairy heifers, the effects on growth rates delay the onset of puberty and first service, leading to a greater age at first calving (Veronesi et al, 2013), with estimated losses of milk production of up to 108 litres in their first lactation (Andrews, 2008).

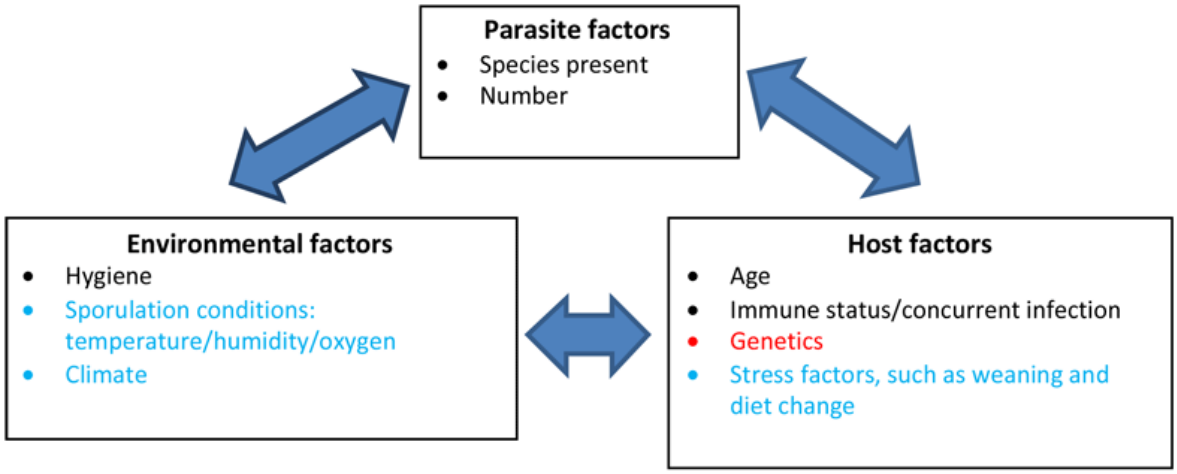

Figure 3. Multiple factors are involved in the development of cryptosporidiosis (red) and coccidiosis (blue). Common factors are shown in black.

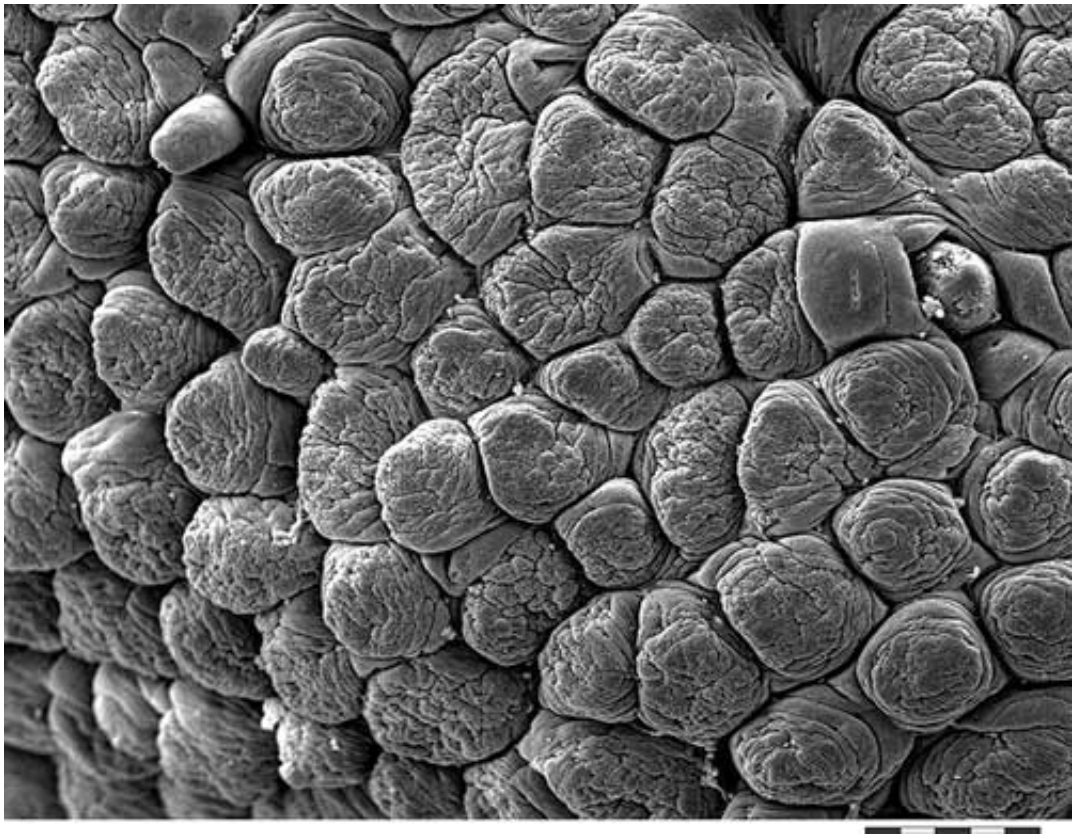

Figure 4. Normal, healthy intestinal lining. Image: © Bayer Animal Health.

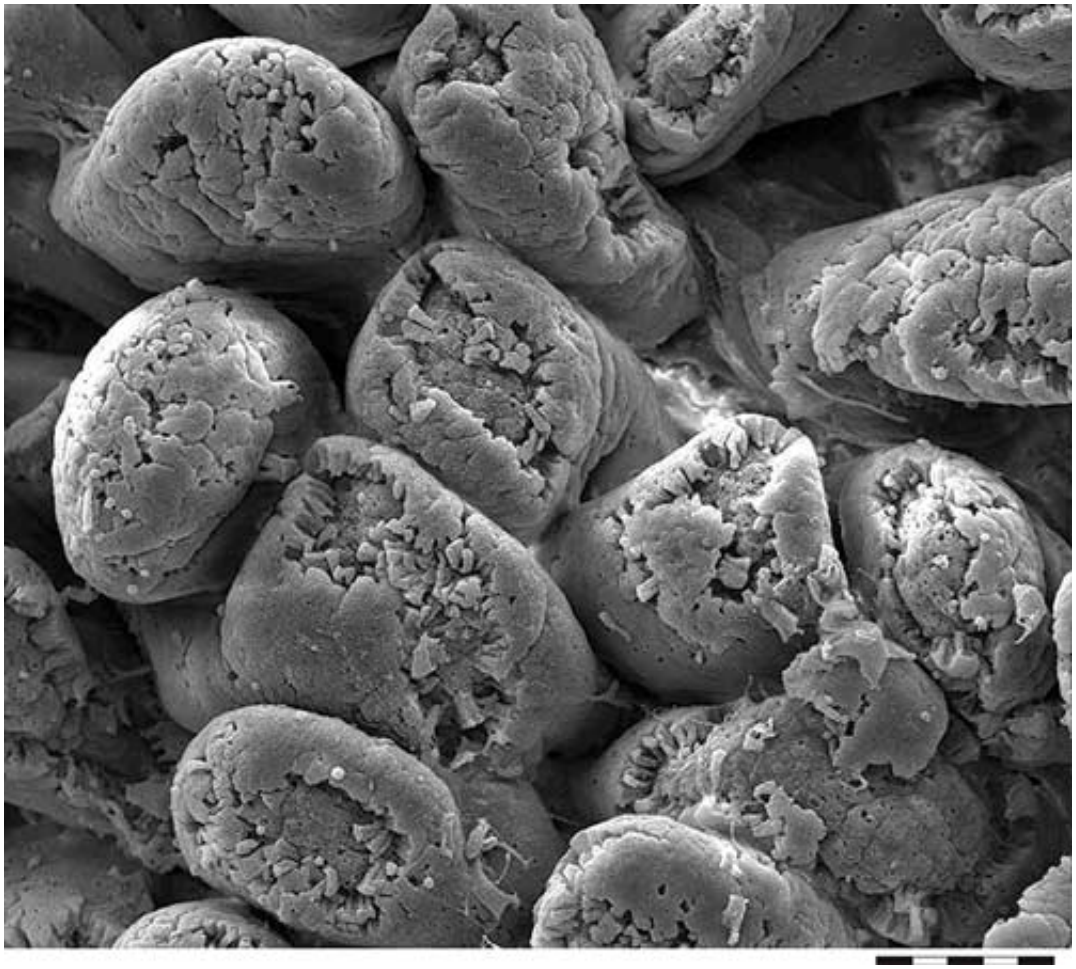

Figure 5. Damaged intestinal lining due to coccidiosis. Image: © Bayer Animal Health.

Cryptosporidiosis

While the life cycle of *C parvum* is less complex, it has the ability to multiply rapidly in its host, with each oocyst ingested having the potential to lead on to the production of billions more in a short time frame. Following ingestion of the oocysts, four sporozoites are released in the intestinal lumen, which invade the epithelial cells, multiply and develop into oocysts, which are then passed in the faeces. Completion of the life cycle takes two days to seven days.

Additionally, “auto-infection” may occur through the production of thin-walled oocysts, which burst before they are excreted, increasing the multiplication rate further.

Our full understanding of all the factors involved in determining the outcome of infection remains

largely unknown due to the difficulties of replicating the life cycle in vitro (Hotchkiss et al, 2015). However, understanding is based on the interaction between host, environmental and pathogen factors, including some overlap with coccidiosis (**Figure 2**).

Infection does not necessarily result in disease, as with coccidiosis, and it has been shown, on farms where cryptosporidiosis is present, 100% of calves may shed oocysts during the first weeks of life and can occur in apparently healthy calves (O'Handley et al, 1999; Hotchkiss et al, 2015).

When infection does lead to clinical signs, in calves these include diarrhoea, anorexia, colic and a mild fever. In lambs, profuse yellow/green diarrhoea is present and they rapidly become dull and reluctant to follow the dam.

In the majority of cases, infections are mild and self-limiting; however, when the challenge is severe enough, it can result in severe dehydration and mortality. It is important to note, even when the diarrhoea has resolved, the animal will continue to shed oocysts for a number of days afterwards.

Treatment

No "cure" exists for either disease as clinical signs are only seen once the damage to the intestinal lining has already taken place and the long-lasting effects are irreversible. Licensed treatments are available for both diseases; however, it is important treatment of the clinically affected animal is not limited to these alone.

Where dehydration is evident, any electrolyte and fluid imbalances must be corrected and additional fluids administered to account for ongoing losses during recovery.

For coccidiosis, toltrazuril and diclazuril can be used for treating lambs and calves showing clinical signs and halofuginone lactate for cryptosporidiosis in calves (no licenced treatments exist in lambs). All are also licensed for prophylactic use.

Due to the nature of both coccidiosis and cryptosporidiosis, and the rapid multiplication of oocysts in the environment from infected animals, it is important to isolate affected animals, as well as remove at-risk animals from the source of infection. Where it is likely a whole group has been exposed, it may be prudent to treat all at-risk animals. This is particularly important for coccidiosis, as failure to treat all animals in a group can lead to reinfection.

Prophylactic treatment

Coccidiosis

Table 1. Products licensed for the treatment and prevention of coccidiosis

Active ingredient		Toltrazuril (oral suspension)	Decoquinate (premix)	Diclazuril (oral suspension)
Lambs	Treatment	Single dose 0.4ml/kg bodyweight	1mg decoquinate/kg bodyweight daily for at least 28 days	1ml/2.5kg bodyweight
	Prevention	Single dose 0.4ml/kg bodyweight. Animals should be treated during the prepatent period	0.5mg decoquinate/kg bodyweight for at least 28 days	1ml/2.5kg bodyweight at 4 to 6 weeks or at time coccidiosis normally expected. Second treatment may be required
	Meat withdrawal	42 days	0 days	0 days
Calves	Treatment	Single dose 3ml/10kg bodyweight	1mg decoquinate/kg bodyweight daily for at least 28 days	–
	Prevention	Single dose 3ml/10kg bodyweight. Animals should be treated during the prepatent period	0.5mg decoquinate/kg bodyweight for at least 28 days	1ml/2.5kg bodyweight as a single dose 3 weeks after moving to a potentially high risk environment
	Meat withdrawal	63 days	0 days	0 days

Decoquinate can also be fed to ewes in the pre-lambing period to reduce oocyst shedding and thus environmental contamination. Not all toltrazutil products are licensed for sheep.

Table 1. Products licensed for the treatment and prevention of coccidiosis.

Decoquinate, totrazuril and diclazuril are licensed coccidiocides for prophylactic treatment. **Table 1** outlines the recommended treatment regime for each.

Decoquinate works very early in the life cycle, targeting sporozoites as they infect the animal. As a result, it is not as effective in animals already infected and must be fed prior to exposure. It also takes longer for the animal to develop immunity during treatment; therefore, if the treatment is not continued for long enough, animals can succumb to disease when removed from the medicated feed.

In comparison, diclazuril and toltrazuril target coccidia during the intracellular stages of the life cycle, such that the extracellular stages are still able to stimulate immunity. Treatment is recommended during the prepatent period once the animal has already been exposed, allowing immunity to develop without the onset of clinical signs or shedding of oocysts.

Cryptosporidiosis

The only licensed prophylactic treatment for cryptosporidiosis in calves is halofuginone lactate. Administered orally for seven days from birth, it does not prevent infection, but aims to stop the formation of oocysts in the intestinal lining, thus reducing excretion and environmental contamination.

As discussed by Hotchkiss et al (2015), mixed reports exist on its effectiveness in studies and anecdotally from experience on farms. Trotz-Williams et al (2011) reported a reduction in oocyst shedding and mortality, despite treatment having no effect on the incidence of diarrhoea, whereas further studies have shown a reduction in the severity of disease (Joachim et al, 2003) and mortality (Naciri et al, 1993).

The inconsistent responses to prophylactic treatment, impracticalities and risk associated with prolonged dosing of multiple calves (particularly in suckler herds) and potential toxicity from overdosing (double the dose rate is toxic) mean halofuginone lactate should not be relied on as a “sticking plaster” at the expense of management changes. Where outbreaks have occurred and

prophylactic treatment is necessary, it should not be used in isolation, but as part of a cryptosporidiosis management plan.

Management

Complete eradication of coccidia and cryptosporidia is virtually impossible due to their ability to persist in the environment and the fact adult animals can act as reservoirs of infection. The focus should be on reducing risk of exposure and ensuring neonatal animals are more resilient to the challenges they face.

Hygiene and disinfection

Since infection is via the faecal-oral route, every effort must be made to reduce environmental contamination of the calving and lambing areas and also the housing areas of young animals. Frequent and effective disinfection is required to reduce the risk of one group of animals passing on infection to the next.

Removing the calf from the dam as soon as possible after calving also reduces the risk of infection. This is particularly important in cryptosporidiosis control since calves left with their dam for more than one hour are at a 39% greater risk of picking up infection (Trotz-Williams et al, 2007).

Coccidia and cryptosporidia oocysts are very resistant to the commonly used disinfectants at the recommended concentrations. Instead, steam cleaning is recommended and highly effective if carried out correctly after gross decontamination of the area.

Panel 1. Disinfectants suitable for cryptosporidium control (Hotchkiss et al, 2015)

- 2% to 3% Kenocox: kills 99% of oocysts after 2 hours contact time.
- 2% to 4% Neopredisan: kills 99% of oocysts after 2 hours contact time.
- 10% Ox-Virin: reduced oocyst infectivity after 1 hour contact time.
- 3% hydrogen peroxide: reduced oocyst infectivity after 4 minutes.

Panel 1. Disinfectants suitable for cryptosporidium control (Hotchkiss et al, 2015).

Ammonia-based disinfectants are also effective against coccidial and cryptosporidial oocysts when used at the correct concentration. However, due to the caustic fumes, they can only be used in buildings where animals are not housed at the time.

Other examples of disinfectants effective against *C parvum* oocysts are given in **Panel 1**.

Additional measures that can be taken to reduce the risk of faecal contamination of water and feed troughs include regular cleaning or moving regularly when outside. Contamination can also be reduced by raising feed troughs off the ground.

Housing

If animals are housed inside then bedded areas must be kept clean and dry and the building well-ventilated to avoid optimal conditions for sporulation of coccidial oocysts.

Bedding is also a significant factor in the risk of the development of cryptosporidiosis, with calves housed on bedding 11cm to 15cm deep being at a lower risk of infection compared to those on beds 0cm to 5cm deep (Brook et al, 2008).

Overcrowding, both at pasture and when housed, should also be avoided as this leads to additional stress and also allows contamination to build up more rapidly, increasing the risk of infection.

Older animals are a risk factor for spreading infection to younger ones, so avoid mixing age groups, particularly animals at peak shedding age with neonates.

If lambing or calving over a prolonged period, young animals should be turned out on to clean pasture where possible.

Summary

Coccidiosis and cryptosporidiosis remain challenges during the lamb and calf rearing periods.

Their ability to persist in the environment and carry infection between groups of animals, coupled with the fact the infective oocysts are resistant to many commonly used disinfectants, makes them difficult diseases to tackle.

However, while pharmacological control is available it must not be used as a sticking plaster or at the expense of the implementation of improved hygiene and management measures.

References

- Andrews T (2008). The cost of coccidiosis, *Vet Prac* **40**: 28-29.
- APHA (2013). Veterinary Investigation Diagnosis Analysis (VIDA) report, 2012.

- Brook E, Anthony Hart CA, French N and Christley R (2008). Prevalence and risk factors for *Cryptosporidium* spp infection in young calves, *Vet Parasitol* **152**(1-2): 46-52.
- Hotchkiss E, Thomson S, Wells B, Innes E and Katzer F (2015). Update on the role of cryptosporidiosis in calf diarrhoea, *Livestock* **20**(6): 316-320.
- Joachim A, Krull T, Schwarzkopf J and Daugschies A (2003). Prevalence and control of bovine cryptosporidiosis in German dairy herds, *Vet Parasitol* **112**(4): 277-288.
- Laven R (2003). Coccidiosis in cattle, *NADIS Cattle Disease Focus*.
- Mundt HC, Bangoura B, Rinke M, Rosenbruch M and Daugschies A (2005). Pathology and treatment of *Eimeria zuernii* coccidiosis in calves: investigations in an infection model, *Parasitol Int* **54**(4): 223-230.
- Naciri M, Mancassola R, Yvore P and Peeters JE (1993). The effect of holfuginone lactate on experimental *Cryptosporidium parvum* infections in calves, *Vet Parasitol* **45**(3-4): 199-207.
- O'Handley RM, Cockwill C, McAllister TA, Jelinski M, Morck DW and Olson ME (1999). Duration of naturally acquired giardiosis and cryptosporidiosis in dairy calves and their association with diarrhoea, *J Am Vet Med Assoc* **214**(3): 391-396.
- Taylor MA, Coop RL and Wall RL (2007). *Veterinary Parasitology* (3rd edn), Blackwell, Oxford: 874.
- Trotz-Williams LA, Wayne Martin S, Leslie KE, Duffield T, Nydam DV and Peregrine AS (2007). Calf-level risk factors for neonatal diarrhoea and shedding of *Cryptosporidium parvum* in Ontario dairy calves, *Prev Vet Med* **82**(1-2): 12-28.
- Trotz-Williams LA, Jarvie BD, Peregrine AS, Duffield TF and Leslie KE (2011). Efficacy of halofuginone lactate in the prevention of cryptosporidiosis in dairy calves, *Vet Rec* **168**(19): 509.
- Veronesi GF, Nisoli L, Diaferia M, Falcini R, Ficola E and Fioretti DP (2013). Influence of a metaphylactic treatment with Baycox Bovis on the reproductive performances of Friesian heifers: a preliminary study, *Parasitol Res* **112**(6): 2,137-2,142.
- Wells B, Shaw H, Hotchkiss E, Gilray J, Ayton R, Green J, Katzer F, Wells A and Innes E (2015). Prevalence, species identification and genotyping *Cryptosporidium* from livestock and deer in a catmint in the Cairngorms with a history of a contaminated water supply, *Parasit Vectors* **8**(1): 66.