Chapter 2

# Giardiasis Epidemiology

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Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.70338

**Abstract**

Giardiasis is the intestinal infection caused by *Giardia lamblia*, in which pathogenicity was cast doubted for decades but now is recognized as one of the most common causes of diarrheal disease worldwide. Originally described as waterborne transmitted, it has been broad described as of fecal-oral, person-to-person contact, and sexual transmission also. Although it is recognized as endemic throughout the world, most cases are reported from tropical countries with regular outbreaks commonly reported from developed countries. In humans, giardiasis normally produces a self-limited infection without symptoms, but some patients may present intestinal symptoms such as diarrhea and abdominal pain, and few show symptoms long after parasites clear up. Upon diagnosis, we may choose among several effective treatment alternatives, but not every patient responds to such therapies. Hence, having specific knowledge about the epidemiology of *Giardia*, it is critical for its prevention, which is the best strategy to protect us against such important disease.

**Keywords:** giardiasis, *Giardia*, *Giardia lamblia*, epidemiology, life cycle stages

## 1. Introduction

Giardiasis is the intestinal infection caused by *Giardia lamblia* (also known as *Giardia intestinalis* or *Giardia duodenalis*). *G. lamblia* is a unicellular eukaryotic protozoan that was first described by Antonie van Leeuwenhoek in 1681—in his own stool sample [1]. For decades, it was considered of uncertain pathogenicity but now is recognized as common causes of diarrheal disease worldwide. Its clinical significance was broadly accepted after many symptomatic cases of giardiasis were diagnosed and reported among visitors to the Soviet Union in the early 1970s. Since then, giardiasis has been reported as responsible for many outbreaks throughout the world.

The epidemiology of giardiasis still is a matter of great discussion. From the original debates around its pathogenicity to the later ones about its speciation and biology, *G. lamblia* has proven to be an enigmatic and interesting organism [2]. Although giardiasis is currently recognized as one of the main causes of diarrheal disease and a leading cause of death and illness among children under 5 years old in developing countries [3], the long-term impact of pediatric giardiasis remains unclear. Recent cohort studies have confirmed a high prevalence of persistent, subclinical giardiasis and its association with growth shortfalls [4], but such evidence has not been consistently reported in the literature.

Commonly, giardiasis prevalence among poor populations is reported as very high, and when the infection became chronic, it has been associated also with malnutrition and cognitive deficits [5]. In developed countries, giardiasis represents the leading cause of traveler's diarrhea and is frequently reported among citizens that traveled to developing countries and expose themselves to untreated water from lakes, streams, and swimming pools [6–8]. These and other epidemiologic characteristics of giardiasis will be discussed in detail in this chapter based on the classical and latest literature.

## 2. Etiologic agent

*G. lamblia* is a parasitic protozoan of the order *Retortomonadida* that alternates between trophozoites and cysts forms within its life cycle, stages responsible for the clinical illness, and the transmission of the disease, respectively. Under the light microscope, trophozoites appear actively swimming and with its characteristically teardrop (viewed dorsoventrally) or spoon (viewed from the side) shaped, measuring 10–20 μm by 5–15 μm by 2–4 μm, containing four pairs of flagella, two identical nuclei, with a convex dorsum and a ventral disc that acts as a suction cup to facilitate attachment of the organism to the small bowel villi (**Figure 1A**). On high-quality slides, the parasite movement shows "falling leave mobility" and resembles a human face because of the positions of the median bodies, nuclei, and axonemes. Uncharacteristically *Giardia* trophozoites lack definable Golgi, peroxisomes, and true mitochondria, but have a menant mitosome. Trophozoites divide by binary fission, and cyst develops as feces dehydrated in transiting to the large bowel.

Microscopically, *Giardia* cysts look oval shaped, measures about 11–14 by 7–10 μm, contains four nuclei (mature cyst)—usually situated at one end—and curved median bodies and linear axonemes (**Figure 1B** and **C**). During the process of encystment, which can be observed under the microscope, trophozoites initially become inactive, rounded, and increasingly refractile as encystment begins. Then, nuclear division (but not cytoplasmic) occurs to produce the quadrinucleate infectious cyst. *Giardia* cysts have a thick hyaline wall that protects them from environmental stressors such as the alkaline environment that characterize the small intestinal, water chlorination, high-altitude, or extreme temperatures such as in boiling water. Also, such strong protection allows cysts to survive in water up to 3 months [9]. Upon excystation in the small bowel, each cyst releases two trophozoites, which continue the life cycle.

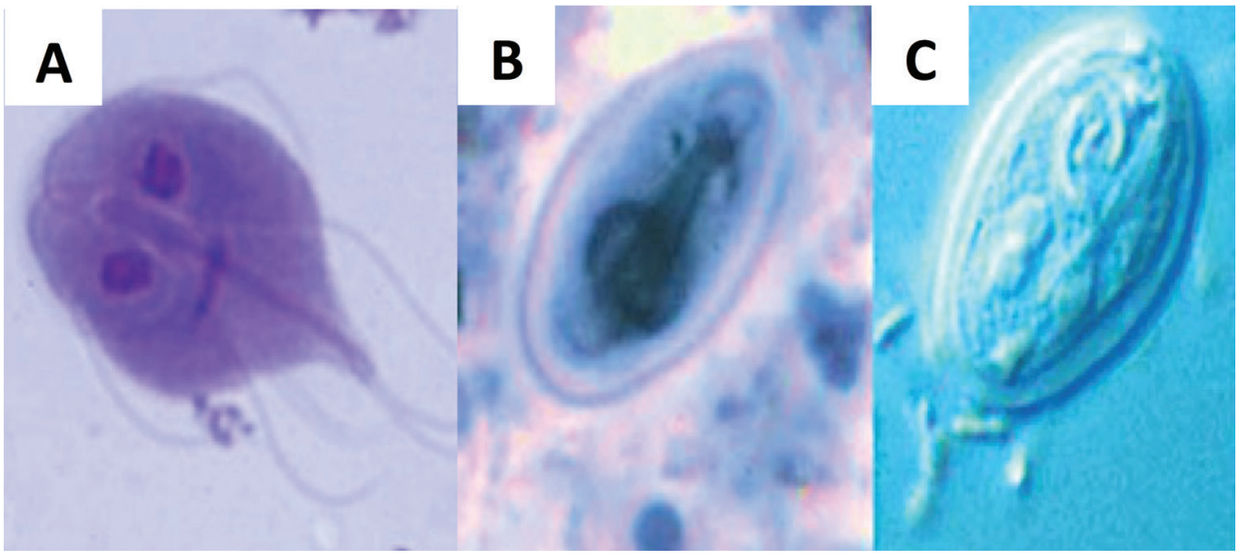


**Figure 1.** *Giardia lamblia*. Stained trophozoite (A) with its characteristic teardrop shape, binucleate structure, and four pairs of flagella clearly visible. Stained (B) and unstained (C) cysts with its characteristic median (parabasal) bodies and four nuclei. Images are some of the best public-domain light micrographs of *Giardia*, published by the National Institute of Infectious Diseases of Tokyo, Japan. Images were originally published at http://www.nih.go.jp/niid/ja/kansennohanashi/410-giardia.html.

*Giardia* species are currently classified in six species, which are distinguished based on its morphology and hosts: *Gracilinanus agilis* (amphibians), *G. lamblia* (mammals), *Giardia muris* (rodents), *Giardia psittaci*, and *Giardia ardeae* (both mainly in birds) (**Table 1**) [2]. Previously, many more species of *Giardia* were listed based only on microscopic and epidemiological criteria as well, but using molecular tests such as polymerase chain reaction (PCR), the list was shortened. Later on, genotyping studies confirmed that the species of *Giardia* could be classified in eight genetic groups (A–H): Groups A and B, which are found in humans and

| Species | Hosts | Morphology |
|---|---|---|
| *G. agilis* | Amphibians | Longer and slender than *G. lamblia*, with a teardrop-shaped median body |
| *G. ardeae* | Herons and other birds | Similar to *G. lamblia* |
| *G. lamblia* | Mammals including humans, dogs, and some wild species | Teardrop shaped with claw-shaped median bodies |
| *G. microti* | Rodents, voles, and muskrats | Similar to *G. lamblia* |
| *G. muris* | Rodents | Shorter and rounder than *G. lamblia*, with small rounded median body |
| *G. psittaci* | Psittacine birds | Similar to *G. lamblia* |

Adapted from Adam [2] and Caccio and Ryan [14].

**Table 1.** *Giardia* species.

many domestic animals and wildlife [10], and the host-specific groups C–H. Among the latest ones, groups C and D infect dogs, cats, coyotes, and wolves [11]; group E infects cattle, sheep, goats, pigs, water buffaloes, and mouflons [12]; group F infects cats [12]; group G infects rats [12]; and group H infects marine animals [13]. This classification could be of great clinical value given that in addition to many genetic differences found, these genotypes exhibit several relevant biologic differences also. In example, genotype B seems to be more pathogenic than genotype A in humans, although they appear to grow slower [14]. Contradictory, the genetics of parasites of the genus *Giardia* is still poorly understood. Furthermore, recent evidence has cast doubt the classical view of an asexually replicating organism in favor of the occurrence of meiosis and genetic exchange. Such game breaker discovery demanded that the whole population genetics of *Giardia* should be reevaluated to take into account the effect of recombination on *G. lamblia* species [14].

## 3. Life cycle

Life cycle begins with the infection by the ingestion of the cyst. Then the excystation continues, which starts at the stomach triggered by the exposure of the cyst to the gastric acid, the presence of bile and trypsin in the duodenum and/or the alkaline, protease-rich milieu, duodenum [2]. Excystation ends at the proximal small intestine where the emerging parasites (excyzoites) quickly transform into trophozoites that attach to the intestinal epithelial cells using the adhesive disc. The adhesive disc is essential for attachment and appears to play a major role in the virulence of *Giardia* [15]. Several disc-associated proteins have been identified using proteomics [16], and it is clear that the disc is an advanced cytoskeletal structure [17]. At the jejunum, the trophozoites start to encyst forming the wall that enables the parasite to survive outside the host for several weeks in cold water. This process is triggered by a particular composition of biliary secretions, possibly by a deprivation of cholesterol [18]. Regulatory factors are encystation-specific transcription factors, chromatin remodeling enzymes, and posttranslational modifications, which vary their expression in correlation with the variation of antigens on the parasite surface [19]. Finally, trophozoites and cysts are released with the stool, with cysts continuing the transmission of the disease when ingested by another host.

Reservoir hosts include humans, as well as a variety of animals, including cats, dogs, dairy cattle, beavers, and other farm, wild, and domesticated animals such as horses, pigs, cows, chinchillas, alpacas, lemurs, sheep, guinea pigs, monkeys, goats, and rats [20]. However, among all these animal hosts, only beavers, dogs, and humans have been implicated as a source of infection in different waterborne epidemics and outbreaks of giardiasis in humans. Additionally, it is important to highlight the key role of "reverse zoonotic transmission" (zooanthroponotic) in the epidemiology of *Giardia* infections, which means that humans have been identified as the source of infection for beavers, muskrats, and coyotes in the Unites States and Canada [21–23], muskoxen in the Canadian arctic [24], nonhuman primates (gorillas) and painted dogs in Africa [25], marsupials in Australia, house mice in remote islands [26], and marine mammals (seals) in various parts of the world [27].

Even in developed countries such as the United States, it is common to isolate *Giardia* cysts in the water reservoirs and unfiltered water supplies of major cities, the water supply that is not filtrated [28].

## 4. Incidence and burden of disease

*G. lamblia* has been consistently reported as one of the most common pathogens worldwide [29]. Due to high endemicity among humans, and domestic and wildlife animals, it is considered of public health and veterinary health importance [12]. Symptomatic infections have been reported by millions in Asia, Africa, and Latin America by the World Health Organization, which have estimated that it causes 183 million (confidence interval of 95%, 130–262 million) cases of giardiasis [30].

Worldwide, the incidence of Giardiasis has been estimated in $2.8 \times 10^8$ cases per year [31]. However, several epidemiological studies have reported that such rates could be significantly underestimated, with giardiasis prevalence rates ranging from 10 to 20% of the general population [32], from 10 to 50% in developing countries [33, 34], and from 2 to 5% in developed countries [35, 36]. This could be explained by the large fraction of asymptomatic carriers, which regardless of the absence of symptoms also contribute to the transmission of the diseases.

Giardiasis is a ubiquitous disease so it occurred across broad epidemiological contexts and with a broad range of distributions. On one side, in most developed countries such as the United Kingdom [37] and Germany [8], *Giardia* is mostly reported as a rare disease affecting travelers. Furthermore, in the countries such as the Unites States, where *Giardia* is continually under surveillance, *Giardia* has higher incidences (incidence rate ratios, 1.2–1.5) in counties with higher private well reliance compared to counties with lower well reliance [38]. On the other side, in most developing countries, *Giardia* has been associated with poor health hygiene, poor toilet training, overcrowding, and low socioeconomic status [39]. Furthermore, due to the high prevalence of *Giardia* among children, and its higher exposure to dogs in poor setting, it is believed that *Giardia* has developed zoonotic transmission. This possibility has been reported concordantly by genotyping and molecular studies from Mexico [40], Jamaica [41], and Cambodia [42].

The high prevalence of *Giardia* among children raised a major concern about their long-term impacts, which currently have been well documented and reported as more worrisome due to the association between persistent *Giardia* and children's growth [43]. *Giardia* infections can be detected repetitively in over 40% of children suggesting that persistent infections are common and associated with a damage of the intestinal permeability, which—independently of diarrhea—can lead to stunted growth. In fact, according to the results from the MAL-ED birth cohort study, the persistence of *Giardia* before 6 months of age was associated with a −0.29 (95% CI, −0.53 to −0.05) deficit in weight-for-age z score and −0.29 (95% CI, −0.64 to 0.07) deficit in length-for-age *z* score at 2 years [44].

## 5. Giardiasis epidemiology

The *Giardia* cysts are overall highly infectious, and as few as 10 cysts can cause an infection in an individual. Giardiasis prevalence rates have been reported consistently as high among young children from developing countries, with high rates of repeated infection even within the first year of life. However, many developed countries have many regions with endemic giardiasis or regular outbreaks. At these countries giardiasis outbreaks are particularly common during the summer months (likely due to recreational swimming exposure) or throughout the year around day-cares and nurseries, infecting children under 5 years old—and their caregivers—the most [45]. In fact, an investigation of 242 outbreaks, affecting 41,000 persons, reported that most outbreaks resulted from waterborne (74.8%), foodborne (15.7%), person-to-person (2.5%), and animal contact (1.2%) transmission, with waterborne outbreaks been that largest ones in terms of number of cases per outbreak [46].

Surveillance data cases have shown that giardiasis infects populations with a bimodal age distribution, peaking at ages 0–9 years and 45–49 years, without gender preferences [35], and within areas that are endemic, giardiasis commonly shows a seasonal pattern, with most cases occurring in the summer months due to a recent history of drinking untreated surface water and a history of swimming in a lake or pond or swimming in any natural body of fresh water [47]. Other risk factors that have been reported as associated to giardiasis in endemic areas include living in areas that use at-risk tap water (i.e., filtered or unfiltered surface water [48, 49] or unfiltered shallow well water [48]) or in rural areas [49].

One of the most common mechanisms of transmission of *Giardia* infections is a waterborne transmission but also can be transmitted by fecal-oral transmission with contaminated food or direct fecal-oral contact among family members, person-to-person contact, and sexual transmission (oral-anal contact). Although it is unclear which ones are clinically the most important, there is a common understanding about the populations at high risk of giardiasis, which include:

- diaper-age children who attend day-care centers [50, 51];
- adults that work in child-care organizations or day-care centers [52];
- institutionalized individuals [53];
- men who have sex with men [54];
- immunocompromised individuals (chronic variable immunodeficiency, hypogammaglobulinemia, HIV, immunosuppressed individuals, cystic fibrosis, and others) [55, 56]; and
- international travelers or any subject (hikers, campers, sportsman's adventures, and others) exposed to drinking untreated water from lakes, streams, and swimming pools [57].

Waterborne transmission is recognized as the most common transmission, with numerously documented outbreaks throughout the world [46, 58]. This includes the consumption of contaminated water from pools, rivers, or lakes, as well as from contaminated drinking water, either unpurified or inadequately purified. There have been multiple documented cases of

cysts in the municipal water supply here in the United States, although such scenarios do not account for the vast majority of infections [35].

Foodborne transmission of *G. lamblia* is much less common than waterborne transmission, but there are many ways food can be fecally contaminated. For example, street food and any food prepared with the unclean hands of an infected subject could easily transmit giardiasis given a few cysts necessary to transmit the disease. Ingestion of 100 or more cysts is required to ensure infection in humans, but as few as 10 cysts have proven to be enough to infect a volunteer [59].

Fecal-oral transmission is also a significant mechanism of transmission and is the one responsible for the outbreaks in day-cares and nurseries. These outbreaks reflect the close contact between young children, who are significantly more likely to pass the parasite fecal-orally at day-cares than at home. For example, in the Netherlands, where around half of preschool children are cared for in day-care centers, a mean of 2.5 days a week, children at day-care centers are twice as likely to test positive to *G. lamblia* as their home-care counterparts [45], infecting around 4.2% of them [60].

Sexual transmission of *Giardia* is now a very well described form of oral-anal transmission and fecal-oral transmission among men who have sex with men. Currently, there exist a large body of publications that have led to improving our understanding of giardiasis as a sexually transmitted infection. According to these studies, prevalence rates of giardiasis among men who have sex with men range from 2 to 30% [61]. Although giardiasis is not a major cause of AIDS-associated diarrhea, the prevalence of giardiasis, as well as the chronicity of symptoms, is greater in patients with AIDS, especially in developing countries [62]. Overall, every immunocompromised group, like AIDS patients, is recognized as more susceptible to the development of chronic giardiasis [63].

To sum up, it is really important that healthcare providers consider *Giardia* as a differential diagnosis among high-risk populations that match giardiasis epidemiology, and if patients tested positive, it is really important that they provide patients with appropriate therapy and follow-up, as well as proper counseling to increase treatment compliance rate. And in the case of men who have sex with men, also encourage partner notification, and teach them strategies for preventing the transmission of this disease, including the discussion of the risk of enteric infections after oral-anal sexual contact.

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