

PREVALENCE AND CLINICAL IMPORTANCE OF *CRYPTOSPORIDIUM* AND *GIARDIA* IN HUMAN AND ANIMALS

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ABSTRACT

The protozoan parasites *Cryptosporidium* and *Giardia duodenalis* are worldwide considered as an important cause of gastrointestinal disease in human patients and in animals. The high number of (oo) cysts excreted shortly after infection, together with the low infectious dose, results in an easy spread of infection. The aim of this literature review is to introduce *Cryptosporidium* and *Giardia* by addressing their taxonomy, life cycle, prevalence and clinical importance for both human and animals.

Key words: Prevalence, *Cryptosporidium*, *Giardia*, Clinical importance

INTRODUCTION

Access to safe drinking water and basic sanitation is a fundamental human right, but currently more than one billion people worldwide do not have access to either safe drinking water or adequate sanitation. It is estimated that almost 60% of deaths following diarrhoeal diseases in developing countries are attributable to lack of access to safe drinking water and basic sanitation, children under 5 being at the highest risk. Important water-borne diarrhoeal diseases are cryptosporidiosis and giardiasis (Xiao and Fayer, 2008; Geurden *et al.*, 2009). These diseases are caused by the protozoan parasites *Cryptosporidium* and *Giardia*, respectively, which are able to cause disease in humans and animals. Because of their impact on socio-economic development, especially in developing countries, both *Cryptosporidium* and *Giardia* are since 2004 included in the 'Neglected Disease Initiative' of the World Health Organization (WHO) (Savioli *et al.*, 2006).

However, *Cryptosporidium* and *Giardia* also pose an important risk on the safety of drinking water in developed countries. For example, in 1993 more than 400,000 people were affected by cryptosporidiosis in Milwaukee (Wisconsin, USA) due to an ineffective filtration process in the production of drinking water. Since this outbreak screening of tap water for the presence of *Cryptosporidium* has become compulsory in the UK, The Netherlands and the USA, but water-borne outbreaks are still reported on a regular basis.

The aim of this literature review is to introduce *Cryptosporidium* and *Giardia* by addressing their taxonomy, life cycle, prevalence and clinical importance for both human and animals.

Prevalence of *Cryptosporidium* in humans

In developed countries, the prevalence of *Cryptosporidium* generally is low in asymptomatic people (<1%) and in patients with diarrhoea (1-2%) (Current and Garcia, 1991; Guerrant, 1997; Geurden *et al.*, 2009). In developing countries, high rates of asymptomatic carriage (10-30%) are common in comparison to patients with gastroenteritis (3-20%) (Current and Garcia, 1991; Haque *et al.*, 2003). Among the common *Cryptosporidium* species in humans, *C. parvum* and *C. hominis* are responsible for >90% of human cases of cryptosporidiosis in developed nations (Xiao and Feng, 2008). The distribution of *C. parvum* and *C. hominis* in humans differs between geographic regions. In Europe, both *C. parvum* and *C. hominis* are common in humans (Leoni *et al.*, 2006; Chalmers *et al.*, 2009; Zintl *et al.*, 2009). In the Middle East, *C. parvum* is the dominant species in humans

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(Sulaiman *et al.*, 2005; Pirestani *et al.*, 2008). Geographic variations in the distribution of *C. parvum* and *C. hominis* can also occur within a country. For example, *C. parvum* is more common than *C. hominis* in rural areas in the United States and Ireland (Feltus *et al.*, 2006; Zintl *et al.*, 2009). In the rest of the world, especially developing countries, *C. hominis* is usually the predominant species in humans, responsible for 70-90% of the infections (Xiao and Feng, 2008). This suggests that zoonotic infection is much less common in developing countries than in developed countries.

Temporal and age-associated differences in the distribution of *C. parvum* and *C. hominis* infections have been reported. Peaks in *Cryptosporidium* infections have been observed in spring and late summer (Casemore, 1990). *C. parvum* was more prevalent in spring (in Ireland, the United Kingdom and New Zealand) and *C. hominis* was more prevalent in autumn (in the Netherlands, the United Kingdom and New Zealand) (McLauchlin *et al.*, 2000; Learmonth *et al.*, 2003, 2004; Hunter *et al.*, 2004; Wielinga *et al.*, 2008; Chalmers *et al.*, 2009; Zintl *et al.*, 2009). In The Netherlands, *C. hominis* was more commonly found in children and *C. parvum* more in adults (Wielinga *et al.*, 2008). In the UK, *C. hominis* was more prevalent in infants less than one year, females aged 15-44 years and international travelers (Chalmers *et al.*, 2008, 2009). *C. viatorum* was identified among travellers with gastro-intestinal symptoms returning to Great Britain from the Indian subcontinent (Elwin *et al.*, 2012). In South American countries, a relatively high proportion of *C. meleagridis* infections has been identified in children and in immunocompromised patients (Cama *et al.*, 2007, 2008; Meireles, 2010).

Clinical importance

Cryptosporidium is reported to infect people in at least 106 countries (Fayer, 2008). The most common clinical feature of cryptosporidiosis is diarrhoea. Characteristically, the diarrhoea is profuse and watery; it may contain mucus but rarely blood and leucocytes and it is often associated with weight loss. Other less common clinical features include abdominal pain, nausea, vomiting and low-grade fever. Occasionally, nonspecific symptoms such as myalgia, weakness, malaise, headache and anorexia occur (Current and Garcia, 1991).

The severity of a *Cryptosporidium* infection can vary from an asymptomatic shedding of oocysts to a severe and life-threatening disease. The duration and the severity of the symptoms and the outcome may vary with host factors such as the immune status of the person. Most immunocompetent persons experience a short-term illness with complete and spontaneous recovery (Current and Garcia, 1991). However, for immunocompromised patients, cryptosporidiosis can be a critical illness with persistent symptoms leading to dehydration and wasting (O'Donoghue, 1995; Chen *et al.*, 2002; Blackburn *et al.*, 2004), and eventually leading to death (Juraneck, 1995; Manabe *et al.*, 1998). In addition, *Cryptosporidium* infections can cause atypical manifestations in immunocompromised patients, such as biliary tract disease, respiratory tract disease and pancreatitis (Hunter and Nichols, 2002).

The severity of the infection is also related to the age of the patient. Diarrhoea is a leading cause of illness and death among children aged <5 years in developing countries and *Cryptosporidium* is one of the most important diarrhoeal pathogens (Shirley *et al.*, 2012). Children are more likely to be infected with *Cryptosporidium*, which can be explained by a lack of an effective immunity at this age. In a study of 191 children with *C. parvum* in Uganda, 13% died, compared with 6% for children without *C. parvum* (Tumwine *et al.*, 2003). Wielinga *et al.* (2008) found that the majority (80%) of the human cases were children aged between 0 and 9 years and >70% of these were caused by *C. hominis*. *C. hominis* is more common than *C. parvum* in children and is associated with heavier infections and greater growth shortfalls, even in the absence of symptoms (Bushen *et al.*, 2007). Patients >25 years of age were infected mainly with *C. parvum*.

The clinical symptoms may also depend on the parasite species involved. Infections with *C. hominis* are associated with diarrhoea, nausea, vomiting, malaise and non-intestinal sequelae such as joint pain, eye pain, recurrent headache and fatigue, whereas infections with *C. parvum*, *C. meleagridis*, *C. canis* and *C. felis* cause only diarrhoea (Bouzig *et al.*, 2013).

Prevalence of *Cryptosporidium* in animals

Cattle are commonly infected with *C. parvum*, *C. andersoni*, *C. bovis* and *C. ryanae* (Xiao, 2010). In dairy cattle, *C. parvum* is mostly found in pre-weaned calves, *C. bovis* and *C. ryanae* in weaned calves and *C. andersoni* in yearlings and adult cattle (Fayer *et al.*, 2006b, 2007; Santín *et al.*, 2008). Parasite prevalence varies from 1% (Kvác *et al.*, 2006) to 59% (Olson *et al.*, 1997) in individual calves and up to 100% on farm level

(Santín *et al.*, 2004). The highest prevalence is observed in calves under the age of 5 weeks (Quílez *et al.*, 1996). The prevalence of *Cryptosporidium* in flocks of small ruminants varies considerably, ranging from 5% to 70% for sheep and from 5% to 35% for goats. This difference in prevalence results can be explained by differences in age of the animals, management, and diagnostic methods applied (Robertson, 2009). *C. xiaoi*, *C. ubiquitum* and *C. parvum* are the predominant species in small ruminants (Ryan *et al.*, 2005; Goma *et al.*, 2007; Santín *et al.*, 2007; Geurden *et al.*, 2008a; Mueller-Doblies *et al.*, 2008; Pritchard *et al.*, 2007, 2008; Quílez *et al.*, 2008; Yang *et al.*, 2009; Díaz *et al.*, 2010; Wang *et al.*, 2010; Tzanidakis *et al.*, 2014).

In pigs, herd prevalences range from 8% to 100% with individual animal infection rates of between 1% and 34%. *C. suis* (36-83%) and *C. scrofarum* (formerly *Cryptosporidium* pig genotype II) (9-61%) are the major *Cryptosporidium* spp. (Kváč *et al.*, 2009; Chen *et al.*, 2011; Budu-Amoako *et al.*, 2012). *C. suis* preferentially infects suckling piglets, whereas *C. scrofarum* is more frequently found in weaners (Langkjaer *et al.*, 2007; Johnson *et al.*, 2008; Kváč *et al.*, 2009; Yin *et al.*, 2013; Zhang *et al.*, 2013). Occasionally, *C. muris* (Kváč *et al.*, 2009; Budu-Amoako *et al.*, 2012; Němejc *et al.*, 2013) or *C. tyzzeri* (Kváč *et al.*, 2012) are found in pigs. With the exception of one study where *C. parvum* was the predominant species (Farzan *et al.*, 2011), this species is less frequently found in pigs, suggesting that pigs are not an important source of zoonotic transmission (Chen and Huang, 2007; Zintl *et al.*, 2009; Budu-Amoako *et al.*, 2012; de la Fé Rodríguez *et al.*, 2013; Němejc *et al.*, 2013).

Low prevalences of *Cryptosporidium* have been reported in horses in the USA (7%) and Italy (8%) (Burton *et al.*, 2010; Veronesi *et al.*, 2010). Both *C. parvum* and *Cryptosporidium* horse genotype were found in horses (Ryan *et al.*, 2003; Chalmers *et al.*, 2005; Grinberg *et al.*, 2008; Veronesi *et al.*, 2010) and rarely the hedgehog genotype (Laatamna *et al.*, 2013). These findings support a potential role of infected horses in zoonotic transmission.

A *Cryptosporidium* prevalence ranging from 0% to 13% has been reported in privately owned and stray dogs (Chermette and Blondel, 1989; Grimason *et al.*, 1993; Díaz *et al.*, 1996; Giangaspero *et al.*, 2006; Claerebout *et al.*, 2009; Yoshiuchi *et al.*, 2010; Bajer *et al.*, 2011). Most infections in dogs are caused by the host-specific *C. canis*. In addition to *C. canis*, other *Cryptosporidium* spp. were detected occasionally in dogs such as *C. Muris* (Lupo *et al.*, 2008; Ellis *et al.*, 2010), *C. Parvum* (Hajdušek *et al.*, 2004; Giangaspero *et al.*, 2006; Sotiriadou *et al.*, 2013) and *C. Meleagridis* (Hajdušek *et al.*, 2004). Thus the risk of zoonotic transmission from *Cryptosporidium*-infected dogs is low (Lucio-Foster *et al.*, 2010; Uehlinger *et al.*, 2013).

Cryptosporidium has been detected in cats with a range of 2% to 25% (Rambozzi, *et al.*, 2007; Hoopes *et al.*, 2013). In addition to the cat specific species, *C. felis*, *C. parvum* and *C. muris* were also identified (Palmer *et al.*, 2008; Yoshiuchi *et al.*, 2010; FitzGerald *et al.*, 2011; Scorza *et al.*, 2011; Sotiriadou *et al.*, 2013).

Veterinary importance

C. parvum is a well-known cause of diarrhoea in neonatal ruminants. Clinical symptoms are most frequently observed in calves between the age of 5 days and 1 month and include profuse watery diarrhoea with acute onset, lethargy, anorexia and dehydration, which is usually self-limiting within 2 weeks (O'Handley *et al.*, 1999, Schnyder *et al.*, 2009). Mortality is variable and is most often observed in calves with multiple infections and in certain beef breeds, such as the Belgian Blue and White (de Graaf *et al.*, 1999) and it can be as high as 30% (Olson *et al.*, 2004). However, morbidity in endemic herds can be as high as 100% (Santín *et al.*, 2008). Abomasal cryptosporidiosis, caused by *C. andersoni*, does not result in any visible clinical signs (Kváč *et al.*, 2008). Infections with *C. andersoni* do not cause diarrhoea and follow a more chronic course than infections with *C. parvum* (Kváč and Vitovec, 2003). *C. andersoni* infections may result in a decrease in daily weight gain, decreased feed efficiency and less milk production (Anderson, 1987; Esteban and Anderson, 1995; Ralston *et al.*, 2003).

Cryptosporidium is a major cause of neonatal diarrhoea in lambs, usually within the first 2 weeks of life and diarrhoea can be mild to severe. Cryptosporidiosis outbreaks in lambs are most common in crowded flocks and are associated with a decrease in liveweight, dressing percentage, growth rate and carcass productivity (Angus *et al.*, 1982; Alonso-Fresán *et al.*, 2005; Sweeny *et al.*, 2011; de Graaf *et al.*, 1999; Sari *et al.*, 2009). Caprine cryptosporidiosis is characterised by diarrhoea and mortality in kids. Morbidity can reach 100% and mortality 50% in some herds (Vieira *et al.*, 1997; Johnson *et al.*, 1999; Sevinc, *et al.*, 2005; Paraud *et al.*, 2010; Santín, 2013).

Diarrhoea is the major clinical sign in foals affected by cryptosporidiosis. Foals are more susceptible to the infection than older animals (Grinberg *et al.*, 2003; 2009; Veronesi *et al.*, 2010) and most *Cryptosporidium* infections in adult horses are asymptomatic (Majewska *et al.*, 2004; Sturdee *et al.*, 2003).

Inappetance, depression, vomiting and/or diarrhoea developed in piglets experimentally infected with *C. parvum*, whereas mild or no clinical signs developed with *C. suis* (Enemark *et al.*, 2003). However, an association between diarrhoea and infections with *C. suis* and *C. scrofarum* in nursing piglets has been described (Hannes *et al.*, 2007). In contrast, other studies did not find any significant association between diarrhoea and cryptosporidial infections (Quilez *et al.*, 1996; Maddox-Hyttel *et al.*, 2006; Vitovec *et al.*, 2006).

Cryptosporidiosis in dogs has been reported in both asymptomatic and diarrhoeic dogs (Santín and Trout, 2008). Infections with *C. canis* are usually asymptomatic but severe diarrhoea, malabsorption, weakness and weight loss have been reported (Irwin, 2002; Miller *et al.*, 2003). Dogs infected with *C. muris* showed chronic vomiting and profuse diarrhoea in one study (Ellis *et al.*, 2010) but in another study no gastrointestinal signs were observed (Lupo *et al.*, 2008).

Cryptosporidium oocysts were detected more frequently in cats without diarrhoea than in cats with diarrhoea (Sabshin *et al.*, 2012) and shedding of *Cryptosporidium* oocysts without the presence of clinical signs was reported in experimentally and naturally infected cats (Mtambo *et al.*, 1991; Nash *et al.*, 1993; Fayer *et al.*, 2006c). However, oocysts were also detected in the faeces of cats with persistent diarrhoea (Goodwin and Barsanti, 1990; Lent *et al.*, 1993; Morgan *et al.*, 1998).

Prevalence of *Giardia* in humans

In developed countries *Giardia* is detected in up to 14% of symptomatic patients and 2% in asymptomatic humans (Geurden *et al.*, 2009; Homan and Mank, 2001). In developing countries, the prevalence of giardiasis in patients with diarrhoea is around 20%, ranging from 5-43% (Islam, 1990; Haque *et al.*, 2005). *Giardia* assemblages A and B are considered more infectious for humans, with the latter being more prevalent. Sub-assemblage AII is more prevalent in humans than AI and is distributed globally, except in Asia and Australia. Assemblage AIII has not yet been detected in humans (Sprong *et al.*, 2009). The geographic distribution of sub-assemblages BIII and BIV in human shows marked difference between continents. In Africa, infection with BIII is more prevalent (81%) than with BIV, whereas the opposite is found in North America where 86% of infections are associated with BIV and 14% with BIII. A more balanced distribution was found in Australia and Europe (Sprong *et al.*, 2009). To a much lesser extent, assemblage C, D, E and F were identified in human samples (Gelanew *et al.*, 2007; Foronda *et al.*, 2008). However, it remains unclear whether the presence of these assemblages in human stool is due to patent infections or merely represents passage through the intestinal tract.

Clinical importance

Approximately 200 million people in Asia, Africa and Latin America have symptomatic infections with about 50,000 cases reported each year (Xiao and Fayer, 2008). In symptomatic patients, mostly children, the severity of symptoms and the duration of *Giardia* infection are highly variable. In some patients, symptoms last for only 3 or 4 days, while in others the symptoms last for months. Higher prevalence of chronic *Giardia* infection in patients with immunodeficiency supports that the failure to develop an effective immune response against *Giardia* may account for the chronicity of the infections (O'Handley *et al.*, 2003). In developed countries, the incidence rate peaks at the age of 1-4 years (Flannagan, 1992); a second peak is observed at the 20-40 age groups, partly due to the care for the young children and partly due to travelling (Medema, 1999). The main symptoms include diarrhoea, abdominal pain, nausea, vomiting, flatulence, anorexia and fever (Nash *et al.*, 1987; Farthing, 1996; Katz *et al.*, 2006). In most instances the diarrhoeal illness is short-lived and self-limited. However, a proportion of individuals develop persistent diarrhoea (Farthing, 1996; Katz *et al.*, 2006), sometimes accompanied by malabsorption of sugars and fat and by weight loss. There is evidence that infection with *Giardia* results in 'failure to thrive' in children, by impairment of the uptake of nutrients (Farthing, 1994; Hall, 1994). A high prevalence of chronic fatigue syndrome has been reported as a post-infection sequel in patients (Naess *et al.*, 2012; Wensaas *et al.*, 2012; Mørch *et al.*, 2013).

Prevalence and clinical importance of Cryptosporidium and Giardia

The relation between clinical symptomatology and the *Giardia* genotype is controversial. In a study in The Netherlands, assemblage A isolates were solely detected in patients with intermittent diarrhoeal complaints, while assemblage B isolates were present in patients with persistent diarrhoeal complaints (Homan and Mank, 2001). A strong correlation between infection with assemblage B and diarrhoea was observed in Saudi children (Al-Mohammed, 2011). An association between assemblage B and flatulence in children was reported by Lebbad *et al.* (2011). In contrast, Read *et al.* (2002) found that assemblage B genotypes were more prevalent in asymptomatic children than those of assemblage A and according to Haque *et al.* (2005, 2009) only assemblage A was an important cause of diarrhoea in children in Bangladesh. A systematic review and meta-analysis confirmed that *Giardia* infections of both assemblages A and B can cause acute or persistent diarrhoea (Muhsen and Levine, 2012).

However, *Giardia* infections are often asymptomatic. In some studies no significant association between *Giardia* and diarrhoea was found (Guerrant *et al.*, 1983; Schorling *et al.*, 1990; Hollm-Delgado *et al.*, 2008; Boeke *et al.*, 2010). In both volunteers and outbreak situations, a sizable proportion of the infected subjects are asymptomatic, often exceeding the proportion with manifest clinical illness (Muhsen and Levine, 2012). It has been estimated that between 50% and 75% of *Giardia*-infected persons may be asymptomatic (USEPA, 1998a). Children with asymptomatic *Giardia* infection serve as unidentified carriers and may be responsible for transmission of the infection. Secondary transmission among family members may occur. Asymptomatic infections may last for months or years (ICAIR, 1984).

Prevalence of *Giardia* in animals

In calves younger than six months, the prevalence varies between 17% (Muhid *et al.*, 2012) and 73% (Olson *et al.*, 1997) and on farm level it can be as high as 100% (Olson *et al.*, 1997; Hunt *et al.*, 2000; Geurden *et al.*, 2010, 2012). In cattle the livestock specific assemblage E is most prevalent, although up to 59% zoonotic assemblage A isolates and mixed infection with both A and E have been reported (Geurden *et al.*, 2008b; Sprong *et al.*, 2009). This suggests that calves should be considered as a potential reservoir for human infections. However, within assemblage A, sub-assemblage AI is predominantly found in livestock and companion animals, while assemblage AII is more prevalent in humans (Sprong *et al.*, 2009). The prevalence of *Giardia* in sheep and goats is reported to range from 10% to 40% with assemblage E detected in 75%, assemblage A in 27% and assemblage B in 2% of cases (Robertson, 2009).

The prevalence of *Giardia* in pigs ranges from 1% to 51% (Armson *et al.*, 2009; Farzan *et al.*, 2011; Budu-Amoako *et al.*, 2012). The herd prevalence was 18%, 22% and 84% for sows, piglets and weaners in Denmark (Maddox-Hyttel *et al.*, 2006) and 12% in Zambia (Siwila and Mwape *et al.*, 2012). DNA sequencing demonstrated that assemblage E was the most common genotype in Australia and the UK (Armson *et al.*, 2009; Minetti *et al.*, 2013), while in Canada assemblage B was predominant (Farzan *et al.*, 2011). Assemblage A was found in both weaners and piglets in Denmark (Langkjaer *et al.*, 2007) and in pigs in Australia (Armson *et al.*, 2009). Unexpectedly, the canine-specific assemblages C and D and the feline-specific assemblage F were also found occasionally in pigs in different countries (Langkjaer *et al.*, 2007; Armson *et al.*, 2009; Minetti *et al.*, 2013). As for humans, it remains unclear whether the presence of these assemblages represents a patent infection or merely indicates carriage.

The prevalence of *Giardia* in horses was 1% in Brazil (De Souza *et al.*, 2009), 9%-23% in Italy (Veronesi *et al.*, 2010; Traversa *et al.*, 2012) and 17% in Colombia (Santín *et al.*, 2013). *G. duodenalis* isolates from horses belonged predominantly to the zoonotic assemblages A and B (Traub *et al.*, 2005; Traversa *et al.*, 2012; Santín *et al.*, 2013) and to a lesser extent to assemblage E (Veronesi *et al.*, 2010; Traversa *et al.*, 2012).

In dogs, the prevalence of *Giardia* infections varies from 1% to 55% (Itoh *et al.*, 2005; Jafari Shoorijeh *et al.*, 2008). The most prevalent assemblages in dogs are the dog-specific assemblages C and D (Beck *et al.*, 2012) but other sub-assemblages such as AI, AII, BIII and BIV are also detected in dogs worldwide (Souza *et al.*, 2007; Palmer *et al.*, 2008; Claerebout *et al.*, 2009; Sprong *et al.*, 2009), sometimes in higher frequencies than the dog-specific assemblages (*e.g.* Leonhard *et al.*, 2007; Claerebout *et al.*, 2009; Covacin *et al.*, 2011).

Worldwide the prevalence of *Giardia* in cats ranges from 1%-40% (De Santis-Kerr *et al.*, 2006; Itoh *et al.*, 2006; Gow *et al.*, 2009; Mohsen and Hossein, 2009; Mircean *et al.*, 2011; Sabshin *et al.*, 2012). Assemblage F and sub-assemblage AI are predominant but assemblage D and sub-assemblages AII, AIII were also detected in cats (Papini *et al.*, 2007; Souza *et al.*, 2007; Palmer *et al.*, 2008; Sprong *et al.*, 2009).

Veterinary importance

Although *G. duodenalis* is recognised worldwide as the most common parasitic cause of gastrointestinal disorder in human patients, the relevance of infection in production animals is open to debate (Geurden *et al.*, 2010). The clinical signs may vary considerably between animals and animal species due to the involvement in the pathogenesis of giardiasis of both parasite and host factors. This lack of consistency in clinical outcome resulted in the perception that *Giardia* is not a major cause of clinical disease in ruminants. However, several studies reported clinical signs caused by *Giardia* both in natural infections (St. Jean, 1987; O'Handley *et al.*, 1999; Aloisio *et al.*, 2006; Geurden *et al.*, 2006b) and in experimental infections (Olson *et al.*, 1995; Geurden *et al.*, 2006a). Infection can result in diarrhoea that does not respond to antibiotic or coccidiostatic treatment. The excretion of pasty to fluid faeces with a mucoid appearance may be indicative for giardiasis, especially when the diarrhoea occurs in young animals.

A study in dairy calves showed that calves did not begin to excrete *Giardia* cysts until approximately 1 month of age. Passive immunity through colostrum may have the potential to provide initial protection against *Giardia* infections as colostrum contains a high level of anti-*Giardia* antibodies. Failure to develop a humoral immune response from natural infections by these calves could account for the high prevalence and chronic duration of the infections (O'Handley *et al.*, 2003).

In pigs, a significant association was found between the presence of assemblage E and soft to diarrhoeic stool, whereas assemblage A was not correlated with diarrhoea (Armson *et al.*, 2009). This is in contrast to previous studies that have reported no association between *Giardia* infections in pigs and diarrhoea (Maddox-Hyttel *et al.*, 2006, Hammes *et al.*, 2007; Langkjaer *et al.*, 2007). Next to diarrhoea, there is an economic impact of giardiasis for farmers. In goat kids and lambs an experimental infection resulted in a decreased feed efficiency and subsequently a decreased weight gain (Olson *et al.*, 1995; Sweeney *et al.*, 2011, 2012). Infections with *Giardia* in dogs and cats are common. Clinical signs vary from asymptomatic to small bowel diarrhoea and associated discomfort (Fiechter *et al.*, 2012).

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