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Review

The public health and clinical significance of *Giardia* and *Cryptosporidium* in domestic animals

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Abstract

Giardia and *Cryptosporidium* are common enteric parasites of domestic animals, particularly dogs, cats and livestock. Their occurrence is of potential significance from both clinical and public health perspectives yet, until recently, confusion over the taxonomy of these organisms prevented a clear understanding of the epidemiology of infections with both *Giardia* and *Cryptosporidium*. The recent application of molecular epidemiological tools has helped to resolve taxonomic issues, allowing cycles of transmission to be determined. In addition, advances have been made in elucidating mechanisms associated with pathogenesis, whereas only limited progress has been achieved in the areas of chemotherapy and prophylaxis.

Keywords: *Giardia*; *Cryptosporidium*; Dogs; Cats; Livestock; Molecular epidemiology; Public health; Zoonoses

Introduction

Giardia and *Cryptosporidium* are the most common enteric parasites of domestic animals, including livestock, dogs and cats (Fayer, 2004; Thompson, 2004; Thompson and Monis, 2004). Both are also common parasites of humans and wildlife. An important aspect of the epidemiology of infections with both parasites is to understand the host range of different species and strains/genotypes, how they are maintained in nature, and the potential for cross-transmission. This is particularly important in determining the zoonotic potential of *Giardia* and *Cryptosporidium* infections in domestic animals.

Both parasites are maintained in a variety of transmission cycles that can operate independently, but what is not understood are the circumstances under which such cycles may interact and result in zoonotic transfer (Fig. 1). In this respect, establishing a correct taxonomy for both parasites has provided the basis for better understanding the links between infections in domestic animals and humans (Table 1). It is only recently with the advent of molecular typing tools that both the taxonomy and epidemiology of infections with *Giardia* and *Cryptosporidium* are now being resolved.

The taxonomy of *Giardia* and *Cryptosporidium* is summarised in Table 1 and has been extensively reviewed (Monis and Thompson, 2003; Thompson and Monis, 2004; Xiao et al., 2004; Caccio et al., 2005). With both *Giardia* and *Cryptosporidium*, a large number of species and genotypes are now recognised that differ principally in their host range. Some species and genotypes appear to be restricted to particular species or types of hosts (e.g. *Giardia psittaci* and *Cryptosporidium baileyi* in birds; *Cryptosporidium canis* in dogs; *Giardia* assemblage E [*Giardia bovis*] in livestock; Table 1), whereas others have broad host ranges, including humans (e.g. *Giardia duodenalis*; and *Cryptosporidium parvum*; Table 1), and are therefore of zoonotic significance. In addition to *C. parvum*, several

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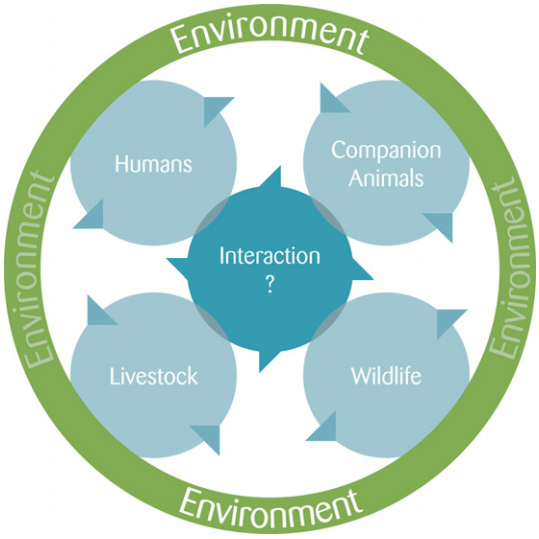

Fig. 1. The most important cycles of transmission for maintaining *Giardia* and *Cryptosporidium*. Although there is evidence for interaction between the cycles, there is uncertainty regarding the frequency of such interaction. Both *Giardia* and *Cryptosporidium* can be transmitted directly from host to host but infective stages can remain infective in the environment serving as a reservoir of infection and, in the case of water, as a vehicle of transmission.

other species and genotypes of *Cryptosporidium* have occasionally been recorded in humans, but usually in paediatric cases associated with immunosuppressive disorders or other factors that may predispose to lowering host resistance (Thompson et al., 2005).

Life cycle

The life cycles of both *Giardia* and *Cryptosporidium* are direct and the infective stages of both parasites, the cysts/oocysts, are encysted when released in the faeces and are immediately infectious (Kirkpatrick, 1987). Cysts and oocysts remain infectious for months in cool, damp areas and rapidly accumulate in environments, such as refuges, kennels, catteries and dairies. They can also survive in water for considerable periods. The host ingests the cyst/oocyst stage of the parasite and, following exposure to gastric acid, gastric and pancreatic enzymes, excystation occurs in the duodenum and the trophozoites/sporozoites are released. The life cycles of each parasite include asexual phases of proliferation on the brush border villous epithelium of the mucosal surface, in addition to a sexual phase of reproduction in *Cryptosporidium* that also exhibits an unusual intracellular phase of development in its life cycle (Thompson et al., 2005).

The prepatent period of *Giardia* in dogs and cats is 5–16 days and cyst shedding is often cyclical (Leib and Zajec, 1999). The prepatent period ranges from 7–8 days in calves (Taminelli et al., 1989) to 6–10 days for goats (Koudela and Vitovec, 1998) and 10–21 days for sheep (Taminelli et al.,

Table 1
Species and genotypes of *Cryptosporidium* and *Giardia*

Cryptosporidium		*Giardia*	
Species	Major hosts	Species	Major hosts
C. muris	Rodents	*G. duodenalis* (=Assembiage A)	Humans and other primates, dogs, cats, livestock, rodents and other wild mammals
C. parvum	Cattle and other livestock, humans	*G. enterica*[a] (=Assemblage B)	Humans and other primates, dogs
C. melegridis	Birds	*G. agilis*	Amphibians
C. wrairi	Guinea pigs	*G. muris*	Rodents
C. felis	Cats	*G. psittaci*	Birds
C. serpentis	Reptiles	*G. ardeae*	Birds
C. baileyi	Poultry	*G. canis*[a] (=Assemblage C)	Dogs
C. saurophilum	Lizards	*G. cati*[a] (=Assemblage F)	Cats
C. galli		*G. bovis*[a] (=Assemblage E)	Cattle and other hoofed livestock
C. andersoni	Cattle	*G. simondi* (=Assemblage G)	Rats
C. canis	Dogs		
C. molnari	Fish		
C. hominis	Humans		
C. suis	Pigs		
Genotypes			
Ferret	Deer mice		
Mouse	Squirrel (x2)		
Skunk	Bear		
Marsupial (x4)	Goose (x2)		
Horse	Duck		
Rabbit	Bovine		
Monkey	Snake		
Pig (x2)	Tortoise		
Cervine (x3)	Lizard		
Fox	Woodcock		
Muskrat (x2)			

[a] Proposed (see, Thompson and Monis, 2004; Xiao et al., 2004).

1989). In cattle, calves can begin shedding *C. parvum* as early as 2 days of age, but peak shedding occurs at ~14 days of age (reviewed in Thompson et al., 2005). In sheep, the prepatent period for cryptosporidial infections is ~4 days (Thompson et al., 2005).

Epidemiology

Giardia has been reported in dogs and cats worldwide. The reported prevalence of *Giardia* tends to vary considerably between studies and is often influenced by the sensitivity of the diagnostic test used and whether only a one-off faecal sample was examined, given the intermittent nature of cyst excretion. Surveys of a variety of canine populations for the presence of *Giardia* reported a prevalence of approximately 10% in well cared for dogs, 36–50% in pups and up to 100% in breeding establishments and kennels (Hahn et al., 1988; Kirkpatrick, 1988).

Giardia has been reported in cattle (St. Jean et al., 1987; McDonough et al., 1994; Xiao and Herd, 1994; O'Handley et al., 1999), sheep (Buret et al., 1990; Taylor et al., 1993), goats (Bomfim et al., 2005; Castro-Hermida et al., 2005), elk and deer (Deng and Cliver, 1999), and most likely infects all ruminants (O'Handley and Olson, 2006). The prevalence of *Giardia* reported in both dairy and beef calves has been as high as 100 (Xiao and Herd, 1994; O'Handley et al., 1999; Ralston et al., 2003). Although all ruminants are likely to be exposed to *Giardia* shortly after birth, infections are most common toward the end of the neonatal period (O'Handley and Olson, 2006).

C. canis and *Cryptosporidium felis* do not appear to be common parasites of dogs and cats, respectively, although few targeted surveys have been published (see Thompson et al., 2005). The fact that oocyst shedding of both parasites is more common in younger animals and that stress can induce shedding suggested that chronic, sub-clinical infections may be more common than surveys indicate (Thompson et al., 2005).

C. parvum is considered to be the most common enteropathogen in calves during the first week of life, although it may be associated with other viral, bacterial and parasitic pathogens (Fayer et al., 1998; de Graff et al., 1999a; O'Handley et al., 1999). *Cryptosporidium andersoni* and *Cryptosporidium bovis* occur in adult cattle, but their distribution and prevalence are not well determined (O'Handley and Olson, 2006). Similarly, the prevalence of *C. parvum* and the cervine genotype in sheep and goats is not well understood.

Public health significance

Molecular tools have not only helped to resolve the taxonomy of *Giardia* and *Cryptosporidium*, but have also made a major contribution to our understanding of the host range of the different species and genotypes (Table 1). In particular, the ability to characterise cysts/oocysts directly from faeces or environmental samples using polymerase chain reaction (PCR)-based procedures has been useful in determining risk factors (Hunter and Thompson, 2005), and offers great potential in determining the sources of infection in outbreak situations and the transmission dynamics of the parasites in endemic foci.

Numerous isolates of *Giardia* and *Cryptosporidium* collected from different species of hosts in different geographical locations have been genotyped and the occurrence of the same species/genotype in humans and other animals has been demonstrated (Monis and Thompson, 2003). Such data are indicative of zoonotic potential and most authorities would agree that *G. duodenalis* and *C. parvum* are zoonotic. It has been known for some time that dogs can harbour infections of either zoonotic or host-specific assemblages of *Giardia* (Caccio et al., 2005), and this has been demonstrated in a number of recent studies in urban areas of Mexico, Brazil, Japan, Italy and Poland (Berrilli et al., 2004; Itagaki et al., 2005; Lalle et al., 2005; Eligio-Garcia et al., 2005; Zygner et al., 2006; Volotão et al., 2007). The most recent report from Germany found that of 60 *Giardia* positive samples collected randomly from dogs in urban areas, 60% were infected with zoonotic *Giardia* from assemblage A, 12% with dog-specific assemblages C and D, and the remaining 28% harboured mixed infections (Leonhard et al., 2007). Few studies have been undertaken in cats, but Vasilopulos et al. (2007) examined 250 cats from Mississippi and Alabama, USA, and of 17 positive for *Giardia*, found six infected with Assemblage A-I and 11 with Assemblage F (the cat genotype). As such, the finding of *Giardia* in the faeces of companion animals is justification for treatment.

Although studies on the occurrence of the different genotypes of *Giardia* serve to emphasise the potential public health risk from domestic dogs and cats, data on the frequency of zoonotic *Giardia* transmission is lacking. Such information can be obtained from molecular epidemiological studies that genotype isolates of the parasites from susceptible hosts in localised foci of transmission or as a result of longitudinal surveillance and genotyping of positive cases. In the former, recent research in localised endemic foci of transmission have provided evidence in support of the role of dogs in cycles of zoonotic *Giardia* transmission involving humans and domestic dogs from communities in tea growing areas of Assam, India, and in temple communities in Bangkok, Thailand (Traub et al., 2004; Inpankaew et al., 2007). In both studies, some dogs and their owners sharing the same living area were shown to harbour isolates of *G. duodenalia* from the same assemblage.

Although companion animals have long been considered as potential sources of human *Cryptosporidium* infection, the only studies in which oocysts recovered from dogs and cats have been genotyped have shown that they are most commonly infected with what appear to be predominantly host-adapted species; *C. canis* and *C. felis* (Morgan et al., 1998; Abe et al., 2002; Fayer et al., 2006). Thus dogs and cats, and possibly other companion animals, may not be important zoonotic reservoirs of *Cryptosporidium*

infection. However, with *Cryptosporidium*, there is considerable epidemiological data demonstrating strong links between contact with infected livestock and human infections (Fayer et al., 2000; Stantic-Pavlinic et al., 2003). This is not the case with *Giardia*, but with both organisms, infected livestock have long been incriminated as sources for the waterborne transmission of cryptosporidiosis and giardiasis (Fayer et al., 2000; Thompson, 2004). Interestingly, the application of genotyping procedures to the contaminating isolate(s) has often incriminated human effluent as the source (Thompson, 2004; Hunter and Thompson, 2005). However, in a study undertaken of cryptosporidiosis patients in Scotland, *C. parvum* was shown to be the causative agent in 84% of 67 cases, supporting livestock faecal pollution of water sources as the leading cause of human sporadic cryptosporidiosis (Goh et al., 2004).

Clinical signs

Although infections with *Giardia*, and also possibly *Cryptosporidium*, are common, most dogs and cats remain asymptomatic. If clinical disease manifests, it is usually associated with young animals and those in kennel or cattery situations (Robertson et al., 2000), where the effects of overcrowding, weaning and nutritional deficiency, may cause stress and exacerbate the effects of an infection (Thompson, 2004). The most consistent clinical sign of giardiasis in dogs and cats is small bowel diarrhoea, which may be acute or chronic, and self-limiting, intermittent or continuous in nature. Cryptosporidiosis in dogs and cats tends to manifest as an acute bout of small bowel diarrhoea.

Giardia infection in ruminants is often asymptomatic, but may also be associated with the occurrence of diarrhoea and ill-thrift in calves (O'Handley et al., 1999; Geurden et al., 2006). The importance of giardiasis as a cause of diarrhoea in ruminants is unclear, especially given that diarrhoea in ruminants is often multifactorial with more than one pathogen detected (O'Handley and Olson, 2006). Nevertheless, the significance of *Giardia* infection in ruminants warrants further investigation, particularly with regard to production loss. Production parameters were carefully examined in bottle-fed specific pathogen-free (SPF) lambs experimentally infected with *Giardia*; infection was shown to be associated with extended times for lambs to reach slaughter weight and decreased carcass weight (Olson et al., 1995).

Clinical cryptosporidial infection in calves is manifested by diarrhoea (varies from pale yellow with mucus to profuse watery diarrhoea), depression, anorexia and abdominal pain. Clinical signs can persist for 4–14 days and the severity and duration is highly variable, but is more common in housed animals where stress associated with overcrowding may predispose to disease. The pathogenesis of disease is frequently complicated by concurrent viral (rotavirus, coronavirus), bacterial (*Escherichia coli*, *Salmonella*) and parasitic (*Giardia*) infections (Fayer et al., 1998; O'Handley et al., 1999; de Graff et al., 1999a). Calves can die from dehydration and cardiovascular collapse, but cryptosporidiosis mortalities are highly variable (de Graff et al., 1999a). In endemic herds, morbidity rates are usually 100%, but mortalities are infrequently observed.

Pathogenesis

Giardia and *Cryptosporidium* infections can cause malabsorptive diarrhoea, but the factors associated with this are still unclear and much of what we know about the pathogenesis is confined to experimental infections. Pathogenesis results from interaction between parasite products, such as proteinases that break the epithelial barrier, and host inflammatory and immunological responses (Chai et al., 1999; Scott et al., 2000, 2004; Guk et al., 2003). Both *Giardia* and *Cryptosporidium* induce enterocyte apoptosis, associated with disruption of cytoskeletal and tight junctional proteins in a strain-dependent manner (Chin et al., 2002).

Villus atrophy, diffuse shortening of microvilli, reduced disaccharidase activity, loss of epithelial barrier function, increased permeability and apoptosis have all been reported in *Giardia* infections (Buret, 2007). Recent evidence also showed that *Giardia* infection can cause hypersecretion of chloride ions (Troeger et al., 2007). These changes are thought to be due to a combination of parasite products, possibly a toxin, and host immune factors, particularly involving CD8+ cells (Buret, 2007).

Cryptosporidium infection is associated with villus atrophy, villus fusion and inflammation (Koudela and Jirí, 1997), which results in loss of absorptive surface area and impaired nutrient transport. It is not clear how the parasite interferes with cell function, but it appears to be able to prevent and induce apoptosis (Buret et al., 2003; Mele et al., 2004). It has been suggested that a cholera-like enterotoxin may be involved in the development of secretory diarrhoea, but this has yet to be identified (Guarino et al., 1995).

It should be emphasised that clinical signs do not always occur in calves or companion animals naturally infected with *Giardia* or *Cryptosporidium*, and it is still not clear how the changes referred to above relate to the expression of clinical disease.

Diagnosis

Light microscopy remains the most practical approach for the diagnosis of *Giardia* in a clinical setting, using zinc sulphate centrifugation for concentration of cysts in faecal specimens (Zajac et al., 2002). Because cyst excretion is sporadic, several faecal samples should be examined over 4–5 days. There are several enzyme-linked immunosorbent assay (ELISA)-based methods available that detect coproantigens, and these work well but are relatively expensive. Due to the high cost, indirect immunofluorescence and PCR are normally restricted to epidemiological studies and as research tools.

Current diagnostic laboratory methods generally rely on microscopic examination of faecal samples for detecting *Cryptosporidium* oocysts. A number of staining techniques have been developed, but many suffer from problems of sensitivity and specificity, often with variable results between laboratories (Elliot et al., 1999). The most recent staining method to have been described for detecting *Cryptosporidium* in stools employs a negative staining technique with malachite green, which has demonstrated superior results to other methods (Elliot et al., 1999). Concentration of faecal samples using saturated sugar is recommended particularly for livestock samples. The oocysts of *C. parvum*, *Cryptosporidium hominis* and many other species and genotypes of *Cryptosporidium* are morphologically indistinguishable in terms of size and it is only the larger oocysts of *C. andersoni* and *C. muris* that can be reliably distinguished from these. Copro-ELISAs are available, but are expensive and, as with *Giardia*, immunofluorescence and PCR are not practical clinically.

With both *Giardia* and *Cryptosporidium*, the big advantage of microscopy is that it is not specific and therefore other parasites can be detected, which may be important in determining the cause of non-specific symptoms, such as diarrhoea. It should also be remembered that *Giardia* and *Cryptosporidium* can be found in domestic animals in the absence of clinical signs.

Treatment

There are many different causes of diarrhoea in dogs and cats, and *Giardia* is not necessarily a common cause. If *Giardia* is found on faecal examination, it should be treated, regardless of whether the animal is ill or asymptomatic. Treatment is necessary given the zoonotic potential of this parasite and, importantly, because the significance of infection in animals is not completely understood.

There are a number of drugs which have been used to treat giardiasis in dogs, such as metronidazole, furazolidone (Kirkpatrick, 1987), quinacrine (Zimmer and Burrington, 1986), albendazole (Barr et al., 1993b), oxfendazole (Villeneuve et al., 2000) and fenbendazole (Barr and Bowmann, 1994; Meyer, 1998; Zajac et al., 1998). Drugs reported to be effective for treating cats include metronidazole, quinacrine (Brightman and Slonka, 1976) and furazolidone (Kirkpatrick, 1986). Although quinacrine has been shown to improve clinical signs, it does not eliminate infection in cats (Brightman and Slonka, 1976) and can cause anorexia, lethargy and fever (Zimmer and Burrington, 1986). Barr et al. (1993b) demonstrated that albendazole was effective for treating giardiasis in dogs, however, in another study albendazole caused bone marrow suppression (Stokol et al., 1997) and was ineffective for the treatment of giardiasis in a small group of cats (Barr et al., 1993a). Oxfendazole given orally was tested experimentally at a single and double dose (11.3 and 22.6 mg/kg, respectively) and proved very effective in removing *Giardia* from dogs with no signs of toxicity (Villeneuve et al., 2000).

Oral metronidazole has been used frequently to treat giardiasis in dogs and cats. However, a number of detrimental side effects have been associated with this drug, such as the acute development of anorexia and vomiting with progression to signs of central nervous system toxicity (Dow et al., 1989; Fitch et al., 1992; Caylor and Cassimatis, 2001). Metronidazole has also been shown to be only 67% effective in eliminating *Giardia* from infected dogs (Zimmer and Burrington, 1986) and its common usage may cause parasitic resistance to the drug (Upcroft and Upcroft, 1993).

Fenbendazole has been used to treat giardiasis in dogs and is effective at stopping the shedding of *Giardia* cysts at a dosage of 50 mg/kg given orally once a day for 3 days (Barr and Bowmann, 1994; Meyer, 1998; Zajac et al., 1998). Fenbendazole is also effective against hookworms, whipworm and roundworms, and is safe to administer to pregnant dogs and puppies as young as 6 weeks of age (Barr and Bowmann, 1994). Febantel is a probenzimidazole, which is metabolised into fenbendazole and oxfendazole, and has a wide safety margin in dogs. A combination product containing 50 mg of praziquantel, 144 mg of pyrantel embonate and 150 mg of febantel (Drontal Plus, Bayer) has been registered in some countries for use in dogs to treat *Giardia* with one dose of the combination tablet given daily over three consecutive days to treat *Giardia* infection (Bayer HealthCare). A combination of praziquantel, pyrantel and febantel is currently not used to treat cats (Scorza et al., 2006) and further research is needed into the potential application of febantel in cats.

There is currently no licensed drug available to treat giardiasis in ruminants, although the need to treat infections in ruminants is questionable (O'Handley and Olson, 2006). A number of drugs have been shown to be efficacious against giardiasis in calves (for a summary of these drugs and their dosages see O'Handley and Olson (2006)). Treatment alone is not sufficient for controlling *Giardia* infection in ruminants because re-infection occurs rapidly and, given the high level of environmental contamination, daily administration of drugs would be needed (O'Handley et al., 2000; Geurden et al., 2006). Good husbandry, including the prompt removal of faeces from an animal's environment is likely to minimise the chances of re-infection and transmission of *Giardia* in all species.

In spite of extensive screening of a large number of chemotherapeutic agents, there is no reliable curative treatment for cryptosporidiosis (Armson et al., 2003). Most of the chemotherapeutic agents that have been shown to be effective in controlling coccidiosis in cattle, pigs and poultry have limited or no efficacy against cryptosporidiosis, emphasising the non-coccidian features of *Cryptosporidium* (Barta and Thompson, 2006). Halofuginone lactate (Halocur, Intervet) has been used as an anticoccidial agent in poultry and has recently been registered in Europe as a chemotherapeutic agent for cryptosporidiosis in domestic cattle. Halofuginone has a cryptosporidiostatic effect on sporozoite and merozoite stages of the parasite. It has been

shown to reduce incidence and severity of diarrhoea, but does not prevent oocyst shedding (Villacorta et al., 1991; Joachim, 2003). In companion animals, promising results have been reported for treating infections with *Cryptosporidium* in cats using paromomycin, tylosin or azithromycin (Lappin, 2005).

Treatment of cryptosporidiosis is currently focused around rehydration and electrolyte replenishment during the early stages of infection before host immunity is expressed.

Vaccination

A *G. duodenalis* vaccine, produced from trophozoites isolated from sheep, is available for dogs and cats in North America (Olson et al., 2000). Puppies and kittens inoculated with the vaccine subcutaneously and subsequently challenged with infection did not develop clinical signs of giardiasis. They demonstrated a reduction or elimination of intestinal trophozoites and faecal cyst excretion, while vaccinated animals had higher weight gains compared to non-vaccinated animals (Olson et al., 1996, 1997). Furthermore, the vaccine has been used as a therapeutic agent in dogs chronically ill with giardiasis which had not responded to chemotherapeutic drugs, and vaccination resulted in the cessation of clinical signs and faecal cyst shedding (Olson et al., 2001). However, a number of other studies have failed to demonstrate a significant effect of the vaccine on infected animals (Payne et al., 2002; Stein et al., 2003; Anderson et al., 2004). Currently, there is no *Giardia* vaccine available for use in ruminants.

Vaccination has been proposed as a method to control cryptosporidiosis in animal populations. Whole oocyst preparations, subunit vaccines and DNA vaccines have been prepared and vaccination trials have been conducted in mice and calves (Harp and Goff, 1998; de Graaf et al., 1999b; Perryman et al., 1999; Sagodira et al., 1999; Jenkins, 2001). Vaccines have been shown to reduce clinical signs, but in most cases have not eliminated or reduced oocyst shedding. As calves, lambs and goats are infected with *Cryptosporidium* spp. during the first or second week of life, passive immune protection by vaccination of dams is the approach for these species (Harp and Goff, 1998). Colostrum containing a high concentration of immunoglobulin G antibodies to *Cryptosporidium* (hyperimmune colostrum) has been reported to reduce diarrhoea and oocyst shedding in calves and lambs (Harp et al., 1989; Fayer et al., 1998; Naciri et al., 1994). Vaccination of dams will enable dams to produce protective hyperimmune colostrum.

Conclusions

Molecular epidemiology has had an enormous impact on the taxonomy of both *Giardia* and *Cryptosporidium* at the species and intraspecific levels. As such, we are in a much better position to evaluate risk factors for public health from infections in companion animals and livestock. The public health significance of infections in domestic animals does not appear to be as great as previously thought, yet there is a need to undertake molecular epidemiological studies in localised, well defined endemic foci, particularly in developing countries and among disadvantaged groups. Our understanding of the pathogenesis of infections with *Giardia* and *Cryptosporidium* has improved and we are closer to being able to answer why clinical disease occurs in some individuals, but may not be apparent in others. Drugs are available to treat infections with *Giardia* but the question is when to use them. There are no effective drugs to treat cryptosporidial infections, highlighting the atypical features of this parasite. The prospects for vaccines against both organisms seem a long way off, but there would be clear value for their use in livestock.

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