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Giardiasis: Livestock and Companion Animals

Joyce Siwila

Abstract

*Giardia* spp. are flagellates that are found in the intestinal tract of humans and domestic and wildlife animals, including birds and amphibians, worldwide. The genus *Giardia* comprises several species which are morphologically similar. *Giardia* infections have been reported widely in livestock and companion animals with varying prevalence in different parts of the world. Giardiasis, the disease caused by *Giardia*, may result in numerous episodes of diarrhoea, especially in young animals, which in turn adversely affect production resulting in economic losses. The affected animals may also act as a source of zoonotic infections. Evidence of infection in both animals and humans of *Giardia duodenalis* especially of assemblage A and B has firmly established giardiasis as a zoonotic disease. The zoonotic assemblage A and B have been reported in livestock (cattle, sheep, goats, pigs) and companion animals (dogs, cats, horses). However, questions regarding the direct transmission of *Giardia* from domestic animals to humans still need to be explored. Appropriate prevention and control measures are cardinal in preventing both animal and human infections. This chapter discusses *G. duodenalis* infection and the disease including treatment options in livestock and companion animals.

Keywords: *Giardia*, *Giardia duodenalis*, giardiasis, livestock, companion animals, treatment

1. Introduction

For years, man has relied on livestock for food, drought power, hides and other production activities. In less developed countries, livestock production is mostly done using traditional methods due to limited resources and these small-scale production systems accounts for most agricultural output in these countries [1]. On the other hand, companion animals are equally kept throughout the world. Dogs and cats are particularly kept as pets with increasing numbers in nations that previously did not do so but kept dogs mostly for security. Now, dogs are
widely used for different purposes, including companionship, life-saving actions, security as well as hunting and farming [2, 3]. Other than entertainment and sports, horses are also being kept for companionship. These livestock and companion animals are however hosts to many parasites, some of which have detrimental effects on the health and productivity of those affected. Protozoa such as \textit{Giardia duodenalis} affect a wide range of domestic and wild animals, with serious clinical consequences especially in young animals.

\textit{G. duodenalis} (syn. \textit{Giardia lamblia}, \textit{Giardia intestinalis}), a flagellate protozoan parasite, and the aetiological agent of giardiasis, is one of the most prevalent and widespread intestinal parasite in humans and several vertebrate animal species worldwide [4]. The taxonomy of the genus is mainly based on morphology and genetic evidence. According to these criteria, six species have been recognised in the genus \textit{Giardia} and these include \textit{G. duodenalis} in humans and other mammals, \textit{G. agilis} in amphibians, \textit{G. muris} and \textit{G. microti} in rodents, \textit{G. psittaci} and \textit{G. ardeae} in birds. In recent years, phylogenetic analysis and enzyme electrophoresis have revealed the existence of eight assemblages A–H within the species \textit{G. duodenalis} [5–7]. \textit{Giardia} from humans appears to fall exclusively into Assemblage A and B while C and D are dog specific assemblages. Assemblage E is isolated from hooved animals, a characteristic of isolates from sheep, goats, cattle and pigs [8]. Cats are hosts for Assemblage F while rats are hosts for Assemblage G [9, 10]. Assemblage H has been reported in the grey seal [11].

\textit{G. duodenalis} is a frequently encountered intestinal parasite of domestic animals, especially livestock, dogs and cats. \textit{Giardia} infections have been reported widely in livestock and companion animals with varying prevalence in different parts of the world, but high frequency was mostly in dairy calves [12–16]. As a parasite, \textit{Giardia} has a broad host range, however, the adverse consequences of infection and its pathogenic potential are best recognised in humans [6]. It causes an estimated $2.8 \times 10^8$ human cases per annum [17]. In Asia, Africa and Latin America, about 200 million people have symptomatic giardiasis with some 500,000 new cases reported each year [18]. Its simple life cycle involving an environmentally resistant cyst (Figure 1) provides greater opportunities for the parasite to be transmitted directly from one infected individual to another, or indirectly through contamination of the environment or food [4].

\textbf{Figure 1.} \textit{Giardia} cyst: wet smear stained with iodine (source: https://www.cdc.gov/dpdx/giardiasis/index.html).
2. Transmission and clinical disease

The cyst is the infective stage and represents the resting stage of the organism. Its rigid outer wall protects the parasite against changes in environmental temperature, dehydration and chlorination, all of which would destroy the trophozoite [6, 19, 20]. Transmission occurs by the faecal-oral route, either by direct contact with an infected host, or through contaminated food or water [21, 22]. Mechanical transmission of the parasite through insect vectors has also been reported [23]. Factors that facilitate infection include overcrowding, the high excretion of cysts by infected animals and the low infectious dose (between 10 and 25 cysts) [24, 25].

*Giardia* is not invasive and therefore lives and multiplies by asexual multiplication on the luminal surface of the small intestine of the vertebrate host [6]. Although the pathogenesis of *Giardia* is not completely understood, the pathophysiological process is initiated by infection with the parasite resulting in variable clinical signs such as abdominal pain, diarrhoea and weight loss [26]. A rise in numbers of intraepithelial lymphocytes increases epithelial permeability. Activation of T-lymphocytes has also been observed in *Giardia* infections [27, 28]. Trophozoite toxins and T-cell activation initiate a diffuse shortening of brush border microvilli and decreased activity of the small intestinal brush border enzymes, particularly lipase, proteases and disaccharidases [29–31]. The microvillus shortening leads to a decrease in overall absorptive area in the small intestine and an impaired uptake of water, electrolytes and nutrients resulting in malabsorptive diarrhoea [29, 32]. The steatorrhea and mucous diarrhoea usually observed in giardiasis are attributed to reduced activity of lipase and increased production of mucin by goblet cells [33]. Severity of the disease is dependent on factors like developmental, nutritional and immunity of the host as well as virulence factors of the parasite [30, 34, 35]. Although gross intestinal lesions are rarely observed, microscopic lesions consisting of villous atrophy and cuboidal enterocytes may be reported [33].

3. Giardiasis in livestock

3.1. Cattle

In cattle, *Giardia* is considered an important emerging parasite of dairy cattle and also as a cause of zoonotic disease with negative effect on public health [19]. Calves have been reported to be infected with *G. duodenalis* as early as 4 days of age, and the highest intensity of cyst excretion of $10^5$–$10^6$ cysts per gram of faeces between the ages of 1 and 3 months has been documented [36, 37]. A periparturient rise in cyst excretion has also been demonstrated [37]. Transmission occurs among infected calves as well as chronically infected adults [12, 38, 39] and is particularly high among dairy calves [38, 39]. There are four main proposed cycles of transmission that are believed to maintain host-specific and zoonotic assemblages of *Giardia* in mammalian hosts: human cycle, livestock cycle, dog/cat cycle and wildlife cycle (Figure 2).
The livestock cycle is thought to maintain Assemblage E within the livestock group [6, 40, 41]. The other cycles maintain the assemblages in the specific hosts. For example, assemblages A and B can be maintained by direct transmission between humans, assemblage C and D between dogs (e.g. puppies in a breeding kennel) and wildlife genotypes among various wildlife species. Some assemblages, however, infect other animal species and humans. The frequency of transmission is however not very clear and still under debate. Zoonotic species have been reported in wildlife, but their role as a potential reservoir for human infection still requires further molecular epidemiological research [4].

The resultant giardiasis from *G. duodenalis* infection can result in diarrhoea that does not respond to treatment with antibiotic or anti-coccidia drugs [33, 36, 42]. *Giardia* has been implicated as an aetiological agent alone and in combination with other enteric pathogens in calf diarrhoea [36, 38, 43, 44]. Infection may also result in numerous diarrhoea episodes which in turn adversely affects production and result in economic losses for farmers [45]. In younger calves, especially below 6 months of age, the excretion of watery faeces with a mucoid appearance may be the only indication of infection with the parasite. Chronic cases of giardiasis in
calves may impact negatively on performance which may be reflected in reduced weight gain, impaired feed efficiency and decreased carcass weight. This was demonstrated in experimentally infected lambs [43].

*Giardia* has been found in both beef and dairy cattle throughout the world with varying prevalence. Infection rates can be as high as 100% [36–38, 46–50]. The infection pattern of *Giardia* appears similar between beef and dairy cattle [36, 37] with cysts appearing in the faeces at approximately 4 weeks of age [12, 36, 38]. Both dairy and beef calves may harbour more than one genotype of *G. duodenalis*, which can be of zoonotic significance [12, 51, 52]. Assemblages A, B and E have been detected in cattle; Assemblages A and B also infect humans [53, 54]. As calves infected with *Giardia* shed large numbers of cysts, there is concern that cattle could represent a reservoir of *G. duodenalis* with the potential to cause disease in humans either through direct contact or by contamination of food and/or water supplies [36]. Because of the risk of contamination of water supplies by water borne parasites such as *Giardia*, it is normally recommended that animal facilities should be located away from streams, lakes, dams and rivers whenever possible, and waterways should be fenced-off in pasture lands in order to prevent possible run-off into these water sources [55].

### 3.2. Sheep

The prevalence of *G. duodenalis* infection in sheep varies considerably and may be as high as 38% in adult sheep and 68% in lambs [56–60]. In a study in central China [61], the prevalence of *G. duodenalis* was 12.36% in pre-weaned lambs and 5.74% in post-weaned sheep [61]. Other studies have also reported great variability in *Giardia* prevalence: in Canada, prevalence of giardiasis was higher in lambs (57%) than in adults (9%) [62]; in Brazil, lambs had a 32% infection rate while that for ewes was 2%; [63]; and in Mongolia, China, lambs had a significantly higher infection rate than ewes (8.6 versus 0.9%, respectively) [64]. All the findings from these studies suggest that the infection rates of *Giardia* tend to decline as the age of the animals increases. However, the opposite has also been reported. In some studies in Australia, a much higher prevalence of was detected in post-weaned lambs and sheep (44%) than in pre-weaned sheep (11.1%) [56, 65]. In a study in Maryland, USA, the prevalence of giardiasis was higher in post-parturient ewes (12%) than in lambs (4%) [59]. Host age and immune status of the host affect the severity of the disease [6] but other factors such as the number of specimens examined, the age structure of the herds, management procedures and the health status of the animals may account for the discrepancies or variations in the infection rates in the different populations [61].

Because of the unexpectedly high levels of infection in sheep, sheep have long been considered a reservoir of human infections [56, 61, 66–68]. In most cases, infections are asymptomatic but infected animals are carriers shedding large numbers of cysts into the environment [58]. Even if most infections are asymptomatic, infections in lambs may result in a malabsorption syndrome, decreased feed efficiency and subsequently a decreased weight gain and sometimes death [19, 43, 69]. In the study by [69], excretion of malodorous and poorly formed faeces was observed. Furthermore, giardiasis may have a negative effect on time to slaughter of the sheep [19, 43] therefore, negatively affecting producers’ income.
Three assemblages of *G. duodenalis* have been recognised in sheep, livestock assemblage E, and the two zoonotic assemblages A and B [13, 56, 59]. The non-zoonotic assemblage E is the most frequently reported compared to the zoonotic ones [59, 66, 68, 69]. However, assemblage E appears to occur most frequently in cattle compared to other livestock; this was demonstrated by an extensive, longitudinal study of dairy herds in Australia over several months and another study in Canada [12, 56, 70].

### 3.3. Goats

In small ruminants, there are considerably more surveys from sheep populations than goat populations and therefore fewer publications on *Giardia* in goats. Furthermore, only a few molecular studies regarding *Giardia* have been performed worldwide [13, 58, 71–74] compared to other ruminant hosts (see [4]). In the reported studies, *Giardia* prevalence was reported to range from <10 to >40% depending on the age, geographical location and diagnostic technique used [75]. Infections are normally significantly higher in pre-weaned goat kids compared to that in older goat kids [74]. Most infections are asymptomatic, however, foul-smelling diarrhoea which is lightly coloured, greasy and mixed with mucous; reduced weight gain are clinical signs that may be observed, mostly in young animals that are symptomatic [71]. A study in Spain reported a high infection rate in young animals, agreeing with the hypothesis that to a great extent, young animals contribute to the environmental contamination with *Giardia* cysts [71]. A study in Nigeria also reported a high prevalence (46.9%) in goats with pre-weaned (≤3 months) goats having a much higher prevalence (58.1%) compared to those that were over 3 months (38.2%) [74].

Even though a large number of *G. duodenalis* genotyping studies in ruminants report a higher occurrence of genotype E, with genotypes A and B being less frequent [13, 58, 76, 77], other studies, [13, 72] have reported zoonotic genotype A infections in goats in Belgium and Côte d’Ivoire, respectively. In Malaysia, one study [73] reported genotypes A and B in goats. These findings suggest that goats could be a potential source of zoonotic infection.

### 3.4. Pigs

There is limited information on the *Giardia* infections in pigs. From the limited studies, *Giardia* infections have been reported in all age groups from nursing piglets to boars and sows worldwide, from Australia, Asia, Europe and North America, Africa with varying prevalence ranging between 0.1 and 20% [62, 78–86]. Natural infections are typically asymptomatic with no evidence of illness.

Both assemblages E and A have been identified in pigs with assemblage E being most common [4]. In one study in Australia, assemblage E was the most common genotype detected in positive specimens of both pre-weaned (64%) and post-weaned (67%) pigs [87]. In Denmark, assemblage E was also the most common genotype, being identified in 62% of samples from post-weaned pigs, while assemblage A was detected in only 12% of specimens [85]. Interestingly, the canine assemblage D has also been reported in pigs [85, 88].
Since pigs also harbour the zoonotic assemblage A, they should be considered as potential sources of infection. One case–control study in eastern England found an association between giardiasis and exposure to farm animals, pigs included [89].

4. Companion animals

4.1. Dogs and cats

*Giardia* is commonly recovered from the faeces of both symptomatic and asymptomatic dogs worldwide [90, 91]. Several studies have reported high prevalence of *Giardia* in stool samples of companion animals (i.e. cats and dogs) (reviewed by [92]). *Giardia* infection rates in dogs differ considerably based on many variables, including the composition of dog populations (owned/stray/kennel), the test used for diagnosis and its sensitivity. Similar to other animal species, severity of disease depends on host age and ability of the immunity to eliminate the infection. Reports of giardiasis range from 0.1% in owned dogs to as high as 100% in kennelled dogs, the risk factor being overcrowding and intensive contact between large numbers of dogs sharing the same shelter in kennelled dogs. This favours transmission of infections [6, 15, 93–96]. Some studies have indicated *Giardia* to be the most common enteric parasite of dogs and cats. For example, studies in Australia found that *G. duodenalis* was the most common enteric parasite of domestic dogs and cats [97, 98] while [99] also reported the parasite to be widely prevalent in dogs and cats in the USA. The prevalence of *Giardia* in these companion animals is however, believed to be underestimated because of the following reasons: the low sensitivity of the conventional detection methods, cyst excretion is intermittent and the disease is usually subclinical [98].

In most of the studies that have been conducted in dogs, puppies, free-roaming dogs, and shelter dogs have been shown to be at higher risk for infection than adult dogs and owned dogs [15, 94]. Transmission of the parasite appears to be maintained within the dog/cat cycle (Figure 2) as evidenced from the host specific assemblage C/D and F commonly isolated in dogs and cats respectively [15, 100]. However, zoonotic transmission of *Giardia* between humans and dogs in the same household has been reported previously [101]. In another study in Brazil, zoonotic assemblage A1 was isolated from dogs and children in the same locality suggesting the existence of a zoonotic cycle of the parasite in that community [102], and a study in Thailand revealed that dogs were a potential source of *Giardia* infections for humans [103]. In this study [103], assemblages A (79%) and B (21%) in addition to the dog specific assemblages C (12%) and D (31%) were isolated from the 104 dogs tested. In the United States, one study reported that 28 and 41% of client-owned dogs presenting with infection with *Giardia* to veterinary clinics had potentially zoonotic assemblages A and B, respectively, while 15 and 16% had host specific assemblages C and D, respectively [104]. The findings from the American study suggest the possibility of the potential for transmission of non-canine-specific assemblages from owners to their dogs as well as zoonotic transmission from dogs to humans. Furthermore, such reports highlight the possibility of two transmission cycles existing in domestic urban
environments, that is, transmission of dog-specific assemblages among dogs and the possible transmission of assemblage A between pets and humans. However, it has been reported that in household dogs, the frequency of dog-to-dog transmission may be lower because they are less crowded than kenneled dogs where prevalence is normally higher due to intensive contact among a large number of dogs [54, 91].

Although *Giardia* is common in dogs and cats, it is rarely associated with clinical disease and affected animals suffer minimal consequences of the disease, but may act as a source of zoonotic infection [103, 104]. However, complications such as persistent infections and impairment of growth and development may occur especially in young animals such as puppies and kittens [105]. Such infections with manifestation of clinical signs are usually associated with kennel or cattery setup, where there is overcrowding [106].

4.2. Horses

There is very few data on *Giardia* in horses and giardiasis is an uncommon condition in these animals. However, the parasite may be commonly found in faeces of asymptomatic animals. The parasite was first reported in horses in South Africa in 1921 [107]. Since then a number of reports have been made regarding the presence of the parasite in horses of all age groups. Relatively high rates of giardiasis among foals (17–35%) and lactating mares (1.9–27.8%) have been documented using the fluorescent antibody method [38]. Lower rates have been observed in weanlings (0–9.1%) [108]. Varying prevalence of giardiasis has been reported in different geographic areas and they differ considerably between locations [62, 109, 110] with age and physiological status of the animal playing an important role in the infection rates [38, 110].

Although giardiasis in horses has been found to be associated with diarrhoea, poor hair coat, ill thrift and weight loss [111, 112], infected horses rarely show any clinical signs [108] and no subclinical consequences have been reported previously. However, infected horses may show signs ranging from a mild and self-limiting to, occasionally, severe diarrhoea (with heavy infections). These are commonly seen in young and aged or immunologically suppressed horses [110, 113, 114]. However, some studies have reported no shedding of *Giardia* cysts in young and older horses [115].

*G. duodenalis* assemblages A, B, and E have been detected in horses [110, 116]. A study in Italy also confirmed the presence of both animal and human sub-assemblage of *G. duodenalis* in horses [117]. However, assemblage E appears to be more common in these animals [110]. Because assemblages A and B are known to infect humans [6, 118], horses could represent a reservoir of *G. duodenalis* with the potential to cause disease in humans through direct contact or by contamination of food and/or water supplies.

5. Diagnosis

The diagnosis of giardiasis is commonly established by microscopic identification of cysts or less commonly trophozoites in faecal specimens stained with trichrome (Figure 3) or iron
haematoxylin. This follows the application of faecal concentration techniques, especially zinc sulphate flotation and centrifugation [119]. Direct smear or wet mount examination for trophozoites can also be performed. However, because of the cyclical nature of cyst excretion, several samples need to be examined to detect the organism [120]. The disadvantage of microscopy is that it is of limited epidemiological value as it does not indicate the source of the infection [6].

Faecal immunoassays have been developed and these have improved the sensitivity of detecting the parasite in faecal specimens. The sensitivity and specificity of different assays is reported to range from 87 to 100% [121, 122]. Enzyme-linked immunosorbent assay (ELISA) is the mostly used immunoassay and it has enhanced the detection of the parasite in field samples and a number of kits are commercially available [120]. Furthermore, the development of direct immunofluorescence microscopy (antigen detection) has generally improved the sensitivity of detecting and quantifying faecal Giardia cysts and may allow for more accurate determination of prevalence rates and cyst excretion intensities compared to the conventional microscopy [46]. However, despite antigen detection being more sensitive than conventional microscopy, the method cannot discriminate between species or morphologically similar organisms. The other disadvantage is the need for a fluorescent microscope which is costly [123].

To overcome the non-discriminatory nature of the conventional microscopy, molecular techniques, particularly PCR-based procedures have been developed and have greater sensitivity and specificity than the techniques that rely on microscopy and/or immunodiagnosis [98]. For example, in a survey of dogs in India, microscopy detected only 3% prevalence compared to 20% with PCR [101]. The molecular methods are also able to provide information on the genotypes and species of Giardia, information that is necessary for determining the source of infection. PCR, when combined with restriction fragment length polymorphism (RFLP) analysis is faster when compared to sequencing which is also costly [124]. Although PCR has high sensitivity, results may be affected by amplification inhibitors and barriers to DNA extraction in faecal samples [125]. Moreover, PCR assays are very costly for diagnostic laboratory use [126] and are therefore commonly used in research.

Serodiagnosis cannot be used to differentiate between present and previous infection and is therefore not useful for the diagnosis of giardiasis.
6. Treatment

Treatment of giardiasis in livestock is through use of fenbendazole and albendazole, which have been shown to be effective in the elimination of *Giardia* from both housed and range calves [32, 127–129] as well as improving the mucosal microvillus structure and function within a week [129]. In sheep, treatment with fenbendazole at a dose of 10 mg/kg for three consecutive days, has been shown to successfully clear the infection. In an outbreak of giardiasis on a sheep farm, *Giardia*-infected lambs (30–90 days of age) presenting with malabsorption, decreased weight gain, and reduced feed efficiency recovered rapidly from the symptoms and poor weight gain after treatment with fenbendazole [69]. Similarly, in calves experimentally infected with *G. duodenalis* and treated with fenbendazole, a significant difference in weight gain was noticed between fenbendazole-treated and untreated calves. Animals in the treatment group gained on average 2.86 kg (equal to 102 g per day) more than the animals in the control group [27]. However, in some other treatment studies where fenbendazole or paromomycin sulphate were used, differences in mean body weight, average daily weight gain, or feed intake between the control and treated groups were not significant, although there was a slightly higher weight gain and lower occurrence of diarrhoea in the treated groups [12, 42].

In dogs and cats, fenbendazole is the commonly used therapy, normally given once daily for 3–5 days. Albendazole can be used but it has been associated with bone marrow suppression in both dogs and cats, and so no longer being used in both animal species [130]. Vaccines for *Giardia* in dogs and cats have been developed and they are reported to have the ability to reduce the duration of shedding of cysts which may subsequently reduce environmental contamination [131]. A prolonged treatment up to 5 days was shown not to be statistically better than treatment for three consecutive days [132]. On the other hand, metronidazole has been used to treat giardiasis in horses, with resolution of clinical signs after treatment [112].

7. Conclusions

*Giardia* infections are prevalent in livestock and companion animals. A number of studies have reported and genotyped *Giardia* in domestic animals, particularly livestock and companion animals, and have found that they may be infected with zoonotic or species-specific genotypes. However, there is still limited information on infection rates in pigs and horses. Further, the role of these animals and dogs in the zoonotic transmission of *Giardia* still needs further investigation. Studies reporting the existence of zoonotic assemblages in both animals and humans in the same locality (e.g. for dogs) emphasise the need for further studies on zoonotic transmission of *Giardia*. Such information will assist in further highlighting the public health significance of *Giardia*. Increased interaction and the nature of the interaction between companion animals and their owners can determine whether zoonotic infection occurs or not.

Economic implications of the disease in terms of treatment costs that the farmers have to incur cannot be overlooked especially in livestock (particularly dairy calves). Giardiasis adversely affects production; and chronic cases may impact negatively on the performance of affected animals resulting in reduced weight gain, impaired feed efficiency and delayed maturity. These loses translate into food loses.
Unfortunately, giardiasis in humans is not a health priority in most countries but the effect of the parasite in terms of patient well-being and its effect on quality of life have been highlighted by many authors, highlighting its impact on human health.

A better understanding of the disease in animals (livestock and companion animals), the species and transmission patterns is necessary for appropriate prevention and control strategies which should result in increased livestock production and reduced treatment costs for the farmers or animal owners. More molecular epidemiological studies are required especially in areas where these have not been conducted such as sub-Saharan Africa to understand and probably be able to relate human and animal infections. Treatment of *Giardia* infection in both livestock and companion animals is recommended whether or not they are clinically ill, because of the potential for zoonotic transmission.

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