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REVIEW ARTICLE

Giardiasis: A review on assemblage distribution and epidemiology in India

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Abstract Giardiasis is a significant cause of diarrheal disease and associated morbidity in children and adults worldwide. In addition to diarrhea, it can also lead to malnutrition and cognitive deficits in children from developing countries. *Giardia duodenalis* is considered to be a species complex of several assemblages, of which assemblage A and B are predominantly associated with human infections. Assemblage type has been associated with risk of occurrence of symptoms and duration of illness. Hence genotyping of giardial isolates may help understand better the epidemiology and transmission ecology of the disease in a particular setting or area. In India, prevalence rates of *Giardia* infection in patients with diarrhea range from 0.4% to 70%, and asymptomatic cyst passage has been found to be as high as 50% in rural southern India. In this review, the global distribution of giardial assemblage, zoonotic transmission and the association of assemblage with disease have been discussed, followed by epidemiology of giardiasis in India.

Keywords Assemblage · Diarrhea · Genotyping · *Giardia* · India

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Introduction

Giardia is the most frequent intestinal protozoan infection worldwide with an estimated 280 million cases occurring annually [1]. In resource-poor countries, this parasite alone accounts for 2.5 million cases of diarrhea every year [2]. In these countries, *Giardia* infection is acquired during early infancy and its prevalence peaks at up to 30% in children younger than 10 years of age. Apart from diarrhea, giardial infection in children in these countries can result in faltering of long-term growth and impairment of cognitive function [3, 4]. Given its high prevalence in poor and underdeveloped communities, giardiasis was included in the WHO 'Neglected Diseases Initiative' in 2004.

Giardia duodenalis consists of a highly heterogeneous group of organisms and is referred to as a species complex divided at the genetic level into 'assemblages' (A to G) with different host specificities. Of these, human infections are mainly caused by assemblage A and B. In this review, we describe the global distribution of giardial assemblages and examine their association with symptomatology. The epidemiology of giardiasis in the Indian context has also been discussed.

Parasite, pathogenesis and immune response

Giardia, a protozoan, exists in two forms—trophozoite and cyst. The trophozoite is pear-shaped, measures 10–20 μm by 5–15 μm, and has two functionally identical and transcriptionally active nuclei and four pairs of flagella. On the ventral side of its body lies a concave

sucking disc, comprised ultrastructurally of repeating units of microtubules, that helps in attachment to the intestinal wall. The cyst is oval, measures 11–14 μm by 7–10 μm, has a thick outer shell, a central axostyle and four nuclei, and is the infective form. After ingestion of cysts, excystation occurs on exposure to low pH in the stomach releasing a transient stage that divides into four trophozoites. The trophozoites then localize in the upper small intestine, especially the duodenum, where they multiply by longitudinal binary fission. Encystation occurs when the trophozoite passes down the intestinal tract on exposure to bile salts. The infectious dose for symptomatic giardiasis was found to be as low as 10 to 25 cysts in a study carried out on prisoners in 1954 [5]. In a later study, using the gerbil model, an ID_{50} of 100 cysts was determined [6].

Several pathogenetic mechanisms have been implicated in giardial diarrhea [7, 8], including reduction in intestinal disaccharidase and protease activities, disruption of microvillous brush border, villus shortening or atrophy, crypt hyperplasia, increased epithelial permeability, thiol toxins, mucosal inflammation, bacterial overgrowth and, more recently, intestinal hypermotility. In a recent study on children with giardiasis, chronic mucosal inflammation with eosinophilic infiltrate was found [7].

The immune response of the host and its role in modulating giardial infection and pathogenesis are not completely understood yet. The frequent occurrence of asymptomatic infections points to either an effective host immune response or immune modulation by the parasite. Innate immune responses that have been shown to have an inhibitory effect on *Giardia* include antimicrobial peptides α-defensin and lactoferrin, inducible nitric oxide and complement activation by the lectin pathway. More recently, giardial extract has been found to modulate toll-like receptor signaling in dendritic cells, resulting in an anti-inflammatory response [9]. Anti-giardial antibody directed against the immunodominant variant-specific surface protein (VSP) is associated with acquired immunity to giardiasis and clearance of infection [8]. Secretory IgA antibodies to VSP in the breast milk of lactating mothers have been shown to protect against *Giardia* infection in infants [10]. In mice with deficient IgA production, although infection was not eradicated, the infectious load was reduced over time; this indicated that mechanisms other than IgA may help in host defense [11]. T-cell (αβ) responses to specific giardial soluble proteins have been found to play an important role in activation of B cell clones [12]. Recent animal studies have found that although hypermotility is described as a cause of giardial diarrhea, the association with presence of anti-giardial secretory IgA and clearance of infection may point to a role in host defense [13].

Disease burden and spectrum

A large majority (35% to 70%) of individuals infected with giardiasis remain asymptomatic. A minority develop acute diarrheal illness with fever, epigastric pain, nausea and vomiting, and rarely explosive diarrhea. Chronic giardiasis typically manifests as malaise, abdominal bloating and discomfort, intermittent mild diarrhea, and malabsorption. Giardiasis has also been associated with post-infectious irritable bowel syndrome, dyspepsia and functional gastrointestinal disorders [14]. Allergic skin manifestations including urticaria and angioedema have also been described. The wide spectrum of symptomatology of giardial infection has been attributed to factors such as inoculum size, specific host response and parasitic factors, of which genotypic difference may be an important determinant. Persistent infection and chronic disease in some patients may be explained by immune evasion due to antigenic variation in VSP, regulated by a mechanism similar to RNA interference [15].

Malnutrition in children

The clinical outcome of giardial infection appears to be more significant in infants and young children. In Israeli Bedouin infants, *Giardia* carriers were stunted compared to infants never positive for *Giardia*; this effect of *Giardia* infection on growth occurred even if the infection was not associated with diarrhea [16]. In a recent study from Vellore in southern India, children in an urban slum with a history of giardial diarrhea before the age of 2 years had lower intelligence quotients and wasting, compared to children with no past history of giardial diarrhea [17]. Stunting due to giardiasis has also been observed in school-going children in developing countries [4, 18]. Also, treatment of giardiasis resulted in increased weight and height gain, allowing catch-up growth in pre-school children [19]. Psychomotor development and cognitive development is also adversely affected by early childhood giardial infections [3, 20]. In children, giardiasis is also associated with lower mean hemoglobin levels [21, 22] and vitamin A deficiency [23].

Therapy

Drugs for treatment of giardiasis include metronidazole and other nitroimidazole, and benzimidazole derivates such as albendazole, tinidazole, paromomycin and nitazoxanide. The choice of drug may be influenced by factors such as cost, dosage and duration of treatment, compliance and adverse effects. Recently, drug-resistant strains of metronidazole have been reported [24]. In developing countries where giardiasis is endemic and treatment is often followed by re-infection [25,

26], the role of treatment of asymptomatic infection with these anti-parasitic drugs has been questioned. In these areas, alternative strategies for treatment of asymptomatic infection especially in very young children may need consideration, in view of long-term effects on growth and cognitive function. Probiotics including Lactobacilli spp. [27] and Sacchromyces boulardii [28] and wheat germ lectins [29] with metronidazole have been tried.

Species and genome

Based on differences in morphology and host specificity, several species of *Giardia* have been described, including *G. duodenalis*, *G. muris*, *G. agilis*, *G. ardeae*, *G. psittaci*, and *G. microti*. The species names *G. duodenalis*, *G. intestinalis* and *G. lamblia* have been used interchangeably to describe the *Giardia* species that infect humans. *G. duodenalis* has 5 chromosomes and a genome size of approximately 11.7 Mb, with a GC content of 49% [30]. The genome is more compact than those of other protozoa and codes for a limited repertoire of metabolic functions, indicating that it is a genome adapted to a parasitic life cycle [30]. Reproduction of *Giardia* was previously presumed to be asexual. However, a recent study identified homologs of genes involved in meiosis in *Giardia*, indicating a potential for fusion of nuclei in the cysts followed by somatic recombination [31]. This raises the possibility that this protozoan, previously thought to be clonal, may be genetically more diverse.

Giardial assemblages

Nash, Homan and Mayrhofer in 3 independent studies, described two major groups of *G. duodenalis* that infect humans [32–34]. Subsequent studies comparing the small-subunit rRNA (SSU rRNA), triosephosphate isomerase (TPI) and glutamate dehydrogenase (GDH) genes confirmed the existence of two major genotypes among the human isolates, referred to as assemblage A and B. The whole genomes of these assemblages have been mapped using laboratory adapted WB (assemblage A) and GS (assemblage B) strains. These assemblages have a 77%±5% nucleotide homology and 78%±14% amino acid homology in the protein coding regions [35]; in addition, sequence diversity is more marked in the GS isolate (average 0.5%) than in the WB isolate (<0.01%). The VSP (variant-specific surface protein) repertoires of these two strains were also found to be quite different [35]. Whether these differences between genomes of the assemblages A and B extend to clinical or community isolates is unclear. Further work using molecular tools has revealed additional host-specific assemblages C to G (C and D in dogs, E in cattle and pigs, F in cats and G in rodents) [8].

Assemblage A and B isolates that infect humans have been further divided into sub-assemblages based either on restriction digestion patterns of PCR products at the GDH and the TPI loci (denoted as AI and AII, and BIII and BIV) or on sequence analysis of the β-giardin gene (denoted as AI, AII and AIII and BI, BII, BIII and BIV) [36–39]. Sequence heterogeneity between the TPI, β-giardin and GDH loci in the same isolate has been reported for both human and animal isolates of *Giardia*

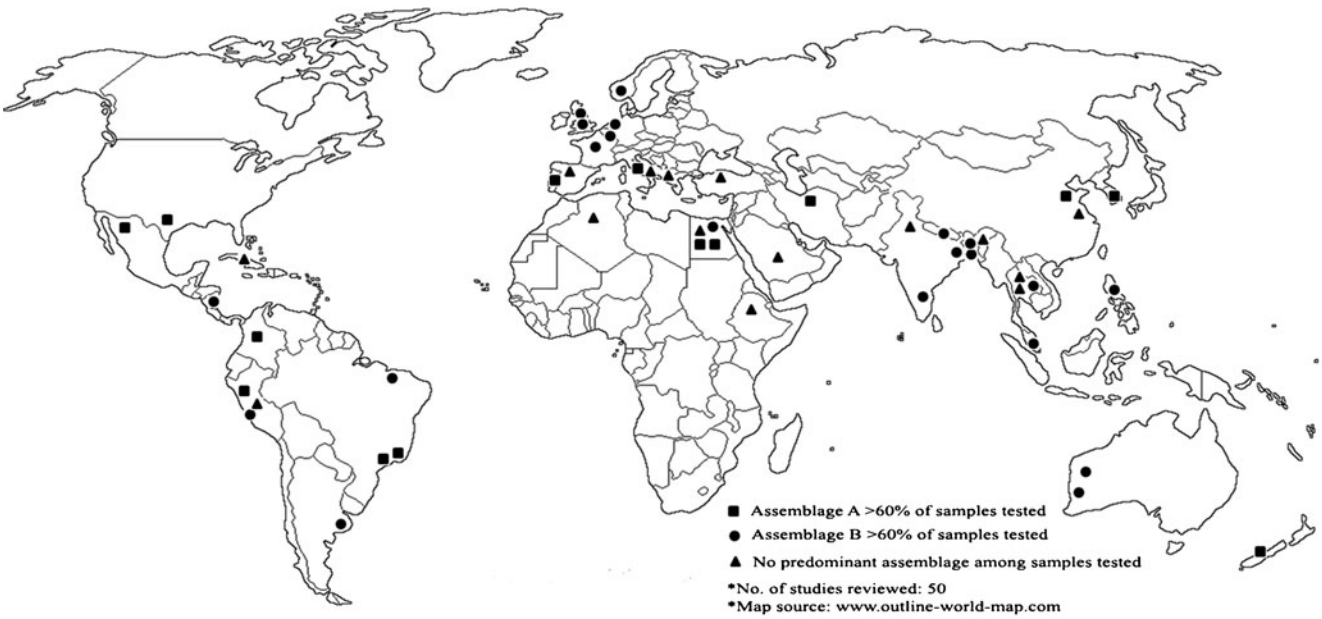

Fig. 1 Geographic distribution of giardial assemblages A and B

Table 1 Global prevalence rates of giardial assemblages

Country/ Region	Year	*n*	Assemblage %			Reference
			A	B	Mixed	
Asia						
South Korea	2000	07	100	0	0	[53]
China (Anhui province)	2000	08	50	50	0	[53]
China (Henan province)	2010	18	66.6	33.4	0	[54]
Bangladesh	2005	283	07	82	06	[45]
Philippines	2007	133	14	86	0	[46]
Nepal	2009	35	20	74	6	[49]
Malaysia	2009	42	02	98	0	[48]
Thailand	2010	61	08	51	41	[52]
Thailand	2008	12	41.7	58.3	0	[51]
India	2009	101	6.9	87	5.9	[47]
Middle East and Africa						
Iran	2008	38	87	7.8	5.2	[55]
Saudi Arabia	2010	40	57.5	37.5	5	[56]
Egypt	2004	105	49	10	15	[57]
	2008	89	65	14	21	[59]
	2009	41	75	20	05	[42]
	2008	18	05	80	15	[60]
Ethiopia	2007	59	53	22	25	[58]
Algeria	2009	41	36	56	06	[43]
Europe						
UK	2002	33	27	64	09	[38]
	2010	267	24	73	03	[44]
Belgium	2009	72	0	70	30	[61]
Turkey	2004	44	43	57	0	[62]
France	2005	25	36	64	0	[63]
Norway	2007	63	05	95	0	[64]
Albania	2006	22	45.5	54.5	0	[66]
Netherlands	2006	98	35	65	0	[65]
Italy	2002	30	80	20	0	[39]
	2005	37	43	27	30	[67]
Portugal	2006	25	100	0	0	[68]
Spain	2008	108	40	57	04	[84]
South America						
Mexico	2008	12	100	0	0	[69]
Colombia	2007	24	100	0	0	[70]
Nicaragua	2008	119	21	79	0	[71]
Argentina	2008	43	07	93	0	[72]
Peru (Trujillo)	2008	16	62.5	37.5	0	[75]
Peru (Lima)	2010	166	39.7	48.7	12	[74]
	2003	25	24	76	0	[73]
Brazil (Sao Paulo)	2007	37	78	22	0	[76]
Brazil (Rio de Janeiro)	2007	62	100	0	0	[77]
Brazil (Ceara)	2008	58	16	74	10	[41]
Cuba	2008	20	45	55	0	[78]
North America						
Texas	2009	8	75	25	0	[79]
Australia and New Zealand						
Australia	2010	124	25	75	0	[81]
	2002	36	30	70	0	[80]
New Zealand	2008	30	77	23	0	[82]

[40]. This could be due either to recombination occurring during meiotic replication or more often to 'mixed' infection with cysts belonging to different assemblages or sub-assemblages in the same host [41–44]. A multi-locus genotyping (MLG) approach has been recommended to assign assemblage and sub-assemblage types and a nomenclature system for assemblage A has been proposed [40].

Geographic distribution of assemblages

Distribution of two assemblages among human-associated *Giardia* isolates varies in different parts of the world (Fig. 1, Table 1). Assemblage B predominates in most studies from south and south east Asia including India [45–52]. The predominance of assemblage B however, did not extend to all the far eastern countries. Reports from South Korea and Henan province in China based on very few samples, showed predominance of assemblage A [53, 54].

Assemblage A was found to the most common in 2 studies from the Middle East, including Iran and Saudi Arabia [55, 56]. Reports from Africa have shown a mixed picture, with predominance of assemblage A of up to 75% in studies from Egypt and one from Ethiopia [42, 57–59] and of assemblage B in one study each from Egypt and Algeria [43, 60]. In Ethiopia an unusually high number of patients with mixed infections of assemblage A and the zoonotic assemblage F (12%) were reported [58].

In Europe, similar to Asia, most reports show a predominance of assemblage B [38, 44, 61–66]. Two studies from Italy however, showed a predominance of assemblage A; one of which also reported 30% of mixed assemblage A and B infections [39, 67]. In a single study from Portugal, only assemblage A was detected [68].

In the Americans, there seem to be pockets of areas with differing predominant assemblages. Studies done in Mexico and Columbia identified assemblage A alone while studies from Nicaragua and Argentina showed assemblage B as predominant [69–72]. Studies from Lima, Peru reported a predominance of assemblage B (both single and mixed infections) while another study from Trujillo district reported a predominance of assemblage A [73–75]. In Brazil, samples from community based studies in the southern cities of Sao Paulo and Rio de Janeiro showed a higher prevalence of assemblage A and those from a shanty town in the northeastern state of Ceará showed more of assemblage B [41, 76, 77]. Cuba had a near equal prevalence of both assemblages [78] while in the United States, a study done in Texas showed predominance of assemblage A [79], similar to that of neighboring Mexico [69]. Australia showed a predominance of assemblage B while neighboring New Zealand showed predominance of assemblage A [80–82].

Association between assemblage and symptoms

Differences in symptomatology of giardiasis with different assemblages were initially described in Dutch patients, among whom assemblage A was associated with mild intermittent disease and assemblage B with severe persistent disease [83]. Since then, several studies have found an association of diarrhea with assemblage A infection [42, 44, 45, 47, 62, 75, 80, 84]; in contrast, some studies have found an association of symptoms with assemblage B [48, 52, 56, 58, 78] (Table 2). Interestingly, most studies where assemblage A was associated with disease or more severe symptoms were from regions where assemblage B predominated. Some studies have however found no relation between symptomatology and the infecting assemblage [41, 59, 67, 71]. Further studies with longitudinal cohorts as well as animal models are required to test this association of assemblage with symptoms.

Zoonotic transmission

The potential for zoonotic transmission of *Giardia* was recognized in the late 1970s, though it has yet not been conclusively proved. Possible sources of such zoonotic transmission are from dairy cattle, companion animals like cats and dogs or wild life. Giardiasis is widespread among livestock and infected calves can shed as many as 10^5-10^6 cysts per gram of feces [85]. In a study in a remote tea-growing community in India, Traub et al. showed a significant association between human giardiasis and the presence of a *Giardia*-positive dog in the household [86]. In a study from Ecuador, children who lived in households with domestic animals were 2–5 times more likely to be infected with *Giardia* [87]. More recently, molecular epidemiological studies in Europe and Africa have also found potential for cross-species transmission of *Giardia* to humans from animals harboring human-associated assemblages including cattle [85], pet cats and dogs [76, 88] and monkeys [89].

Studies have also identified zoonotic assemblages in humans including the cat-associated assemblage F in mixed infections with assemblage A (8.7%) in Ethiopia [58] and cattle-associated assemblage E in 15% of samples in Egypt [60]. At the sub assemblage level, although assemblage AII is human associated, assemblage AI and more recently assemblage AIII have been found to be more animal associated. Transmission of animal associated assemblage AI has been seen among dairy farm workers in West Bengal [90]. A European network of 9 public and veterinary health institutes called ZOOnotic Protozoa NETwork (ZOOPNET) (http://www.medvetnet.org/cms/) found that though a majority of the human isolates (n=1658) belonged to assemblage B (56%) and assemblage A (43%), around 1% of isolates included zoonotic assemblages C, D, E and F [91]. The European study also found assemblage B was more strictly restricted to humans; in comparison, assemblage A is less strictly restricted to human hosts, being present at

Table 2 Association of giardial assemblages with severity of diarrhea and other symptoms

Country	Age group	Locus used for genotyping	Inference	Reference
Australia	<5 y	SSU rRNA	Assemblage A—26 times more likely in diarrhea	[80]
Bangladesh	All	TPI	Assemblage A—higher odds ratio of diarrhea (2.11)	[45]
Cuba	Children	β-giardin, GDH	Assemblage B—associated with symptoms	[78]
Egypt	All	TPI	Assemblage A—intermittent, more severe symptoms	[42]
England	All	TPI, SSU rRNA	Assemblage A—more frequent fever	[44]
Ethiopia	All	β-giardin, TPI, GDH, SSU rRNA	Assemblage B—stronger correlation with diarrhea	[58]
India	<3 y	TPI	Assemblage A—associated with diarrhea	[47]
Malaysia	All	SSU rRNA	Assemblage B—higher risk of symptoms	[48]
Netherlands	All	GDH	Assemblage B—more severe disease	[83]
Peru	<9 y	GDH	Assemblage A—associated with diarrhea	[75]
Spain	<5 y	TPI	Assemblage AII—associated with symptoms	[84]
Thailand	All	SSU rRNA, β-giardin, GDH, TPI	Assemblage BIII—associated with symptoms	[52]
Turkey	All	TPI	Assemblage A—associated with symptoms	[62]
Saudi Arabia	Children	IGS rDNA	Assemblage B—always symptomatic	[56]

TPI triosephosphate isomerase

GDH glutamate dehydrogenase

comparable rates in humans, companion animals, livestock and wildlife [91].

Giardiasis in India

In several epidemiological studies from India, giardiasis has been found to be prevalent throughout the country (Table 3). In community-based studies from northern India, prevalence rates ranged from 5.5% to 70% [92–95] with the highest rates reported in a low socioeconomic group in Chandigarh [93]. High rates of asymptomatic cyst passage [96, 97] and diarrhea associated with giardiasis were identified in children [98–102]. In studies from southern India, the prevalence rates of giardiasis in children with diarrhea and adults with gastrointestinal symptoms (diarrhea, abdominal discomfort, flatulence, weight loss or anorexia) were 8% to 10% and 37.1%, respectively [103, 104]. In Calcutta, *Giardia* was previously found to be associated with 0.4% to 2.6% of diarrheal episodes in children <5 years; however, more recent studies that used ELISA and PCR-based methods, showed incidence rates of giardial diarrhea of up to 15.8% in this age group [105–108]. The variations in the prevalence of giardiasis described above and in Table 3 may reflect a true variation in infection rates in different areas, or be due to the use of different methodologies used and the number of sequential stool samples tested per patient.

HIV infection may impact on giardiasis. Though *Giardia* is not considered a classical opportunistic infection, its prevalence rate has been found to be higher among AIDS patients and is associated with diarrhea. In India, the prevalence of giardiasis in HIV-infected patients was 1.6% to >30%

Table 3 Prevalence rates of giardiasis observed in various studies in India

Site of study	*n*	Year	Population	% Prevalence	Reference
Northern India					
Amritsar	150	1995	Children <3 y with chronic diarrhea	4	[99]
Chandigarh	550	2004	Low socioeconomic status	6	[94]
Chandigarh	600	2005	General population	5.5	[95]
Chandigarh	970	1991	Low socioeconomic status	69.5	[93]
Chandigarh	82,667	2000	Outpatients	4–9	[115]
Chandigarh	120	1994	Infants with intractable diarrhea	6	[98]
Delhi	175	2008	Children with persistent diarrhea	20	[116]
Delhi	127	2002	Children with diarrhea	11	[101]
Delhi	100	2008	Adult and children with malabsorption	24	[117]
Delhi	939	2002	Urban slum dwellers	8.4	[118]
Lucknow	1,071	2007	Urban and rural population	22	[96]
Lucknow	1,061	1997	Pre-school slum children	32.9	[97]
Punjab	–	1986	Pre-school children	35.1	[92]
Srinagar	514	2007	School children	7.2	[119]
Southern India					
Bangalore	361	1990	Children with diarrhea	8–10 (<6 m–2.1)	[103]
Pune	76	1991	Children <5 y with diarrhea	7.9	[102]
Chennai	324	2002	Rural (n=125) and urban (n=199) population	16 (rural), 22.6 (urban)	[125]
Karnataka	10,000	1998	Adults	37.1	[104]
Karnataka	1,020	1989	Adult and children, healthy	2.5	[120]
Vellore	78	1998	Asymptomatic rural population	53.8	[121]
Vellore	452	2009	Children in urban slum	22.9	[47]
Eastern India					
Varanasi	2,095	1999	Patients with acute diarrhea	1.7	[122]
Bihar	326	1996	Rural and semi-urban	28.2	[123]
Kolkata	383	1984	Under five children with diarrhea	0.4	[105]
Kolkata	1,103	2009	Children and adults with diarrhea	13.3	[107]
Kolkata	2,519	2010	Hospitalized patients with diarrhea	11.2	[108]
Sikkim	2,559	1970	General population	5.9	[124]

[109–112], with some studies showing a higher prevalence in patients with diarrhea and lower CD4 counts [110, 112].

Genotyping has been done for a few giardial isolates from different parts of India [47, 73, 86, 90, 113]. The first study by Paintlia et al. (1998) was done in a small number of young adults from Delhi with (n=6) and without diarrhea (n=6) and 4/6 symptomatic adults were infected with assemblage A while the other 2 had assemblage B. In Vellore, a study in children with and without diarrhea (n=99) showed that assemblage B was predominant both in giardial diarrhea (80%) and asymptomatic giardiasis (94%). However, children with assemblage AII alone or dual infections with both assemblage AII and B had diarrhea more frequently [47]. In a more recent follow up study, assemblage B was predominant in both children and adults (82.4%) followed by assemblage AII (9.4%). Mixed infections with assemblage A and B were seen exclusively in children, and mixed assemblage BIII and BIV were more common in children than adults (p=0.058) [114]. Assemblage AI was not detected. Characterization of nine clinical isolates from West Bengal as part of a larger phylogenetic study of giardial isolates, found all isolates to be assemblage B [73]. In a study in a tea tribe in Assam with some evidence of zoonotic transmission from dogs, a high prevalence of dogs were seen harboring human associated assemblages B and AII while the human samples showed assemblage B and assemblage A as well as mixed infections. This study also identified a few assemblage AI in both human and dog samples [86].

To summarize, the prevalence rates of various circulating assemblages of *Giardia* vary geographically even within the same country. Increasing evidence is appearing to show that diarrheal symptoms may be associated with specific assemblages and this phenomenon may in future explain the wide variation in symptoms among persons infected with *Giardia*. Assemblage B has been found to be more human restricted than assemblage A, and is the most common assemblage in most parts of the world, including India. Several unanswered questions remain about this ubiquitous protozoan. Community based studies on re-infections with the same or with other assemblages or other evidence of assemblage specific immunity are lacking. The association of giardiasis with malnutrition in children and the benefits of treatment have also not been studied in detail. Larger longitudinal studies in endemic settings are required to understand the role that assemblage type plays in giardial infections in vulnerable populations and whether subsequent development of immunity is assemblage specific.

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Author contributions

SL and SSRA collected and analyzed the data and drafted the manuscript. GK critically revised the manuscript. All authors approved the final manuscript.

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