

## GIARDIA AND GIARDIASIS

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### Summary

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The review summarizes the information related to contemporary taxonomy of species within the *Giardia* genus. The established genotypes of *Giardia duodenalis* are presented. Data from research carried out on the main aspects of epidemiology, pathogenesis, clinical signs, diagnosis and treatment of giardiasis in humans and animals are analyzed. The major subjects of interest for future investigations are outlined.

**Key words:** *Giardia*, giardiasis, *G. duodenalis*, zoonoses

*Giardia* is discovered soon after the invention of microscope in 1681 by Leeuwenhoek. Two hundred years later, Lambl (1859) presented its first more precise morphological description. Today, they are among the most extensively studied protozoa because of their traits as parasites and their place in the classification of single-celled organisms.

*G. duodenalis* is a cosmopolitan and the most frequent intestinal parasite among the population of developing countries. About 200 million of people in the world are with clinically manifested giardiasis, with 500,000 new cases per year (WHO, 1996). *G. duodenalis* is frequently encountered in domestic animals, mostly productive species, dogs and cats. Numerous wild mammalian and bird species are also hosts of *Giardia*.

The infections caused by *Giardia* and their pathogenetic mechanisms are the best studied in men. Infected hosts shed cysts that are resistant in the environment and allow the parasite to be transmitted to another host wither directly, or indirectly

through environmental contamination. Water is an essential factor in the transmission of giardiasis in men, that is why this is the commonest human water-borne disease. Together with cryptosporidiosis, it is a major health problem in utilizing water resources in developed and developing countries (Levine *et al.*, 1990; Thurman *et al.*, 1998; Hoque *et al.*, 2002; Leclerc *et al.*, 2002).

Apart men, infected animals are also involved in water contamination but their role in the epidemiology of human giardiasis is not entirely understood.

The present review aimed to collect and summarize the latest knowledge on *Giardia* in animals and men, to outline the main directions for future research on *Giardia* species and their genotypes.

### MORPHOLOGY

*Giardia* are encountered in two forms – trophozoite and cyst. The motile trophozoite is piriform to oval with bilateral symmetry and dimensions 12–15/6–8 µm.

It has a convex dorsal surface and a large ventral adhesive (sucking) disc. The cell is binucleated, with four pairs of flagella and a pair of delineated median bodies. Cysts are oval-shaped with a thin hyaline wall and dimensions 8–12/7–10 µm. Initially, they are binucleated. The mature cyst has four nuclei, curved median bodies and longitudinal axonemes.

It is considered that *Giardia* originated from primitive single-celled organisms because of their simple intracellular organization lacking mitochondria and peroxisomes (Simpson *et al.*, 2002). That is why *Giardia* play an important role in the elucidation of the evolution of eukaryotes.

#### TAXONOMY AND SPECIES

The *Giardia* genus belongs to type Sarcocystidophora, class Zoomastigophorea, order Diplomonadida, family Hexamitidae. In studies performed between 1920 and 1930, more than 50 *Giardia* species are described, distinguished by the host species in which they parasitize. In 1952, Filice revised the differentiation criteria on the basis of morphological

traits and host specificity. At present, 5 *Giardia* species are recognized according to host species and protozoan morphology: *G. duodenalis*, *G. agilis*, *G. muris*, *G. ardeae* and *G. psittaci* (Table 1). The species *G. duodenalis* (*syn. G. intestinalis*, *G. lamblia*) is the only one encountered in men and more domestic and wild mammals (Thompson, 2002).

The advances in methods for cultivation of *Giardia* allowed to cultivate individual isolates and to produce enough material for genotyping purposes via allozyme electrophoresis. As a result, a lot of evidence for the genetic diversity within the *G. duodenalis* species has been collected. However, not all isolates of the parasite could be studied through *in vitro* cultures. This is valid for a major part of human and canine isolates and is the main cause for the inadequate research on epidemiology and transmission of *Giardia*.

The introduction of PCR techniques has made cultivation unnecessary as they allowed the direct identification of parasites in faecal and environmental samples. Differentiation of existing genotypes has been performed (Hopkins *et al.*, 1997,

**Table 1.** *Giardia* species (Thompson, 2002)

Species	Hosts	Trophozoite morphology	Trophozoite dimensions, µm
<i>G. duodenalis</i>	Man, domestic and wild mammals	Piriform trophozoite with claw-shaped median bodies	12–15/6–8
<i>G. agilis</i>	Amphibians	Long, narrow trophozoite with club-shaped median bodies	20–30/4–5
<i>G. muris</i>	Rodents	Rounded trophozoite with small round median bodies	9–12/5–7
<i>G. ardeae</i>	Birds	Rounded trophozoite with prominent ventral disc notch and rudimentary caudal flagellum. Oval to claw-shaped median bodies	~10/~6.5
<i>G. psittaci</i>	Birds	Piriform trophozoite with no ventrolateral flange and claw-shaped median bodies	~14/~6

1999; Monis *et al.*, 1998; Van Keulen *et al.*, 1998). Multiple PCR studies, analysis of specific genetic loci (rDNA) and investigations on the variety of genes, especially those encoding glutamate dehydrogenase (GDH), elongation factor 1- $\alpha$  (ef1- $\alpha$ ) and triosephosphate isomerase (tpi), revealed a big genetic diversity in the *G. duodenalis* group (Monis *et al.*, 1996; 1998; 1999; Hopkins *et al.*, 1997, 1999). It was proved that *G. duodenalis* was not a single species, but a group of species with genetic and phenotype differences. These species are joined in morphologically close genotypes that exhibit differences in host specificity (Thompson, 1998; Thompson *et al.*, 1999, 2000; Monis & Thompson, 2003). Eight genotypes are formed – A, B, C, D, E, F, G and a group without a name. The geographic distribution of these *G. duodenalis* genotypes is wide.

*Giardia* isolates from humans belong to two assemblages – A and B, and the genetic differences between them are more than those specific for other protozoan species (Andrews *et al.*, 1989; Maurhofer *et al.*, 1995; Monis *et al.*, 1996; Monis & Thompson, 2003). Additional subgroups are also reported in each assemblage. Isolates from Assemblage A belong to two subgroups: AI and AII. Subgroup AI consists of closely related animal and human isolates. The extent of their geographic areal is high and they have a significant zoonotic potential. Subgroup AII consists only of human isolates. Assemblage B includes the subgroups III and IV, the latter being human-specific. Assemblages C and D consist only of canine isolates, group E – isolates from productive animals (cattle, sheep, pigs). Assemblage F comprises feline *Giardia* isolates, and group G – isolates from rats. The group without a name includes iso-

lates from wild rodents (voles and muskrats). Some *Giardia* genotypes exhibit a narrower host specificity, especially those isolated from cats, dogs, rats, voles, muskrats and hoofed animals.

#### PATHOGENESIS

The pathogenesis of giardiasis is not completely investigated. *Giardia* lives and replicates asexually on the small intestine's surface of hosts. According to the most recent studies, giardiasis is a complex of pathophysiological alterations. One of them is the changed permeability of enterocytes resulting from the cytopathological effect of parasite's metabolites (Buret *et al.*, 2002a; 2002b). Peripheral membrane proteins, in particular zonula occludin-1 (ZO-1) that is important for the regulation of epithelial permeability, are destroyed. As a result, enterocytic brush border is damaged, the epithelial permeability is increased, resulting in inflammation and gastrointestinal troubles (Scott *et al.*, 2002). *Giardia* also trigger apoptosis causing loss of epithelium barrier function with a subsequent increase in permeability (Chin *et al.*, 2002). Apoptosis and severity of disease are determined by strain-dependent virulent factors of the parasite as well as by physiological and immunological status of the host (Chin *et al.*, 2002; Scott *et al.*, 2002). It is established that the increased intestinal permeability could also result from increased luminal antigens. This could provoke the appearance of allergic reactions – a complication, often observed in humans infected with *Giardia* (Scott *et al.*, 2002; Chakarova, 2004; Chakarova *et al.*, 2009).

Giardiasis depends on both the parasite and the host. Its variability is manifested in symptoms, clinical signs and the severity of the disease. The described pathophysiological changes could be

encountered in most infected hosts but their consequences could vary according to nutrition mode, the immune status and accompanying enteric infections. Chronic giardiasis in underfed infants infected with other parasites such as *Hymenolepis* or *Ancylostoma* is a primary factor for stunted growth or development (Thompson, 2002; Sackey *et al.*, 2003). In young animals with deficient nutrition, overcrowding or low temperature stress, *Giardia* could be an additional factor for a severe disease. It is believed that they could be the cause for mortality encountered among the nestling ibis (Mc Roberts *et al.*, 1996).

#### EPIDEMIOLOGY

Data about the host specificity of *Giardia* collected so far are contradictory. The results from experimental cross infections showed both host-adapted species and species infecting a wide range of hosts.

Experimental cross infections are not considered reliable partly because experimental animals could not be free from *Giardia* and partly because the high doses of infective cysts used are not likely to occur in a natural infection (Thompson *et al.*, 1990; Monis & Thompson, 2003). The utilization of non-genotyped isolates in these experiments further limits their application. The analysis of results from cross-infection is hindered by the contradictory data for success and failure to reproduced cross infections in various animal species. The cause is probably in the genetic variances of used isolates, differences in cysts viability, immune status of experimental hosts or the low sensitivity of diagnostic techniques. Important data evidencing that dogs could be infected with *Giardia* from Assemblage AI and that beavers are susceptible to infection with human *Giardia* strains are obtained from cross-infection studies

(Thompson *et al.*, 1990; Monis & Thompson, 2003).

On the basis of the information about the transmission of *Giardia* in the different animal species and men, and the genetic data for *G. duodenalis* assemblages, four cycles of transmission ensuring the contact of the parasite with mammalian host, are determined.

The transmission from human to human occurs indirectly through water and food contaminated with cysts or directly, when hygiene is poor. In direct transmission the conditions are favourable, the incidence is high and the prevalence of specific genotypes could be expected. A study on *Giardia* from men and dogs living in one community has shown that human isolates were from Assemblages A and B whereas canine isolates – from C and D (Hopkins *et al.*, 1997). Only one exception was recorded – a dog with mixed B and C infection, probably transmitted from a men. Another investigation of giardiasis among gorillas in a protected territory in Uganda demonstrated that they were infected with Assemblage A and that rangers were probably responsible for the infection (Graczyk *et al.*, 2002). In a study examining 35 human samples performed in the UK, 64% were found to be from Assemblage B, 27% from AII and the rest – from groups B and AII (Amar *et al.*, 2002). A similar research performed in Australia showed that infections with *Giardia* from Assemblage B were more common (70%) as compared to those from Assemblage A (30%) (Read *et al.*, 2001). The Assemblage B genotype has provoked an infection in 21/24 children (88%) from a nursery in the UK (Amar *et al.*, 2002). The ratio of infections by Assemblages B and A in 18 people from

communities growing tea in Assam, India was 61% to 39% (Traub *et al.*, 2004).

A small part of human samples is still genotyped and therefore, the distribution and the prevalence of genotypes infecting men could not be determined. Genetic studies have provided data for the virulence of genotypes from Assemblages A and B. Children in kindergartens from Perth, Western Australia, infected with *Giardia* from Assemblage A exhibited diarrhoea 26 times more frequently than those infected by parasites from Assemblage B (Read *et al.*, 2001). The children infected with *Giardia* from Assemblage B were not treated and stayed in kindergartens. A similar situation was observed in Aboriginal communities in northern Australia when giardiasis was implicated as a cause for alimentary disorders and suboptimal growth and development. In them, isolates from Assemblage B were more commonly found than those from assemblage A (Thompson & Meloni, 1993; Meloni *et al.*, 1995; Hopkins *et al.*, 1999). Similar data are reported by Miteva *et al.* (2009), observing that a higher proportion (66.7%) of people with mixed *Giardia* infection from genotypes AII and B exhibited clinical disease than those infected only with genotype B (41.5%).

Among cattle, giardiasis is widely prevalent and the degree of infection is high (Thompson, 2000; Olson *et al.*, 2004). *Giardia* has been established in beef and dairy cattle with prevalence of 100% (Xiao & Herd, 1994; O'Handley *et al.*, 1999; O'Handley, 2002; Ralston *et al.*, 2003). Faecal cysts appear about the age of 4 weeks. Calves exhibit the highest cyst shedding ( $10^5$ – $10^6$  cysts/g) at the age of 4–12 weeks (O'Handley *et al.*, 1999; Ralston *et al.*, 2003). Increased cyst excretion rate has been observed near the parturition (Ralston *et al.*, 2003). Transmis-

sion is observed in infected calves as well as in chronically infected adult cattle but transmission rate is the highest in dairy calves (Xiao & Herd, 1994; O'Handley *et al.*, 1999; 2000).

Calves are reported to be hosts of one out of two *G. duodenalis* genotypes. The livestock Assemblage E is the most frequently encountered among cattle. Research carried out in Canada and Australia has proved that a small part of cattle in herds (<20%) could harbour more pathogenic, human-infecting *Giardia* from Assemblage A (O'Handley *et al.*, 2000; Appelbee *et al.*, 2002). Also, more detailed and extensive studies in dairy cattle herds in Australia have shown that 100% of calves were infected during the first 12 weeks of life with the livestock genotype.

Investigations among domestic dogs and cats in Australia revealed that *G. duodenalis* was the commonest intestinal parasite (Bugg *et al.*, 1999; McGlade *et al.*, 2003). It is widely distributed in the USA and frequently encountered among pets in other countries as well (Thompson & Robertson, 2003). It is established that *Giardia* infections of companion animals are underestimated. Investigations on canine giardiasis showed that this species is infected by two genotypes – their own and the zoonotic one.

Wild animals are carriers of own *Giardia* genotypes but are also susceptible to infection by zoonotic assemblages.

The major part of beavers, nutrias and deers from North America are infected with *Giardia* with infection rate over 50% (Rickard *et al.*, 1999; Dinlap & Thies, 2002; Dixon *et al.*, 2002; Heitman *et al.*, 2002). Data about the specific genotype are however few. Recent investigations confirmed that beavers and white-tailed deers were carriers of zoonotic *G. duode-*

*nalis* genotypes (Apellbee *et al.*, 2002; Trout *et al.*, 2003).

The genotyping of *Giardia* from wild marsupials in Australia showed that they were infected with a new, genetically different genotype (Adams & Thompson, 2002). Various *Giardia* genotypes have been established in rodents and most birds (McRoberts *et al.*, 1996; Thompson, 2002; Monis & Thompson, 2003). Thus, the established high percentage of infection among a wide range of wild animals could not be a hazard for human health (Kettlewell *et al.*, 1998). Another survey in house mice from two Australian islands exhibited that the animals were carrying several different *Giardia* genotypes (Moro *et al.*, 2003). Zoonotic genotypes were encountered in mice on both islands, but the source of infections has not been identified.

Data from molecular studies proved that productive animals, pets and wild animals were carriers of zoonotic and host-specific *G. duodenalis* genotypes (Thompson, 2002). *Giardia* are considered zoonotic agents by the WHO (WHO, 1979), transmitted either directly by the faecal-oral route or through water.

The consumption of water from unreliable sources is a considerable risk with regard to infection with *Giardia* (Hoque *et al.*, 2002; Jakubowski & Graun, 2002). The major part of human giardiasis outbreaks due to filtered water sources are attributed to flaws and damage (Jakubowski & Graun, 2002). Irrigation waters used for raw food processing are also at high risk for infection with *Giardia* (Thurston-Enriquez *et al.*, 2002). Water contamination may be of human, agricultural or wild animal origin (Heitman *et al.*, 2002). Sewage waters most commonly contained *Giardia* but the concentration of cysts was lower as compared to that of cattle faeces. Several investigations have

proved that the concentration of *Giardia* was lower in wild animals, but in aquatic mammalian species such as beavers and muskrats it was quite high. These results should be interpreted in relation to data for the *Giardia* genotypes encountered in these mammals (Thompson, 2004).

The highest zoonotic risk comes from *Giardia* of Assemblage AI and at a lesser extent, of Assemblage B. There are animal-specific genotypes that are host-specific – for productive animals, dogs and rodents. There are no evidence that they appear frequently among humans and therefore their zoonotic risk is assessed as minimal. Cattle breeding is a potential risk for soil and surface water contamination (Donham, 2000), but with very little risk for the population. The genotyping performed in North America and Australia has shown that the livestock genotype was prevailing in cattle (O’Handley *et al.*, 2000; Hoar *et al.*, 2001). They are susceptible for infection by zoonotic *Giardia* genotypes and infected calves shed about  $10^5$  to  $10^6$  cysts/g (Xiao, 1994; O’Handley *et al.*, 1999). This way, a number of calves infected with parasites from Assemblage A could be hazardous for the farm personnel either directly or indirectly, by water contamination. The studies show that zoonotic genotypes appear only transiently among cattle especially when the transmission rate of the livestock genotype (Assemblage E) is high. This is probably due to competition among the different *Giardia* genotypes. On the other side, research carried out in the National Park of Uganda has shown that men were a source for giardiasis for wild animals and dairy cows (Graczyk *et al.*, 2002).

The detection of *Giardia* isolates morphologically identical to *G. duodenalis* in wild mammals is one of the main factors to determine *Giardia* as zoonotic

agents. Yet, the role of wild animals as a source of infection for men is not clear. Water is the main route of zoonotic transmission of *Giardia*. A relationship between infected wild animals (beavers) and humans drinking from the same water source is evidenced, but the information about the genotypes involved in water-borne infections in wild animals and men is scarce (Thompson, 2004).

Wild animals, and aquatic mammals in particular, are often infected with *Giardia*, but few data are provided to assume that these infections are a primary source for water contamination. Probably, these animals are infected from water contaminated with human or least probably, animal faeces (Monzingo & Hibler, 1987; Bemrick & Erlandsen, 1988; Thompson *et al.*, 1990; Thompson, 1998). A study genotyping *Giardia* obtained from beavers confirmed the hypothesis that these animals were probably infected by a human source (Monzingo & Hibler, 1987; Richard *et al.*, 1999; Dixon *et al.*, 2002). In one study (Appelbee *et al.*, 2002), 12 out of 113 (10.6 %) beaver faecal samples were positive for *Giardia* from the Assemblage A zoonotic genotype.

Investigations on some molluscs demonstrated that they could be an important indicator for the presence of pathogens in water. In North America, *Giardia* isolates from clams have been genotyped (Graczyk *et al.*, 1999) and all isolates were shown to belong to Assemblage A, i. e. a sign for contamination from mammalian, possibly human faeces. Such water-filtering molluscs could accumulate pathogens from water sources and after genotyping, they could serve as bioindicators for contamination with *Giardia* cysts (Thompson, 2004).

The clinical importance of *Giardia* isolates from dogs and cats is minor. In an

Australian study, genotypes of Assemblage A and the canine genotype of Assemblage D were found simultaneously in dogs (Thompson *et al.*, 1999). It is therefore assumed that two cycles of transmission could occur: one only among dogs and another – between pets and their owners (Thompson, 2004). Bugg *et al.* (1999) established that dogs in kennels were more often infected with *Giardia* compared to dogs reared individually. A survey among domestic dogs in Japan demonstrated that all isolates belonged to the specific Assemblage D (Abe *et al.*, 2003). Epidemiological studies in isolated endemic areas where the transmission rate of zoonotic and non-zoonotic genotypes is high (for instance, Aboriginal communities in Australia) showed that dog-specific *Giardia* genotypes prevailed among infected dogs (Hopkins *et al.*, 1997). On the contrary, among men and dogs from tea-growing communities in Assam, north-eastern India, 20% of dogs were infected with *Giardia* from zoonotic genotypes, mainly from Assemblage A, probably a result of the close contacts between men and their pets (Traub *et al.*, 2002).

In conditions when giardiasis is common for dogs and humans, dogs would be continuously exposed to infection with canine and zoonotic *G. duodenalis* genotypes (Hopkins *et al.*, 1997; Thompson, 2002). Australian Aboriginal communities are endemic areas with high frequency of *Giardia* transmission and high infection rates in children and dogs, often over 50% (Meloni *et al.*, 1993; Thompson, 2002). Experimental data show that dog-specific genotypes inhibit competitively the other genotypes and prevent their development in canine small intestine (Thompson *et al.*, 1996). In households and tea-growing communities in India, the frequency of dog-to-dog transmission was

lower than that of genotype A and therefore the latter was likely to persist. This study was the first to provide direct evidence for zoonotic transmission between dogs and men through detection of the same *Giardia* genotype in people and dogs (Traub *et al.*, 2004).

#### CLINICAL SIGNS

Clinical symptoms of giardiasis include acute or chronic diarrhoea, dehydration, abdominal pain and weight loss, that could be of various extent and could be not manifested in infected individuals (Thompson *et al.*, 1993; Rodriguez-Hernandez *et al.*, 1996).

The clinical manifestations of human giardiasis are individual and depend on various factors such as the route of infection, the duration of infection and the physiological condition of the host, and probably, parasitic factors. The incubation period lasts usually 9–15 days. The acute stage generally begins with intestinal troubles, colics, followed by nausea and anorexia. Early signs could be low-degree fever and lethargy. Later symptoms include profuse, watery, foul-smelling diarrhoea, meteorism and enhanced peristalsis with extensive flatulence, eructation and bad taste, epigastric cramping. Rarely, faeces could contain mucus or blood. The acute stage of the disease that lasts for 3–4 days, is similar to that caused by other parasites and a tentative diagnosis of giardiasis is rarely made. Most patients have diarrhoea and the other symptoms are less frequently observed (Chakarova, 2004). Although some acute episodes of giardiasis could resolve spontaneously, they usually pass into a subacute or chronic stage (Lalova, 1977). This stage could be characterized with 2 or more years of intermittent diarrhoea. In people returning from endemic area, the acute

stage could be forgotten and they could exhibit only slight to moderate persisting or recurrent signs. During the chronic stage, lethargy, headache and muscle pain with progressive weight loss, loss of appetite and malabsorption could be present. The chronic infection in children could be manifested with slowed growth (Burke, 1975; Craft, 1982). Urticaria (Webster, 1958), cholecystitis (Soto & Dreiling, 1977) and pancreatitis (Kosyarska, 1977) have been also reported in giardiasis. Rare symptoms may include arthritis (Shaw & Stevens, 1987), retinal arteritis and iridocyclitis (Knox & King, 1982) that resolve after specific anti-*Giardia* treatment. According to experienced investigators, the symptomatology of giardiasis is complex and unpredictable, of individually variable and intermittent nature, characterized with incessantly changing symptoms (Jokipii & Jokipii, 1983). Many of infection episodes resolve after a different period of time and up to 13% of infected adults and 50% of infected children remain symptomatic. The duration of asymptomatic shedding of cysts is not known. The haemogramme of patients with giardiasis is usually normal and eosinophilia is rare. Fat, glucose, lactose, xylose, vitamin A and B<sub>12</sub> malabsorption have been established in some patients (Meyer & Radulesku, 1979; Gillon, 1985; Cordingley & Crawford, 1986).

Cattle giardiasis is clinically and economically important because of the occurring reduction of productivity (O'Handley *et al.*, 2001; Olson *et al.*, 2004). *Giardia* are involved either solely or in combination with other intestinal pathogens in diarrhoea of calves (Xiao & Herd, 1994; Olson *et al.*, 1995; O'Handley *et al.*, 1999; Huetink *et al.*, 2001). Investigations have shown that co-infections by *Giardia* and *Cryptosporidium* in calves are very

frequent and are a major cause of diarrhoea in animals older than 30 days of age. *Giardia* only cause diarrhoea in adult calves. Chronic giardiasis in calves result in weight loss, lower feed conversion ratio and reduced slaughter weight. Identical results have been obtained with experimental infection of lambs (Olson *et al.*, 1995). At the same time, other studies have reported no effect of giardiasis on daily weight gain and feed conversion in fattening beef calves, probably due to the small number of animals used to establish the effect of the infection on productivity traits (Ralston *et al.*, 2003).

*Giardia* infections in dogs and cats are rarely manifested clinically. Clinical giardiasis occurs mainly in connection with rearing conditions – overpopulation, causing stress and aggravation of infection (Robertson *et al.*, 2000). Infected dogs and cats should be preferably treated because of their potential for zoonotic transmission.

The factors predetermining the clinical onset of giardiasis are individual host and environmental factors, as well as the parasitic strain type. The elucidation of the nature of these factors and host interactions, resulting in clinical giardiasis, require future investigation.

## DIAGNOSIS

The detection of *Giardia* by conventional microscopic methods such as ZnSO<sub>4</sub> flotation and centrifugation (Zajac *et al.*, 2002) is still a primary parameter of infection. The detection of *G. duodenalis* by microscopy or ELISA of faeces is of limited epidemiological value, especially when the source of infection has to be determined. Direct immunofluorescence microscopy has improved the sensitivity for detection and quantitation of cysts shed, but the morphological differentia-

tion of identical or similar, but genetically different *Giardia* is not possible (O’Handley, 2002). It is believed that PCR and other molecular techniques have the best sensitivity and specificity for *Giardia* differentiation (McGlade *et al.*, 2003). Molecular techniques provide information about the genotype of *Giardia*, by combination of PCR with restriction fragment length polymorphism analysis (Groth & Wetherall, 2000; Amar *et al.*, 2002; Caccio *et al.*, 2002). These procedures, apart being sensitive and specific, are also fast and easy to perform in a large set of samples (Morgan, 2000).

PCR has the major advantage to allow easy interpretation of results. Nevertheless, the very high sensitivity could be a problem as well. For example, microscopy of samples from domestic cats showed that 5% of them were infected with *Giardia*, whereas by means of PCR, 80% of animals were determined as positive (McGlade *et al.*, 2003). A survey among dogs in India has detected 3% infection rate by microscopy and 20% by PCR (Traub *et al.*, 2004). These results do not take consideration about the irregular cyst shedding with faeces (McGlade *et al.*, 2003) and raise concern for the epidemiological importance of infection of low extent. The filtration, flocculation, flow cytometry, immunomagnetic separation and monoclonal antibody immunofluorescence are among the contemporary methods for detection of *Giardia* in tap water (Slifko *et al.*, 2000). These techniques are used for investigation of natural or treated water sources. PCR methods are more and more used in addition to immunofluorescence for water quality control. Molecular techniques could genotype parasites, isolated from water and determine the source of the contamination (Thompson, 2004).

## TREATMENT AND PREVENTION

Nitroimidazoles and benzimidazoles are efficient anti-*Giardia* medications used for therapy of human infections. The pleasant taste and efficacy of benzimidazoles (albendazole) make them an alternative to the use of nitroimidazoles. In dogs and cats, benzimidazoles such as fenbendazole (Fenbentel) are an excellent alternative to nitroimidazoles (Barr *et al.*, 1998; Zajac *et al.*, 1998).

In productive animal species, benzimidazoles are highly efficient for elimination of *Giardia* in house and range calves (Xiao *et al.*, 1996; Garossino *et al.*, 2001; O'Handley *et al.*, 2001). Fenbendazole-treated calves exhibited an improved microvilli structure of intestinal mucosa on the 7<sup>th</sup> day after a single application (O'Handley *et al.*, 2001). Chemotherapy could be very effective for elimination of the infection. Reinfections appear if environmental sources of contamination are not controlled and the frequency of transmission is high. This is also true for animal and human infections in communities or establishments with inadequate hygiene level that could be easily compromised – dairy farms, kennels, catteries (Reynoldson *et al.*, 1998; Thompson, 1998; O'Handley *et al.*, 2001).

The prevention of human infection implies firstly control of drinking water sources. Systems providing drinking water should use coagulosedimentation and filtration as methods of purification in order to prevent waterborne giardiasis outbreak. According to a report of the USA Center for Disease Control, *Giardia* spp. are proved to cause 9 out of 50 outbreaks between 1986 and 1988, the largest of them affecting more than 500 people. Eight of these are due to failure in systems for water decontamination and 6 – to water system type – water is supplied

without filtration, only after chlorination (Levine *et al.*, 1990). Chlorination only is effective and kills most enteropathogenic organisms, but *Giardia* cysts require higher concentrations and more prolonged exposure to chlorine to be killed, especially in cold water (Jarroll *et al.*, 1981). For individual protection purposes, boiling of water for 1 min destroys *Giardia* cysts. When boiling is impossible, 2–4 drops of household bleach or 2% tincture of iodine could be added to water, and it could be used for drinking after one hour. If water is cold, a longer period of stay might be needed (Jarroll *et al.*, 1981). Cooked food prevents ingestion of cysts from contaminated food, water or hands. At present there are no preparations that could be used for prophylaxis of giardiasis. Because of the numerous sources of infection with *Giardia*, the preventive use of medications could not be recommended, except for highly endemic areas.

In animals, there is no developed prevention strategy of giardiasis. It consists only in timely detection, isolation and treatment of affected animals, maintenance of proper hygiene in animal rearing facilities, adequate nutrition, prevention of overcrowding and restriction of contacts with service personnel.

Some attempts for immune prophylaxis of animals are made. *Giardia* infections stimulate humoral immunity and consequently, the infection is limited in many animal species (Olson *et al.*, 2000). The production of antibodies against the parasite could however last for several months. Investigations in calves showed that even after a 100-day infection, no efficient humoral response against *Giardia* is raised (O'Handley *et al.*, 2003). Dairy cows produce colostrum and milk with anti-*Giardia* activity and the intake

of antibodies with the colostrum could protect calves from infection. *Giardia* are usually detected in calves older than 3–4 weeks of age. Dogs and cats could be vaccinated with a commercial vaccine produced from trophozoite extract (Olson *et al.*, 2000). Immunoprophylaxis appears also promising in beef and dairy cattle.

In conclusion, the zoonotic potential of *Giardia* is beyond any doubt. Productive, companion and wild animal species are carriers of specific and zoonotic genotypes. Aquatic animals could contribute to increase in zoonotic *Giardia* genotypes that could contaminate waters. More extensive research is necessary among humans, animals and water sources with regard to *Giardia* carriership. The obtained isolates should be genotyped in order to investigate the sources of contamination and to undertake efficient measures for reduction of infection rates in both animals and people.

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