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Giardia duodenalis Assemblages in Canine Hosts: Association with Mucus in Feces and Gastrointestinal Signs; Veterinary Parasitology

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ABSTRACT

Giardia duodenalis infection frequently occurs in dogs and is predominantly attributed to assemblages C and D. The objective of this work was to evaluate the association between G. duodenalis occurrence, its different assemblages detected in both symptomatic and asymptomatic dogs, and the manifestation of specific clinical signs. All examined dogs (n = 82) underwent a clinical evaluation, and fecal specimens were analyzed for other intestinal parasites and Clostridium spp. In addition, G. duodenalis assemblages were identified, and certain clinical manifestations were recorded. Out of the 82 dogs, 42 (51.2%) exhibited one or more gastrointestinal symptoms, whereas 40 (48.8%) showed no symptoms. G. duodenalis was detected in 25/82 (30.5%) dogs, with assemblage C identified in 10/25 (40%) and assemblage D in 15/25 (60%). Among coinfections, only Cryptosporidium spp. Presented a higher concurrent rate with G. duodenalis, although it did not influence the presence of clinical signs. No relationship was established between the type of G. duodenalis assemblage and the sex of the host or the nature and duration of gastrointestinal symptoms, except for mucus in feces, which appeared more frequently in dogs infected with assemblage C. Additional studies are needed to explore other assemblages further.

Keywords: G. duodenalis, Dogs, Assemblage, Gastrointestinal symptoms

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Introduction

Giardia species are diplomonad flagellates found in many vertebrates. Among them, Giardia duodenalis (G. duodenalis) is now recognized as a multispecies complex containing genetically distinct assemblages. Assemblages C, D, E, F, G, and H occur in diverse wild and domestic hosts, while assemblages A and B are most often detected in humans but can also be found in various animals. G. duodenalis follows a direct life cycle with two distinct stages: trophozoite and cyst. Transmission occurs via ingestion of infectious cysts through contaminated food, water, or direct fecal–oral exposure. Once inside the gastrointestinal tract, cysts excyst, releasing trophozoites. These trophozoites represent the pathogenic phase, attaching to enterocytes in the proximal small intestine where they multiply and absorb nutrients before transforming back into cysts [1–4].

In the past three decades, investigations into canine giardiasis have mainly focused on its prevalence, genotypic variability, and zoonotic implications. G. duodenalis is considered the most widespread intestinal parasite in dogs globally [5–9]. Reported prevalence ranges from 5% to 100% in privately owned dogs, influenced by factors such as age, diagnostic technique, health status, gut microbiota, geographical location, and veterinary consultation frequency [10–13]. Shelter dogs usually show higher infection rates due to dense populations, frequent arrivals of

susceptible animals (particularly puppies), and limited hygiene and management practices. Such conditions enhance the spread of intestinal parasites [13].

The contribution of G. duodenalis to the development of gastrointestinal disease—ranging from subclinical infection to acute or chronic diarrhea—remains debated. Clinical manifestations vary, and its role in other intestinal disorders is unclear. An Australian study demonstrated that G. duodenalis may be implicated in both acute and chronic diarrhea and recurrent gastrointestinal disturbances in dogs [14]. Moreover, infection prevalence among client-owned diarrheic dogs was higher than in stray dogs [15], though infection rates were similar between symptomatic and asymptomatic groups. The disease spectrum depends on host-related factors such as age, nutrition, and concurrent diseases. The main sign of giardiasis is diarrhea, which may be intermittent, self-limiting, or persistent, potentially resulting in dehydration and differing in intensity. Inflammation of the intestinal mucosa may cause severe enteritis, abdominal discomfort, nausea, maldigestion, and malabsorption. Some animals exhibit malodorous stools, steatorrhea, weight reduction, and poor growth. However, many immunocompetent dogs remain asymptomatic carriers and can serve as sources of infection for others [16]. Host characteristics—such as immune response, age, nutritional state, concurrent intestinal pathogens, and gut microbiome composition—can modify disease severity [17].

Research has shown that G. duodenalis infection may alter canine intestinal microbiota, leading to dysbiosis and related diseases [11, 18–20]. Alterations in the intestinal microbial community caused by pathogens are frequently associated with digestive disturbances and can influence systemic health. G. duodenalis infection can induce shifts in microbial composition, functional metabolism, and bacterial biofilm structure, all contributing to acute or chronic symptoms [19]. Besides intestinal signs, some dogs display atypical manifestations such as skin lesions and urticaria [21].

Certain studies have proposed a link between specific G. duodenalis assemblages and clinical severity. One report indicated that diarrheic dogs often carry dog-adapted assemblages C and D [22]. However, other research failed to confirm such associations with diarrhea or coinfections [23, 24]. Perrucci *et al*. (2020) [25] reported milder clinical manifestations in G. duodenalis-positive dogs with chronic enteropathy compared to uninfected ones. Concurrent infections with other microorganisms, parasites, or viruses may predispose dogs to gastrointestinal disturbances [26] and alter the microbiome, potentially aggravating disease [27]. Frequent canine intestinal parasites include Giardia duodenalis, Ancylostoma caninum, Isospora canis, Uncinaria stenocephala, and Trichuris vulpis, which often occur simultaneously [28].

Clostridium perfringens is a Gram-positive, anaerobic, spore-forming bacterium commonly residing in the gastrointestinal tract of both humans and animals. It is categorized into five biotypes based on major toxin genes. The mechanism of C. perfringens-related diarrhea in dogs remains unclear since the bacterium can also be found in clinically normal dogs [29].

The present research aimed to assess the effect of G. duodenalis infection on gastrointestinal clinical manifestations in dogs. Additionally, it sought to determine whether a connection exists between particular G. duodenalis assemblages or coinfections with other agents and the occurrence of clinical symptoms. These findings may contribute to improved understanding of the pathogenic mechanisms of G. duodenalis infection in dogs.

Materials and Methods

Animals

This retrospective investigation involved 82 dogs, comprising 58 privately owned and 24 from shelters. The dogs were brought to the veterinary clinic for various purposes, including digestive disturbances, standard checkups, or overall health assessments. Out of these, 42 animals displayed one or more gastrointestinal symptoms, while 40 showed no clinical signs. Both males and females of different breeds were included, all adults (over one year old) and appropriately vaccinated. Examinations took place at the Clinic for Internal Diseases, Faculty of Veterinary Medicine, University of Zagreb.

Only adult animals were chosen, as puppies are more susceptible to infections due to their still-developing immune defenses and gut microbiota composition. Enrollment required verified vaccination against canine distemper virus (CDV), canine adenovirus types 1 and 2 (CAV1 and CAV2), canine parainfluenza virus (CPiV), and both variants of canine parvovirus (CPV and CPV2c). Dogs were excluded if diagnosed with other gastrointestinal disorders such as inflammatory bowel disease, food intolerance, ingestion of foreign material, or any unrelated intestinal condition.

Clinical assessment

Each animal underwent a full clinical workup that included case history, physical evaluation, hematologic and biochemical profiling, and fecal sampling. Dogs presenting with at least one abnormal sign were defined as symptomatic. Using medical records, examination findings, and lab data, 14 parameters were selected to evaluate digestive health. These were adapted from the established Canine Chronic Enteropathy Activity Index [30] and expanded to include: disease duration, lethargy, appetite reduction, vomiting, stool consistency, increased bowel movement frequency (three or more times daily), vomiting blood, visible blood in stool, melena, mucus in feces, weight loss, low serum albumin (≤20 g/L), ascites or peripheral edema, and pruritus. Each parameter was scored as either present or absent.

Additional diagnostic tests were carried out to eliminate other potential diseases and to assess physiological changes possibly related to Giardia duodenalis infection. These tests included complete blood count, serum biochemical profile, urinalysis, and imaging studies when necessary. Cases lasting under three weeks were considered acute, whereas those exceeding three weeks or recurring were defined as chronic [31]. Fecal consistency was rated following the Purina Fecal Scoring Chart, where normal stool is firm yet soft, segmented, and leaves minimal residue on the surface (https://www.purinainstitute.com/centresquare/nutritional-and-clinical-assessment/purina-fecal-scoring-chart; accessed 1 August 2023).

Fecal examination

Fresh fecal material was obtained immediately after defecation, placed into sterile 50 mL containers, and transported to the Parasitology and Microbiology Laboratory at the Croatian Veterinary Institute within eight hours. All 82 samples were evaluated using the Merifluor® Cryptosporidium/Giardia immunofluorescent assay (Meridian Bioscience, Luckenwalde, Germany), following the supplier's guidelines. This method identifies Giardia cysts and Cryptosporidium oocysts by using fluorescein isothiocyanate (FITC)-labeled antibodies.

Three grams of feces per animal were subjected to centrifugal flotation with magnesium sulfate ($MgSO_4$; specific gravity 1.20) as outlined by Dryden *et al*. (2005) [32] for additional parasite detection. Samples positive for Giardia cysts were further processed for DNA extraction using the QIAamp® DNA Stool Mini Kit (Qiagen, Hilden, Germany) and then refined with the QIAquick® PCR Purification Kit from the same manufacturer.

Assemblage typing was achieved through nested PCR targeting a 175 bp region of the small subunit ribosomal RNA (SSU rRNA) gene [33]. The PCR reaction volume was 50 µL, using 2 µL of template DNA in the first round and 5 µL of the first-round product in the nested amplification. PCR products were separated via capillary electrophoresis using the QIAxcel System® (Qiagen, Hilden, Germany), employing size markers spanning 100–2500 bp. Purified amplicons (ExoSAP-IT®, USB Corp., Cleveland, OH, USA) were sequenced bidirectionally by Macrogen Inc. (Amsterdam, The Netherlands). Sequence assembly and editing were done in SeqMan Pro 12.2.0 and EditSeq (DNASTAR, Madison, WI, USA), and homology searches were conducted using BLAST.

To explore the presence of Clostridium perfringens as a potential co-pathogen, cultures were performed even when toxin production was uncertain. Bacterial identification followed isolation and incubation steps and relied on colony morphology, catalase and oxidase reactions, and biochemical profiling with the BBL Crystal™ Identification System (version 5.05A, Anaerobe ID kit). This system identifies anaerobic bacteria through fluorogenic and chromogenic substrate reactions. An invasion index was calculated by summing the total number of parasite and pathogen species detected per sample.

Statistical evaluation

Depending on the distribution of the data, results were expressed as median, minimum, or maximum values. Statistical comparisons between groups were performed using the t-test or, for non-normally distributed variables, the Mann–Whitney U test. The chi-square or Fisher's exact test was used for categorical variables. Statistical significance was established at $p < 0.05$. All computations were conducted using Stata software, version 13.1 (StataCorp, College Station, TX, USA).

Results and Discussion

A total of 82 dogs participated in this investigation, consisting of 36 crossbreeds and 46 purebreds from 23 distinct breeds. Ages ranged between 1 and 13 years (median age 4 years). The breeds most frequently represented included the German Shepherd (n = 4), Poodle (n = 4), Belgian Shepherd (n = 3), Border Collie (n = 3), and

Labrador Retriever (n = 3), while all other breeds appeared with one or two individuals. The sex ratio was balanced, with males and females each comprising 50% of the population.

Giardia duodenalis infection was confirmed in 25 out of 82 dogs (30.5%). Comparable infection frequencies were recorded in asymptomatic animals (12/40; 30.0%) and in those displaying gastrointestinal signs (13/42; 30.9%). Among privately owned dogs, 10 of 58 (17.2%) were positive, whereas shelter dogs showed a much higher prevalence of 15 of 24 (62.5%) ($p < 0.001$).

To assess the influence of mixed parasitic infections, additional intestinal organisms were investigated. In four symptomatic dogs (9.5%), diarrhea was attributed solely to Giardia, while nine (21.4%) showed Giardia alongside one or more parasites. Overall, 43.9% of all dogs carried intestinal parasites. Apart from G. duodenalis, the most frequent findings included Cryptosporidium spp. (10/82; 12.2%), followed by Trichuris vulpis (8/82; 9.7%), Toxocara canis (6/82; 7.3%), Isospora caninum (4/82; 4.9%), Strongyloides spp. (2/82; 2.4%), and Toxascaris leonina (1/82; 1.2%). The only significant coinfection in Giardia-positive dogs occurred with Cryptosporidium spp. ($p < 0.0001$), Though this combination was not linked with the presence of clinical signs ($p = 0.376$).

Median invasion scores varied significantly depending on Giardia status. Animals harboring G. duodenalis tended to be infected by multiple intestinal agents ($p < 0.0001$). The median invasion score among infected dogs was 3, compared to 1 in those without infection. Clostridium perfringens was isolated from 58 of 82 dogs (70.7%), but this bacterium showed no statistical relationship with clinical manifestations ($p = 0.189$).

Among dogs positive for G. duodenalis, 12 were female (48%) and 13 were male (52%) ($p = 0.699$). No statistical link was identified between infection and the overall occurrence of gastrointestinal signs **(Table 1)**. Symptomatic rates were comparable between infected and noninfected groups, although the type of disease course differed significantly ($p = 0.009$). Infected animals more often exhibited a chronic course of illness.

Table 1. Relationship between G. duodenalis infection and clinical parameters.

Indicator/Clinical Sign		Giardia		p
		Negative	Positive	
Clinical course	Acute	23	5	0.009
	Chronic	6	8	
Decrease in physical activity	No	18	9	0.654
	Yes	11	4	
Decrease in appetite	No	17	9	0.513
	Yes	12	4	
Vomiting	No	19	11	0.205
	Yes	10	2	
Change in feces consistency	No	2	1	0.926
	Yes	27	12	
Increased defecation frequency	No	8	2	0.391
	Yes	21	11	
Weight loss	No	18	9	0.654
	Yes	11	4	
Decrease in serum albumin level	No	28	12	0.550
	Yes	1	1	
Pruritus	No	28	13	0.498
	Yes	1	0	
Ascites/edema	No	29	12	0.131
	Yes	0	1	
Hematemesis	No	28	13	0.498
	Yes	1	0	
Fecal mucus	No	16	5	0.317
	Yes	13	8	
Hematochezia	No	19	10	0.205

	Yes	11	2	
Melena	No	28	13	0.498
	Yes	1	0	

Only dog-specific assemblages, C and D, were identified in this study. BLAST comparison with reference sequences from Sprong *et al*. (2009) [34] confirmed 100% identity with assemblage C (AF199449) and assemblage D (AF199443). The newly obtained sequences were submitted to GenBank under accession numbers SUB13938224 (GDIS1 OR769666) and SUB13938244 (GDIS2 OR769667). Assemblage D predominated, appearing in 15 of 25 (60%) isolates, whereas assemblage C accounted for 10 of 25 (40%). Among privately owned dogs, assemblage C occurred in 4 of 10 (40%) and assemblage D in 6 of 10 (60%), while in shelter dogs, the frequencies were 6 of 15 (40%) and 9 of 15 (60%), respectively (p = 1.0). Approximately half of the infected dogs exhibited symptoms—50% in assemblage C and 53.3% in assemblage D. No link was established between assemblage and sex (p = 0.141).

Duration of gastrointestinal signs did not vary by assemblage: the mean duration was 19.4 days for assemblage C and 22.9 days for assemblage D (p = 0.739). Likewise, the difference in invasion scores between assemblage C (median 2.5) and assemblage D (median 3) was not statistically meaningful (p = 0.50). Most gastrointestinal and systemic signs occurred at similar frequencies in both assemblage groups, except for mucus in feces, which appeared more often among assemblage C infections **(Table 2)**.

Table 2. Frequency of clinical manifestations in dogs with G. duodenalis assemblages C (n = 5) and D (n = 8).

Clinical Sign *		**Assemblage C Positive Dogs (n = 5)**	**Assemblage D Positive Dogs (n = 8)**	
Decrease in physical activity	%	40%	25%	p = 0.569
	N	2	2	
Decrease in appetite	%	20%	37.5%	p = 0.506
	N	1	3	
Vomiting	%	20%	12.5%	p = 0.715
	N	1	1	
Change in feces consistency	%	100%	87.5%	p = 0.411
	N	5	7	
Increased defecation frequency	%	80%	87.5%	p = 0.715
	N	4	7	
Weight loss	%	20%	37.5%	p = 0.506
	N	1	3	
Decrease in serum albumin level	%	0%	12.5%	p = 0.411
	N	0	1	
Ascites/edema	%	0%	12.5%	p = 0.411
	N	0	1	
Fecal mucus	%	100%	37.5%	p = 0.024
	N	5	3	
Hematochezia	%	40%	0%	p = 0.052
	N	2	0	
Chronic clinical course	%	40%	75%	p = 0.207
	N	2	6	

Pruritus, hematemesis, and melena were not recorded in Giardia-positive animals.

Intestinal parasites remain one of the most relevant etiological groups involved in gastrointestinal disorders in dogs. Reported infection rates for canine intestinal parasites range from 16% to 72% [35–39], and the 43.9% prevalence observed in this work aligns well with previously published data. Giardia duodenalis continues to be recognized as one of the most widespread protozoan enteric parasites affecting dogs globally [40]. Our results,

which revealed a 30.5% overall infection rate, corroborate that observation. From a public health standpoint, the zoonotic potential of G. duodenalis originating from canines has raised considerable concern [41]. Nevertheless, in Croatia, available findings indicate minimal zoonotic risk, since only dog-adapted assemblages C and D have been identified in earlier studies conducted both locally [42] and throughout nearby European regions [28, 43–45]. The current study reinforces those reports, having likewise identified only assemblages C and D, neither of which poses a hazard to human health. These host-specific genotypes tend to be highly adapted to canines, capable of faster replication, and can competitively replace assemblages A and B [46]. Contrarily, some investigations have found the possibly zoonotic assemblage A to dominate in dogs, with frequencies exceeding 80% [22, 47], while others also reported assemblages A and B, albeit less frequently [48, 49]. To clarify potential subassemblages or mixed infections, future studies employing additional molecular markers such as β-giardin (BG), glutamate dehydrogenase (GDH), or triose phosphate isomerase (TPI) genes would be valuable [50]. These markers were not applied here, as prior research has not shown a clear relationship between assemblage type and disease severity or even infection status.

The markedly higher occurrence of G. duodenalis detected in shelter animals compared with privately owned pets was anticipated, reflecting dense animal populations and elevated transmission potential in kennels and shelters [13].

The contribution of G. duodenalis to clinical symptom development remains uncertain. Infected dogs can exhibit either overt or subclinical infection [16]. Several works have reported that Giardia is directly associated with diarrheal episodes, with infected dogs being 1.5–2 times more numerous among diarrheic individuals than among healthy controls [15, 35, 40]. Conversely, other studies have demonstrated nearly identical prevalence rates in symptomatic and asymptomatic animals [23, 25], a pattern mirrored in our observations. Scorza *et al*. (2021) [24] similarly found no association between diarrhea and either particular Giardia assemblages or parasitic coinfections.

Diarrhea and vomiting represent the most typical manifestations of gastrointestinal disorders in dogs [51]. Their multifactorial etiology a demands comprehensive diagnostic evaluation to identify causative agents. Common parasitic contributors to such disorders include G. duodenalis, Ancylostoma caninum, Isospora canis, Uncinaria stenocephala, and Trichuris vulpis—either singly or in combination [14, 36, 37, 39]. Within our data, G. duodenalis infection occurred in 30.3% of symptomatic dogs; it was the exclusive etiologic agent in 9.2% and appeared with other pathogens in 21.4%.

Clinical expression of Giardia infection varies considerably [16]. The most frequently observed signs in this work were abnormal stool consistency, increased defecation frequency, presence of mucus in feces, reduced appetite, lower activity levels, and weight loss—consistent with previous descriptions of canine giardiasis [52]. Other authors [24, 25] have reported no correlation between G. duodenalis infection and severe gastrointestinal symptoms. Likewise, in our results, the overall frequency of digestive signs among Giardia-positive symptomatic dogs was similar to that of noninfected dogs, although chronic courses of disease predominated in infected animals. Earlier studies have recognized G. duodenalis as a major cause of persistent diarrhea in canines, though acute episodes can also arise [16]. Interestingly, in human patients, acute systemic involvement is more commonly documented [53–55], while such manifestations are rarely noted in dogs [21].

Despite a higher rate of concurrent parasite or pathogen infection among Giardia-positive dogs compared with uninfected ones, the degree of clinical alteration did not differ substantially. However, Tupler *et al*. (2012) [15] demonstrated that dogs with diarrhea are considerably more likely to harbor multiple enteropathogens than those passing normal feces. Considering that Cryptosporidium spp. and G. duodenalis share similar transmission routes, a notable frequency of dual infection was expected. Both are waterborne protozoa, suggesting shared environmental exposure such as puddles, moist grass, or park wetlands. These observations are consistent with prior work identifying the frequent coexistence of these two parasites in dogs [56, 57].

The higher invasion index found in Giardia-infected animals likely reflects their predominance in the shelter cohort, where exposure risks are greater. Whether host sex plays a role in G. duodenalis infection remains unresolved. Upjohn *et al*. (2010) [58] and Meireles *et al*. (2008) [59] recorded a greater frequency of infection in females, while Pallant *et al*. (2015) [60] reported assemblage D to be more frequent among males. In our investigation, no statistical association between sex and assemblage distribution was identified. Other factors—housing environment, daily activity patterns, and even breed-specific predispositions [61, 62]—appear to exert more influence than sex itself.

Limited attention has been given to how Giardia duodenalis assemblages relate to clinical manifestations in dogs. Scorza *et al*. (2021) [24] reported that Giardia genotypes showed no link with diarrhea, while Uiterwijk *et al*. (2020) [23] similarly found no correlation between assemblage type and soft stools. One of the goals of this work was to determine whether different G. duodenalis assemblages were associated with particular gastrointestinal signs in canines, as such studies are rare within veterinary parasitology. Across 14 clinical parameters examined, there were no statistically significant differences between assemblages, except for the presence of fecal mucus—which was noted in every dog infected with assemblage C and in 37.5% of those harboring assemblage D. The occurrence of mucus in feces usually reflects abnormalities in the large intestine; nonetheless, due to the complex and not yet fully elucidated pathogenesis of giardiasis, infections with these protozoans are believed to facilitate secondary digestive tract disorders, including inflammatory bowel conditions, microbiota imbalance, altered intestinal motility, and epithelial cell apoptosis. These processes collectively promote glandular hypersecretion and mucosal mucus output [12]. Overall, the results suggest that assemblage type does not play a major role in the onset of gastrointestinal symptoms.

In humans, the effect of assemblages A and B on diarrheal disease has been more thoroughly explored, though findings remain inconsistent. Some human-based studies found no relationship between assemblage type and symptom severity [63, 64], whereas others demonstrated differing clinical outcomes depending on assemblage [65–67]. These discrepancies highlight the challenge of linking specific assemblages to clinical presentations, even across similar host systems.

Assessing the role of G. duodenalis assemblages in the emergence of digestive disorders in dogs is methodologically complex, as numerous concurrent variables can influence gastrointestinal disturbances, ranging from other infectious agents and food intolerances to metabolic or endocrine dysfunctions. The coexistence of several causative factors and possible coinfections complicates efforts to clarify the true disease mechanism. Furthermore, interactions between the gut microbial community and intestinal parasites remain insufficiently characterized and represent an area warranting further study.

The primary limitations of this research include the modest sample size, which may have reduced statistical power to detect additional group differences, and the absence of longitudinal clinical follow-up that might have provided more insight into disease progression.

Conclusion

Only canine-adapted assemblages C and D of Giardia duodenalis were detected in the fecal samples analyzed. The presence of G. duodenalis infection or assemblage type did not influence gastrointestinal signs, apart from the observation of mucus in feces. Comparable infection rates in both symptomatic and asymptomatic dogs likely stem from multifactorial causes—such as variations in host immunity, intestinal microbiota composition, parasite adaptation to the canine host, and the exclusive detection of host-specific assemblages C and D. Fecal mucus was identified in all animals carrying assemblage C but only in 37.5% of those with assemblage D, emphasizing the need for deeper exploration of giardiasis pathogenesis and its interactions with the immune system, intestinal motility, and microbial balance, which may trigger glandular hypersecretion and mucus overproduction.

Future research should address how different G. duodenalis assemblages may influence not only the nature of clinical symptoms but also therapeutic response, treatment duration, and the potential emergence of drug resistance.

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References

1. Monis PT, Mayrhofer G, Andrews RH, Homan WL, Limper L, Ey PL. Molecular genetic analysis of Giardia intestinalis isolates at the glutamate dehydrogenase locus. Parasitology. 1996;112(1):1–12.

2. Adam RD. Biology of Giardia lamblia. Clin Microbiol Rev. 2001;14(3):447–75.
3. Cacciò SM, Thompson RCA, McLauchlin J, Smith HV. Unravelling Cryptosporidium and Giardia epidemiology. Trends Parasitol. 2005;21(9):430–7.
4. Gil H, Cano L, de Lucio A, Bailo B, de Mingo MH, Cardona GA, et al. Detection and molecular diversity of Giardia duodenalis and Cryptosporidium spp. in sheltered dogs and cats in northern Spain. Infect Genet Evol. 2017;50:62–9.
5. Drake J, Sweet S, Baxendale K, Hegarty E, Horr S, Friis H, et al. Detection of Giardia and helminths in Western Europe at local K9 (canine) sites (DOGWALKS study). Parasit Vectors. 2022;15(1):311.
6. Utaaker KS, Tysnes KR, Krosness MM, Robertson LJ. Not just a walk in the park: occurrence of intestinal parasites in dogs roaming recreational parks in Chandigarh, northern India. Vet Parasitol Reg Stud Rep. 2018;14:176–80.
7. Kostopoulou D, Claerebout E, Arvanitis D, Ligda P, Voutzourakis N, Casaert S, et al. Abundance, zoonotic potential and risk factors of intestinal parasitism amongst dog and cat populations: the scenario of Crete, Greece. Parasit Vectors. 2017;10(1):43.
8. Yu Z, Ruan Y, Zhou M, Chen S, Zhang Y, Wang L, et al. Prevalence of intestinal parasites in companion dogs with diarrhea in Beijing, China, and genetic characteristics of Giardia and Cryptosporidium species. Parasitol Res. 2018;117(1):35–43.
9. Ferreira JIGdS, Pena HFJ, Azevedo SS, Labruna MB, Gennari SM. Occurrences of gastrointestinal parasites in fecal samples from domestic dogs in São Paulo, SP, Brazil. Rev Bras Parasitol Vet. 2016;25(4):435–40.
10. Sweet S, Hegarty E, McCrann DJ, Coyne M, Kincaid D, Szlosek D. A 3-year retrospective analysis of canine intestinal parasites: fecal testing positivity by age, U.S. geographical region and reason for veterinary visit. Parasit Vectors. 2021;14(1):173.
11. Boucard A-S, Thomas M, Lebon W, Polack B, Florent I, Langella P, et al. Age and Giardia intestinalis infection impact canine gut microbiota. Microorganisms. 2021;9(9):1862.
12. Uiterwijk M, Nijsse R, Kooyman FNJ, Wagenaar JA, Mughini-Gras L, Koop G, et al. Comparing four diagnostic tests for Giardia duodenalis in dogs using latent class analysis. Parasit Vectors. 2018;11(1):439.
13. Raza A, Rand J, Qamar AG, Jabbar A, Kopp S. Gastrointestinal parasites in shelter dogs: occurrence, pathology, treatment and risk to shelter workers. Animals. 2018;8(7):108.
14. Kim MW, Sharp CR, Boyd CJ, Twomey LN. Faecal PCR panel results and clinical findings in Western Australian dogs with diarrhoea. Aust Vet J. 2020;98(12):563–9.
15. Tupler T, Levy JK, Sabshin SJ, Tucker SJ, Greiner EC, Leutenegger CM. Enteropathogens identified in dogs entering a Florida animal shelter with normal feces or diarrhea. J Am Vet Med Assoc. 2012;241(3):338–43.
16. Tangtrongsup S, Scorza V. Update on the diagnosis and management of Giardia spp infections in dogs and cats. Top Companion Anim Med. 2010;25(3):155–62.
17. Certad G, Viscogliosi E, Chabé M, Cacciò SM. Pathogenic mechanisms of Cryptosporidium and Giardia. Trends Parasitol. 2017;33(7):561–76.
18. Peruzzo A, Vascellari M, Massaro A, Mancin M, Stefani A, Orsini M, et al. Giardia duodenalis colonization slightly affects gut microbiota and hematological parameters in clinically healthy dogs. Animals. 2023;13(5):958.
19. Fekete E, Allain T, Siddiq A, Sosnowski O, Buret AG. Giardia spp. and the gut microbiota: dangerous liaisons. Front Microbiol. 2020;11:618106.
20. Šlapeta J, Dowd SE, Alanazi AD, Westman ME, Brown GK. Differences in the faecal microbiome of non-diarrhoeic clinically healthy dogs and cats associated with Giardia duodenalis infection: impact of hookworms and coccidia. Int J Parasitol. 2015;45(9–10):585–94.
21. Williams LBA. Generalized cutaneous urticaria associated with Giardia infection in a five-month old puppy. Vet Parasitol Reg Stud Rep. 2021;26:100643.
22. Claerebout E, Casaert S, Dalemans AC, De Wilde N, Levecke B, Vercruysse J, et al. Giardia and other intestinal parasites in different dog populations in northern Belgium. Vet Parasitol. 2009;161(1–2):41–6.
23. Uiterwijk M, Mughini-Gras L, Nijsse R, Wagenaar JA, Ploeger HW, Kooyman FNJ. Giardia duodenalis multi-locus genotypes in dogs with different levels of synanthropism and clinical signs. Parasit Vectors. 2020;13(1):605.
24. Scorza AV, Buch J, Franco P, McDonald C, Chandrashekar R, Lappin MR. Evaluation for associations amongst Giardia duodenalis assemblages and diarrhea in dogs. Vet Parasitol. 2021;300:109581.

25. Perrucci S, Berrilli F, Procopio C, Di Filippo MM, Pierini A, Marchetti V. Giardia duodenalis infection in dogs affected by primary chronic enteropathy. Open Vet J. 2020;10(1):74–9.
26. Kuzi S, Eshcol Argentaro S, Baneth G. Prevalence of Giardia duodenalis infection, co-morbidities and associated risk factors in dogs admitted to a veterinary teaching hospital in Israel. Comp Immunol Microbiol Infect Dis. 2020;68:101401.
27. Berry ASF, Johnson K, Martins R, Sullivan MC, Farias Amorim C, Putre A, et al. Natural infection with Giardia is associated with altered community structure of the human and canine gut microbiome. mSphere. 2020;5(6):e00670-20.
28. Sommer MF, Rupp P, Pietsch M, Kaspar A, Beelitz P. Giardia in a selected population of dogs and cats in Germany—diagnostics, coinfections and assemblages. Vet Parasitol. 2018;249:49–56.
29. Marks SL, Rankin SC, Byrne BA, Weese JS. Enteropathogenic bacteria in dogs and cats: diagnosis, epidemiology, treatment, and control. J Vet Intern Med. 2011;25(6):1195–208.
30. Allenspach K, Wieland B, Gröne A, Gaschen F. Chronic enteropathies in dogs: evaluation of risk factors for negative outcome. J Vet Intern Med. 2007;21(4):700–8.
31. Washabau RJ, Day MJ, Willard MD, Hall EJ, Jergens AE, Mansell J, et al. Endoscopic, biopsy, and histopathologic guidelines for the evaluation of gastrointestinal inflammation in companion animals. J Vet Intern Med. 2010;24(1):10–26.
32. Dryden MW, Payne PA, Ridley R, Smith V. Comparison of common fecal flotation techniques for the recovery of parasite eggs and oocysts. Vet Ther. 2005;6(1):15–28.
33. Read C, Walters J, Robertson ID, Thompson RCA. Correlation between genotype of Giardia duodenalis and diarrhoea. Int J Parasitol. 2002;32(2):229–31.
34. Sprong H, Cacciò SM, van der Giessen JWB; ZOOPNET network and partners. Identification of zoonotic genotypes of Giardia duodenalis. PLoS Negl Trop Dis. 2009;3(12):e558.
35. Itoh N, Kanai K, Kimura Y, Chikazawa S, Hori Y, Hoshi F. Prevalence of intestinal parasites in breeding kennel dogs in Japan. Parasitol Res. 2015;114(3):1221–4.
36. Guest CM, Stephen JM, Price CJ. Prevalence of Campylobacter and four endoparasites in dog populations associated with hearing dogs. J Small Anim Pract. 2007;48(12):632–7.
37. Oliveira-Sequeira TCG, Amarante AFT, Ferrari TB, Nunes LC. Prevalence of intestinal parasites in dogs from São Paulo State, Brazil. Vet Parasitol. 2002;103(1–2):19–27.
38. Ramírez-Barrios RA, Barboza-Mena G, Muñoz J, Angulo-Cubillán F, Hernández E, González F, et al. Prevalence of intestinal parasites in dogs under veterinary care in Maracaibo, Venezuela. Vet Parasitol. 2004;121(1–2):11–20.
39. Fontanarrosa MF, Vezzani D, Basabe J, Eiras DF. An epidemiological study of gastrointestinal parasites of dogs from southern Greater Buenos Aires (Argentina): age, gender, breed, mixed infections, and seasonal and spatial patterns. Vet Parasitol. 2006;136(3–4):283–95.
40. Bouzid M, Halai K, Jeffreys D, Hunter PR. The prevalence of Giardia infection in dogs and cats: a systematic review and meta-analysis of prevalence studies from stool samples. Vet Parasitol. 2015;207(3–4):181–202.
41. Ballweber LR, Xiao L, Bowman DD, Kahn G, Cama VA. Giardiasis in dogs and cats: update on epidemiology and public health significance. Trends Parasitol. 2010;26(4):180–9.
42. Beck R, Sprong H, Pozio E, Cacciò SM. Genotyping Giardia duodenalis isolates from dogs: lessons from a multilocus sequence typing study. Vector Borne Zoonotic Dis. 2012;12(3):206–13.
43. Pipia AP, Varcasia A, Tamponi C, Sanna G, Soda M, Paoletti B, et al. Canine giardiosis in Sardinia Island, Italy: prevalence, molecular characterization, and risk factors. J Infect Dev Ctries. 2014;8(5):655–60.
44. Piekara-Stępińska A, Piekarska J, Gorczykowski M, Bania J. Genotypes of Giardia duodenalis in household dogs and cats from Poland. Acta Parasitol. 2021;66(2):428–35.
45. Simonato G, Frangipane di Regalbono A, Cassini R, Traversa D, Tessarin C, Di Cesare A, et al. Molecular detection of Giardia duodenalis and Cryptosporidium spp. in canine faecal samples contaminating public areas in northern Italy. Parasitol Res. 2017;116(12):3411–8.
46. Thompson RCA, Monis PT. Variation in Giardia: implications for taxonomy and epidemiology. Adv Parasitol. 2004;58:69–137.
47. Leonhard S, Pfister K, Beelitz P, Wielinga C, Thompson RCA. The molecular characterisation of Giardia from dogs in southern Germany. Vet Parasitol. 2007;150(1–2):33–8.

48. Covacin C, Aucoin DP, Elliot A, Thompson RCA. Genotypic characterisation of Giardia from domestic dogs in the USA. Vet Parasitol. 2011;177(1–2):28–32.
49. Agresti A, Berrilli F, Maestrini M, Guadano Procesi I, Loretti E, Vonci N, et al. Prevalence, risk factors and genotypes of Giardia duodenalis in sheltered dogs in Tuscany (central Italy). Pathogens. 2021;11(1):12.
50. Beck R, Vojta L, Mrljak V, Marinculić A, Beck A, Zivicnjak T, et al. Diversity of Babesia and Theileria species in symptomatic and asymptomatic dogs in Croatia. Int J Parasitol. 2009;39(7):843–8.
51. Gaschen F. The principal syndromes in gastroenterology. In: Canine and Feline Gastroenterology. Alphen aan den Rijn: Wolters Kluwer; 2013. p. 1–44.
52. Buret AG. Pathophysiology of enteric infections with Giardia duodenalis. Parasite. 2008;15(3):261–5.
53. Suzuki Y, Nakamura T, Tokoro M, Togano T, Ohsaka M, Kohri M, et al. A case of giardiasis expressing severe systemic symptoms and marked hypereosinophilia. Parasitol Int. 2010;59(4):487–9.
54. Araki H, Shimizu S, Hayashi K, Yamada T, Kusakabe A, Kanie H, et al. Acute acalculous cholecystitis caused by Giardia lamblia. Intern Med. 2017;56(13):1657–62.
55. Avsar S, Oz A, Çınar T, Ösken A, Güvenç TS. Acute fulminant eosinophilic myocarditis due to Giardia lamblia infection presented with cardiogenic shock in a young patient. Anatol J Cardiol. 2019;21(4):234–5.
56. Wang X, Wang X, Cao J. Environmental factors associated with Cryptosporidium and Giardia. Pathogens. 2023;12(4):420.
57. Cao Y, Fang C, Deng J, Yu F, Ma D, Chuai L, et al. Molecular characterization of Cryptosporidium spp. and Giardia duodenalis in pet dogs in Xinjiang, China. Parasitol Res. 2022;121(5):1429–35.
58. Upjohn M, Cobb C, Monger J, Geurden T, Claerebout E, Fox M. Prevalence, molecular typing and risk factor analysis for Giardia duodenalis infections in dogs in a central London rescue shelter. Vet Parasitol. 2010;172(3–4):341–6.
59. Meireles P, Montiani-Ferreira F, Thomaz-Soccol V. Survey of giardiosis in household and shelter dogs from metropolitan areas of Curitiba, Paraná State, southern Brazil. Vet Parasitol. 2008;152(3–4):242–8.
60. Pallant L, Barutzki D, Schaper R, Thompson RCA. The epidemiology of infections with Giardia species and genotypes in well cared for dogs and cats in Germany. Parasit Vectors. 2015;8(1):2.
61. French SK, Kotwa JD, Singh B, Greer T, Pearl DL, Elsemore DA, et al. Factors associated with Giardia infection in dogs in southern Ontario, Canada. Vet Parasitol Reg Stud Rep. 2023;41:100870.
62. Mohamed AS, Glickman LT, Camp JW, Lund E, Moore GE. Prevalence and risk factors for Giardia spp. infection in a large national sample of pet dogs visiting veterinary hospitals in the United States (2003–2009). Vet Parasitol. 2013;195(1–2):35–41.
63. Kohli A, Bushen OY, Pinkerton RC, Houpt E, Newman RD, Sears CL, et al. Giardia duodenalis assemblage, clinical presentation and markers of intestinal inflammation in Brazilian children. Trans R Soc Trop Med Hyg. 2008;102(7):718–25.
64. Ajjampur SSR, Rajendran P, Ramani S, Banerjee I, Monica B, Sankaran P, et al. Closing the diarrhoea diagnostic gap in Indian children by the application of molecular techniques. J Med Microbiol. 2008;57(11):1364–8.
65. Gelanew T, Lalle M, Hailu A, Pozio E, Cacciò SM. Molecular characterization of human isolates of Giardia duodenalis from Ethiopia. Acta Trop. 2007;102(2):92–9.
66. Pelayo L, Nuñez FA, Rojas L, Furuseth Hansen E, Gjerde B, Wilke H, et al. Giardia infections in Cuban children: the genotypes circulating in a rural population. Ann Trop Med Parasitol. 2008;102(6):585–95.
67. Ignatius R, Gahutu JB, Klotz C, Steininger C, Shyirambere C, Lyng M, et al. High prevalence of Giardia duodenalis assemblage B infection and association with underweight in Rwandan children. PLoS Negl Trop Dis. 2012;6(6):e1677.