Cryptosporidium and Giardia in Ruminants

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KEYWORDS
- Cryptosporidium • Giardia • Ruminants • Cattle • Sheep • Goat

KEY POINTS
- Giardiasis and cryptosporidiosis have been documented in ruminants worldwide regardless of the husbandry system.
- Infections with Cryptosporidium and Giardia are an animal health concern because of the direct economic losses associated with the infection.
- There is also a public health concern because Cryptosporidium and Giardia are highly prevalent in ruminants and humans, indicating potential for human exposure from environmental contamination.
- The application of molecular techniques has resulted in expanded knowledge regarding the taxonomy and epidemiology of Giardia and Cryptosporidium.

INTRODUCTION

Cryptosporidium and Giardia are two common enteric pathogens of animals and humans with global distribution. They are transmitted by the fecal-oral route via direct contact with an infected host or through consumption of contaminated water or food with infective stages (oocysts or cysts). Cryptosporidium and Giardia infections can be asymptomatic or cause mild to severe gastrointestinal illness in both animals and humans. Both parasites are an animal health concern because of the direct economic losses associated with infection, but there is also a public health concern because of the potential for human exposure to environmental contamination of Cryptosporidium oocyst and Giardia cysts from animals.1,2 The application of molecular approaches for the diagnosis of these two parasites has led to significantly improved knowledge regarding the epidemiology of these protozoans.

CRYPTOSPORIDIOSIS

Cryptosporidiosis is caused by the protozoan Cryptosporidium. The genus Cryptosporidium includes ubiquitous protozoan parasites that infect many vertebrate hosts,
including humans. In ruminants, cryptosporidiosis has been reported worldwide regardless of husbandry system. The most common symptom associated with Cryptosporidium is diarrhea, but lack of appetite, fever, or malabsorption could also be observed. Infections with Cryptosporidium are a significant problem for animal health, mostly in neonatal livestock, causing economic losses associated with decreased growth rate and mortality in the infected animals. In addition, cryptosporidiosis increases the cost of animal health care and veterinary services. In humans, cryptosporidiosis infections usually produce self-limited profuse, watery diarrhea that can last up to 3 weeks in persons with healthy immune systems but can lead to life-threatening malnutrition and wasting in immunocompromised individuals.

**Life Cycle and Transmission**

Cryptosporidium has a complex life cycle that involves both sexual and asexual replication in a single host (Fig. 1). After ingestion of the infective stage (sporulated oocyst) by a suitable host, excystation occurs in the gastrointestinal tract and 4 sporozoites are released. Then, the sporozoites invade the gastric or intestinal epithelium depending on the Cryptosporidium species. Although the parasites are intracellular, they are extracytoplasmatic, so there is no contact with the host-cell cytoplasm. Sporozoites undergo asexual reproduction (merogony) and then sexual reproduction (gametogony) upon differentiating into either macrogamonts (female) or microgamonts (male). After fusion of a microgamont with a macrogamont, a zygote is formed, which then develops into an oocyst. Oocysts sporulate in situ and, when mature, contain 4 naked sporozoites. Because oocysts sporulate in situ, autoinfection is possible when sporozoites are released from the oocyst within the same host.

Cryptosporidium is transmitted via the fecal-oral route by both direct and indirect transmission. Direct transmission occurs through ingestion of oocysts present in feces of the infected hosts, whereas indirect transmission occurs by ingestion of water or food contaminated with oocysts. There are several factors that contribute to the transmissibility of Cryptosporidium. These factors include (1) simple fecal-oral transmission route with oocysts excreted fully sporulated and immediately infective to other suitable hosts; (2) oocysts can persist in harsh conditions for long periods of time; (3) oocysts are resistant to many conventional disinfectants, including chlorine; (4) ability of infected hosts to shed very large quantities of oocysts (eg, a neonatal calf can shed up to 30 billion oocysts over a 1–2 weeks); and (5) the low infectious dose (10–30 oocysts).

**Epidemiology, Zoonotic Potential, and Public Health**

Cryptosporidium infections have been documented in ruminants worldwide. Point prevalence studies indicate a wide range of infection rates, whereas longitudinal studies indicate that morbidity rates often reach 100%. Variations in the reported prevalence among studies are related to many different factors, including study design, sample size, geographic region, climate, season, age, breed, management practices, or detection methods used. A recent systematic review and meta-analysis of Cryptosporidium in livestock found that prevalence in farmed animals was higher in the Americas and Europe than in other continents, which was attributed to the intensive farm animal production in those regions.

Infections are most commonly diagnosed in neonatal animals. Young animals can become infected and begin shedding Cryptosporidium oocysts shortly after birth, with the highest rates of infection reported in animals between 1 and 3 weeks of age. A potential source of infection for neonatal lambs and kids could be the presence of adults with subclinical infections, especially during the periparturient period,
when increased oocyst shedding has been shown. In beef cattle, no periparturient increase in *Cryptosporidium parvum* oocysts excretion was observed, but oocysts were frequently detected in asymptomatic cows, suggesting that they may play a role in calf infections. In contrast, no clear connection has been found between the presence of oocyst-excreting adults and the infections in neonate calves, suggesting there

Fig. 1. Life cycle of *Cryptosporidium* species. Sporulated oocysts, containing 4 sporozoites, are excreted by the infected host through feces (1). Transmission of *Cryptosporidium* species occurs by the fecal-oral route (2). Following ingestion by a suitable host (3), excystation (a) occurs. The sporozoites are released and parasitize the epithelial cells of the gastrointestinal tract (b, c). In these cells, the parasites undergo asexual multiplication (merogony) (d, e, f) and then sexual multiplication (gametogony) producing microgamonts (male) (g) and macrogamonts (female) (h). Fertilization of the macrogamonts by the microgametes (i). After fusion of a microgamont with a macrogamont, a zygote is formed, which then develops into an oocyst and sporulates in the infected host. There are 2 different types of oocysts (thick walled and thin walled). Thick-walled oocysts are excreted with feces into the environment (j), whereas thin-walled oocysts are involved in an autoinfective cycle (k). (Image courtesy of DPDx, Centers for Disease Control and Prevention [https://www.cdc.gov/dpdx/].)
are additional factors contributing to infections in newborn calves, such as the presence of wildlife (small rodents or birds) or environmental contamination (dirty water and manure sources). In addition, in longitudinal studies performed on a Scottish dairy farm, adult cows were not considered the source of infection for newborn calves, because different subtypes of \textit{C parvum} were identified in the calves and adults.8

In the last 2 decades, substantial progress has been made in identifying \textit{Cryptosporidium} species present in ruminants. Before the application of molecular tools, most studies reported the presence of oocysts of \textit{Cryptosporidium} species in ruminants. Until the early 2000s, most studies reported only \textit{C parvum} and \textit{“Cryptosporidium muris”} in ruminants, because of the morphologic similarity of \textit{Cryptosporidium} oocysts from different species. Later, molecular studies and cross-species transmission studies identified \textit{“C muris”} from cattle as a new species, namely \textit{Cryptosporidium andersoni}.21 Subsequently, with the assistance of molecular techniques, additional species of \textit{Cryptosporidium} have been described in ruminants.22–25

There are 4 main \textit{Cryptosporidium} species responsible for infections in cattle: \textit{C parvum}, \textit{Cryptosporidium bovis}, \textit{Cryptosporidium ryanae}, and \textit{C andersoni}.9,21–23 Other species have also been sporadically reported in cattle, including \textit{Cryptosporidium hominis}, \textit{Cryptosporidium suis}, \textit{Cryptosporidium canis}, and \textit{Cryptosporidium felis}.3 However, it is likely these species are not maintained within cattle populations but result from occasional contact with their respective primary hosts. \textit{C andersoni}, one of the largest species of \textit{Cryptosporidium}, infects the abomasum, whereas \textit{C parvum}, \textit{C ryanae}, and \textit{C bovis}, which are of similar size and smaller than \textit{C andersoni}, infect the small intestine. Most studies worldwide suggest an age-related distribution of \textit{Cryptosporidium} species in cattle, with \textit{C parvum} being the predominant species in preweaned calves, \textit{C bovis} and \textit{C ryanae} as the predominant species in postweaned calves, and \textit{C andersoni} as the predominant species in juvenile and adult cattle.9,11,26,27 However, this is not the case in China, where \textit{C parvum} is less common and \textit{C bovis} is the dominant species in preweaned dairy calves.14,28 This finding could indicate differences in the transmission of \textit{Cryptosporidium} species in preweaned dairy calves in China.

Molecular epidemiologic studies have identified different species and genotypes of \textit{Cryptosporidium} in sheep. The most common species identified are \textit{Cryptosporidium ubiquitum}, \textit{Cryptosporidium xiaoii}, and \textit{C parvum}, although others, such as \textit{C andersoni}, \textit{C bovis}, \textit{C ryanae}, \textit{C hominis}, \textit{Cryptosporidium fayeri}, and \textit{C suis}, have been identified sporadically in sheep.18,24,25,29–34 Although most studies in sheep showed a predominance of \textit{C ubiquitum} and \textit{C xiaoii} worldwide,10,18 studies from European countries have reported that \textit{C parvum} predominates.13,33,35–37 The distribution of \textit{Cryptosporidium} species in sheep is not as clearly associated with age as it is in cattle. Some studies reported \textit{C ubiquitum} as the most common species in older animals, whereas \textit{C parvum} or \textit{C xiaoii} were the predominant species in lambs younger than 1 month of age.18,35,36 However, other studies have found \textit{C ubiquitum} in similar numbers in lambs and adults.10,38

In goats, the most common \textit{Cryptosporidium} species identified are \textit{C ubiquitum}, \textit{C xiaoii}, and \textit{C parvum}, whereas other species and genotypes, such \textit{C hominis}, \textit{Cryptosporidium baileyi}, \textit{C andersoni}, and rat genotype II, have only been identified sporadically.12,13,39,40

Animals can be reservoirs of zoonotic \textit{Cryptosporidium} species.1 Ruminants have been implicated as a source of zoonotic \textit{Cryptosporidium} outbreaks originating from direct contact with infected animals.41,42 Zoonotic species of \textit{Cryptosporidium} in ruminants include \textit{C parvum} and \textit{C ubiquitum}. \textit{C parvum} is the second most common species that infects humans1 and \textit{C ubiquitum} is considered an emerging human
Both species have a broad host range and are responsible for zoonotic infections with a wide geographic distribution. Although other Cryptosporidium species commonly reported in ruminants have been sporadically found in humans, they are not considered major zoonotic species. *C. andersoni* has been reported in humans in China, France, Malawi, Iran, the United Kingdom, and Australia.\textsuperscript{44–49} *C. bovis* has also been reported in farm workers in India and Australia,\textsuperscript{50,51} and *C. xiaoi* was reported in 2 patients with human immunodeficiency virus/acquired immunodeficiency syndrome in Ethiopia.\textsuperscript{52}

Before the use of molecular subtyping tools to characterize the transmission dynamics of Cryptosporidium infections in humans and animals, it was presumed that infections caused by *C. parvum* were from zoonotic transmission. At present, sequencing a portion of the hypervariable 60-kDa glycoprotein gene (*gp60*) allows further characterization into subtypes that are grouped within families. The use of *gp60* subtyping has identified human-specific, animal-specific, and zoonotic subtypes for *C. parvum* and *C. ubiquitum*.\textsuperscript{1,43,53} Host adaptation for *C. ubiquitum* subtypes is apparent with subtypes in XIIa found in ruminants, XIIb to XIIf in rodents, and XIIa (ruminant adapted) and XIIb to XIIId (rodent adapted) in humans.\textsuperscript{33,43} Similarly, *C. parvum* subtypes within families IIb, IIc, and IIf are found only in humans, whereas subtypes within families Ila and IId are found in both humans and animals.\textsuperscript{1} Subtyping of *C. parvum* from preweaned calves has identified almost exclusively Ila and IId subtypes with differences in subtypes across geographic locations or host age.\textsuperscript{1,3,28,53–56} There are still few data available on *C. parvum* subtypes in small ruminants, and there are inconsistencies on the distribution of Ila and IId subtypes among different studies with respect to most common subtypes in goat kids or lambs.\textsuperscript{9,35,57} These discrepancies could be associated with different management strategies and the ability of small ruminants to get in contact with other livestock such as cattle, the main reservoir of Ila subtypes.

**Pathogenesis, Clinical Features, and Impact in Production**

*Cryptosporidium* infections are frequently reported as a cause of diarrhea in neonatal ruminants. Neonatal diarrhea in ruminants can involve multiple pathogens (parasites, bacteria, and viruses) in addition to management and nutritional factors, but *Cryptosporidium* infection is unequivocally recognized by veterinarians as a major cause of diarrhea in neonatal ruminants.\textsuperscript{4} A wide range of clinical signs can be observed in infected animals and can range from asymptomatic to death.\textsuperscript{3,4} Differences in the severity of cryptosporidiosis are likely multifactorial and associated, among other factors, with host immune status, coinfections with other pathogens, and with different species/genotypes of *Cryptosporidium*. In cattle, most reports of disease are associated with *C. parvum* and are characterized by an acute onset of profuse watery diarrhea, often associated with loss of appetite, depression, and weakness.\textsuperscript{53,58,59} Mortality from dehydration has been reported in neonatal calves, but it is rare in endemic herds with high morbidity rates.\textsuperscript{11,60} *C. parvum* is mostly found in the epithelium of the lower small intestine causing enteritis, decreased villous length, villous atrophy, and fusion of villi.\textsuperscript{61} A recent study that evaluated management practice and environmental factors associated with average daily gain in preweaned dairy calves in US dairy operations showed a significantly lower average daily gain in calves infected with *Cryptosporidium*.\textsuperscript{62} Infections in cattle with the host-adapted cattle species *C. bovis* and *C. ryanae* have not been associated with illness.\textsuperscript{22,23} Infections with *C. andersoni* follow a more chronic course and do not cause diarrhea.\textsuperscript{63} Although cattle infected with *C. andersoni* have no clinical signs, it has been suggested that infections may interfere with milk production in dairy cows\textsuperscript{64} and may cause significant reduction
in rate of weight gain in beef cattle. In addition, increased plasma pepsinogen levels, pale and thickened gastric mucosa, and dilated gastric glands have been associated with *C. andersoni* infections.

In small ruminants, the major clinical signs of cryptosporidiosis are diarrhea and weight loss. Different clinical manifestations have been associated with age and the species of *Cryptosporidium*. Although molecular information on cryptosporidiosis in small ruminants is still limited, clinically ill lambs and kids seem to be more often infected with *C. parvum*, whereas *C. xiaoii* and *C. ubiquitum* are mostly reported in healthy lambs. No clinical signs were observed in lambs experimentally infected with *C. xiaoii* and *C. ubiquitum*. However, there are reports of *C. parvum* in healthy animals, which could perhaps serve as asymptomatic carriers, and the presence of *C. xiaoii* and *C. ubiquitum* in diarrheic animals. In addition, *C. andersoni* has been identified in sheep without clinical signs. In general, there is limited information on the impact of cryptosporidiosis in small ruminant production, and future studies should investigate the potential impacts of infection, including studies of asymptomatic animals. In Australia, *Cryptosporidium* infection in lambs had a negative impact on carcass profit indicators (carcass weight and dressing percentage) as well as reduced live weight, and an increase in fecal consistency scores. In goats, *Cryptosporidium* infection was associated with lower growth rate with and without diarrhea beyond preweaning.

**Diagnosis**

Cryptosporidiosis should be included in the differential diagnosis in all cases of neonatal diarrhea in ruminants (intestinal cryptosporidiosis) and in cases in which dairy and feedlot cattle are not performing well (abomasal cryptosporidiosis associated with *C. andersoni*). Detection and identification protocols for *Cryptosporidium* oocysts can be used directly on fecal specimens, but generally feces should be subjected to concentration methods to increase sensitivity. Flotation with saturated sugar solution is the most common method to concentrate oocysts in ruminants. For decades microscopic examination was the only method for detecting *Cryptosporidium* using the modified acid-fast protocol and later immunofluorescent antibody techniques to visualize oocysts. Under the microscope, oocysts are colorless, spherical or slightly ovoid, smooth, thick walled, and contain 4 elongated sporozoites. The size of *Cryptosporidium* oocysts reported in ruminants range from 3.7 × 3.2 μm (*C. ryaeanae*) to 7.4 × 5.5 μm (*C. andersoni*). However, differentiating *Cryptosporidium* species/genotypes using microscopy is not possible because oocysts are similar in shape and overlap in size. Oocyst size is helpful to differentiate gastric and intestinal *Cryptosporidium* species because oocysts from species infecting the stomach (eg, *C. andersoni*) are larger than those from intestinal species (eg, *C. parvum*, *C. xiaoii*, or *C. ryaeeae*).

Antigen detection kits based on enzyme-labeled antibodies to detect *Cryptosporidium* oocysts are commercially available. Because there is wide variability in sensitivity of the different coproantigen detection immunoassays, they do not seem to increase sensitivity compared with microscopy, but they are less time consuming and easier to perform. However, a disadvantage is that not all commercial antibody kits recognize all *Cryptosporidium* species or genotypes.

Molecular methods that use polymerase chain reaction (PCR) and DNA sequencing, PCR restriction fragment polymorphism, real-time PCR assays, or multiplex PCR are more sensitive than microscopy and immunologic assays for the detection of *Cryptosporidium* in feces. The small subunit ribosomal RNA (rRNA) gene is currently considered the most reliable locus for detection and identification of *Cryptosporidium*.
species and genotypes. In addition, subtyping using the gp60 gene is essential to better understand the dynamics of Cryptosporidium transmission. A major advantage of molecular methods is that they allow genetic characterization to identify species, genotypes, and subtypes, a requirement for epidemiologic studies designed to understand Cryptosporidium transmission routes. However, molecular methods are mostly restricted to research and specialized laboratories.

Treatment and Prevention

Although vaccine trials to prevent cryptosporidiosis have been conducted in calves, currently there is no vaccine available. However, it is unclear whether vaccination is justified because cryptosporidiosis is usually self-limiting and infected animals with normal immune systems improve without treatment. In severe cases, clinical symptoms such as watery diarrhea or dehydration can be managed by supportive treatment with electrolytes. Several drugs have been tested to treat Cryptosporidium in ruminants, but with limited success. Only the halofuginone lactate (which is not available in the United States) has been licensed for treatment of Cryptosporidium in ruminants, but its efficacy is controversial. In newborn calves, treatment with halofuginone lactate is recommended orally (after colostrum or milk/milk replacer feeding) for the first 7 days of life at a dose of 100 µg of halofuginone base per kilogram of body weight (https://www.ema.europa.eu/en/medicines/veterinary/EPAR/halocur). A recent study indicated that administration of the correct prophylactic treatment with halofuginone (based on the calf’s age and duration of treatment) is critical and that incorrect dosing has minimal impact on mortality and is equivalent to not treating, regarding the proportion of calves shedding oocysts. A delay in Cryptosporidium oocyst shedding was also shown coupled with improved neonatal survival, but there was a negative effect on weight gain also reported. Similarly, other studies showed efficacy in reducing Cryptosporidium oocyst shedding compared with placebo groups, but with no clear differences in the occurrence of diarrhea. A study evaluating the effect of halofuginone in goat kids experimentally infected with C parvum reported a reduction in oocyst shedding, diarrhea, and mortality in kids that received the treatment.

Cryptosporidium oocysts are resistant to most common commercial disinfectants. To successfully control Cryptosporidium, proper management strategies to prevent transmission among animals are necessary. The use of good husbandry practices are needed, including decreased stocking density on farms, separating young and adult animals, ensuring that neonatal animals have received adequate colostrum, and keeping animals with diarrhea in isolation. In addition, adequate sanitation practices should include cleaning stalls before the introduction of new animals, especially newborn animals, to destroy oocysts using either heat or chemical disinfection (hydrogen peroxide), sterilization processes using steam, or ultraviolet light. From a public health perspective, manure must be properly managed to minimize fecal contamination of food and water with oocysts of zoonotic Cryptosporidium species, and good hygiene practices among those handling animals, especially young ruminants, should include frequent hand washing.

GIARDIASIS

The protozoan flagellate Giardia is the causal agent of giardiasis. Giardia species can infect multiple hosts, ranging from mammals to birds and amphibians. At present, 8 species of Giardia are considered valid: Giardia microti, Giardia cricetidarum, and Giardia muris infect rodents; Giardia ardeae and Giardia psittaci birds; Giardia agilis
amphibians; *Giardia peramelis* marsupials; and *G duodenalis* (syns. *Giardia intestinalis* and *Giardia lamblia*) infects most vertebrates, including humans. *G duodenalis* is one of the most common enteric parasites in humans and mammals worldwide, causing an estimated 280 million human cases of gastroenteritis annually, with higher infection rates in developing countries. *G duodenalis* is considered a species complex consisting of 8 assemblages (designated A to H) based on genetic analysis but with little variation in morphology. Assemblages A and B have low host specificity, infecting humans and a wide range of animals. In contrast, assemblages C to H are host adapted, with C and D reported mainly in canines, E in artiodactyls, F in felines, G in rodents, and H in seals. It has been proposed to recover previously used *Giardia* species names based on host occurrence; however, to accept this proposal, proper redescriptions that include morphologic, biological, and genetic data will be necessary.

**Life Cycle and Transmission**

The life cycle of *Giardia* is simple and comprises only 2 stages: the cyst and the trophozoite (Fig. 2). When infective stages (cysts) are ingested by the host, they excyst in the duodenum, releasing 2 trophozoites. Trophozoites are not invasive and undergo repeated mitotic division on the mucosal surface of the small intestine. Later, in response to bile salt and other conditions present in the gut, trophozoites develop to cysts (the environmentally resistant form) that are passed in the feces.

Transmission is fecal-oral and occurs via direct contact with infected humans (anthroponotic transmission) or animals (zoonotic) and indirectly by ingestion of water or food contaminated with cysts (water-borne and food-borne transmission). Factors that contribute to the successful spread of giardiasis include (1) large numbers of cysts released into the environment by infected hosts; (2) cysts that are immediately infectious after excretion and that remain viable for extended times under the right conditions (cool temperatures and moisture); and (3) low infectious dose.

**Epidemiology, Zoonotic Potential, and Public Health**

Reports of *G duodenalis* are frequent in ruminants worldwide. Infection rates vary greatly among studies and range from ~1% to as much as ~60% in cattle, sheep, and goats. However, in longitudinal studies the cumulative incidence increases to 100% in ruminants. Differences in prevalence among studies are most likely associated with different management practices, age of the animals examined, and detection methods used. A higher prevalence is consistently observed in younger animals compared with adults, with the highest occurrence reported around 2 months of age. Among the different methods used to detect infections, higher prevalences are reported by PCR and enzyme-linked immunosorbent assay than by microscopy.

Molecular epidemiologic surveys in ruminants worldwide have reported E as the predominant assemblage followed by lower frequencies of assemblage A, and sporadic reports of assemblage B. There are also rare reports of canine assemblages C and D in ruminants, but it is unclear whether they represent true infections. Assemblage E is not usually identified in humans and thus its zoonotic risk is minimal. In contrast, assemblages A and B are common in humans and can be transmitted zoonotically, indicating a significant public health impact, and there are reports of farmers infected with those assemblages.
Pathogenesis, Clinical Features, and Impact in Production

Giardiasis is a common disease in ruminants, characterized by diarrhea, weight loss, and malabsorption, but asymptomatic infections are also common.\textsuperscript{60,92,100,101} The importance of *Giardia* as a cause of diarrhea in ruminants is still unclear, because diarrhea in ruminants is associated with a combination of other host factors, including infections with other pathogens and husbandry practices. In addition, field studies have suggested that even asymptomatic infections can have a negative impact in production and growth performance in livestock.\textsuperscript{100,101} A recent study evaluating average daily gain in preweaned dairy calves in the United States reported that calves negative for *Giardia* gained more weight than calves positive for *Giardia*.\textsuperscript{62}
**Diagnosis**

Microscopic examination of fecal samples remains the cornerstone of diagnostic testing for *Giardia*. During *Giardia* infections, cysts are shed sporadically, and the detection of cysts may require the examination of several fecal samples.\(^2\) To increase sensitivity, concentration techniques are recommended.\(^1^0\) Both cysts and trophozoites can be observed in fecal samples, but trophozoites are only present when watery diarrhea occurs. Under the microscope, cysts have an ovoid shape (8–15 µm long) with 2 to 4 nuclei and fibrils and median bodies, and trophozoites (10–20 µm long) have a pyriform shape with ventral sucking disk, 2 nuclei, 2 median bodies, and 8 flagella (4 lateral, 2 ventral, and 2 posterior).\(^1^0^3\) Microscopy is rapid and inexpensive but requires a skilled parasitologist for proper identification and has decreased sensitivity when low numbers of cysts are present.\(^1^0^2\) The use of immunofluorescent antibodies to stain and visualize the cysts using immunofluorescent microscopy assists in detection. In addition, immunologic and molecular methods are also commonly used and are reported to be highly sensitive and specific.\(^1^0^2,1^0^3\) Antigen detection immunoassays have the advantage of being easy to standardize, requiring a short time to provide results, and showing higher sensitivity and specificity than microscopy.\(^1^0^2\) PCR assays are becoming more common and are considered more sensitive than microscopy and immunoassays. The loci most commonly used in PCR are small subunit rRNA, triose phosphate isomerase, glutamate dehydrogenase, and β-giardin.\(^2\) A clear advantage of molecular assays is the ability to do further sequence analysis from PCR-positive samples to allow assemblage identification. However, PCR and sequencing are used for research and are not routinely used for diagnosis.

**Treatment and Prevention**

There are no drugs or vaccines currently licensed for *Giardia* in ruminants. A vaccine is available for dogs (GiardiaVax), but a study that evaluated its efficacy in prevention of *Giardia* infections in calves reported lack of success in preventing giardiasis or reducing cyst excretion in this species.\(^1^0^4\) Chemotherapy treatment is controversial. Giardiasis in ruminants is likely to be chronic and, because there are high levels of *Giardia* cysts in the farm environment, reinfections are common, requiring repeated treatments that make chemotherapy cost-prohibitive for producers. In addition, repeated treatments increase the potential for developing drug resistance. Some studies have reported that the use of fenbendazole and albendazole (currently approved as anthelmintics) and paromomycin (broad-spectrum antibiotic) were effective and reduced cyst excretion, improved fecal consistency, and resulted higher weight gains.\(^1^0^5–1^0^8\) However, another study found conflicting evidence and reported a higher risk of *Giardia* infection in calves associated with the administration of anthelmintics.\(^1^0^9\)

Limiting the spread of *Giardia* among ruminants is difficult, and longitudinal studies indicate that most animals eventually become infected. However, improved husbandry and good management can be beneficial and can decrease cyst numbers in the farm environment, thus reducing the risk of transmission. These practices should include regular cleaning and disinfection, the use of floor surfaces that are easy to clean in barns, prompt removal of feces from animal housing, use of single-cow calving areas, and adequate intake of colostrum by neonatal animals to ensure adequate transfer of passive immunity.\(^8^9,1^0^9\)

**SUMMARY**

Infections with *G. duodenalis* and *Cryptosporidium* species are common in ruminants, and most animals become infected within the first months of life. Both parasites are
causal agents of diarrhea in neonatal ruminants and have also been proved to have a negative impact on growth and performance, even in subclinical infections. In addition to potential production losses, some \textit{G. duodenalis} assemblages and \textit{Cryptosporidium} species frequently identified in ruminants are zoonotic, indicating that ruminants may serve as a potential source for human infections either through direct contact with infected animals or through ingestion of cysts or oocysts via contaminated food or water. Control measures based on good husbandry management practices would be beneficial for both animal health and public health.

\textbf{DISCLOSURE}

The author has nothing to disclose.

\textbf{REFERENCES}


