CHAPTER

Parasites 15

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15.1 INTRODUCTION

The structure of this chapter is based on the classes of foods that are most frequently the vehicles for parasites. Four main sections include meatborne parasites, fish-borne parasites, parasites transmitted by freshwater-raised plants, and other parasites disseminated in fecally contaminated food and water. Most of the parasites are protozoan.

15.2 MEATBORNE PARASITES

15.2.1 *TOXOPLASMA GONDII*

Toxoplasma gondii is an intracellular protozoan parasite belonging to the coccidian Apicomplexa. Three different genotypes (I, II, and III) are more frequently involved in human cases, type I being the most virulent. Felids, such as the domestic cat (*Felis catus*) and wild Felidae, are the definitive hosts, whereas various warm-blooded animals, including humans, act as intermediate ones. The definitive hosts, more commonly cats, during the primary phase of infection excrete unsporulated noninfective oocysts ($10 \times 11\,\mu m$ in size) along with their feces, which, within one or more days depending on environmental conditions (temperature and humidity), sporulate outdoors in cat feces. The oocysts, being resistant to freezing and drying, can survive and maintain infectivity for many months. In each sporulated oocyst two sporocysts are contained, in each of which four banana-shaped sporozoites ($8 \times 2\,\mu m$) are included.

After ingestion of sporulated oocysts through food or water, sporozoites are released from them into the gut lumen and become tachyzoites that are not gastro resistant, and so are not orally transmissible. Tachyzoites ($6 \times 2\,\mu m$) can enter into all nucleated cells by the apical complex and multiply in many cells of the body by division into two zoites, which may encyst in tissues within 3–4 days as bradyzoites. These are gastro resistant, and so are orally transmissible. Bradyzoites ($7 \times 2\,\mu m$) are enclosed in a tissue cyst, characterized by a thin wall. Tissue cysts are formed in many sites: the CNS where they are round; striated and smooth muscles (where they

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are elongated); and in many edible organs. Tissue cysts may persist for the life of the host, depending on the immune function of the host. When this is depressed, the cyst breaks and bradyzoites transform again into tachyzoites (reactivation process). The production of nitric oxide by activated macrophages is crucial for the maintenance of the bradyzoite stage.

All hosts, including the definitive felid hosts, can acquire infection by ingesting tissue cysts. In the intermediate host, bradyzoites become tachyzoites within 18 h of infection, and the tachyzoite–bradyzoite cycle is repeated. However, in felids, bradyzoites underlie a conventional coccidian cycle [asexual cycle (schizonts) followed by the sexual cycle (gamonts)] in the epithelium of the small intestine. The microgamete (male) fertilizes the macrogamont (female) to obtain an oocyst. The entire asexual and sexual cycle can be completed in the intestine of the definitive host within 3 days of ingestion of tissue cysts (Fig. 15.1).

Parasitemia follows the ingestion of either tissue cysts or oocysts and in the pregnant female, infection of the fetus may follow. Congenital *T. gondii* infections occur frequently in humans, sheep, and goats in which the parasitic infection can cause

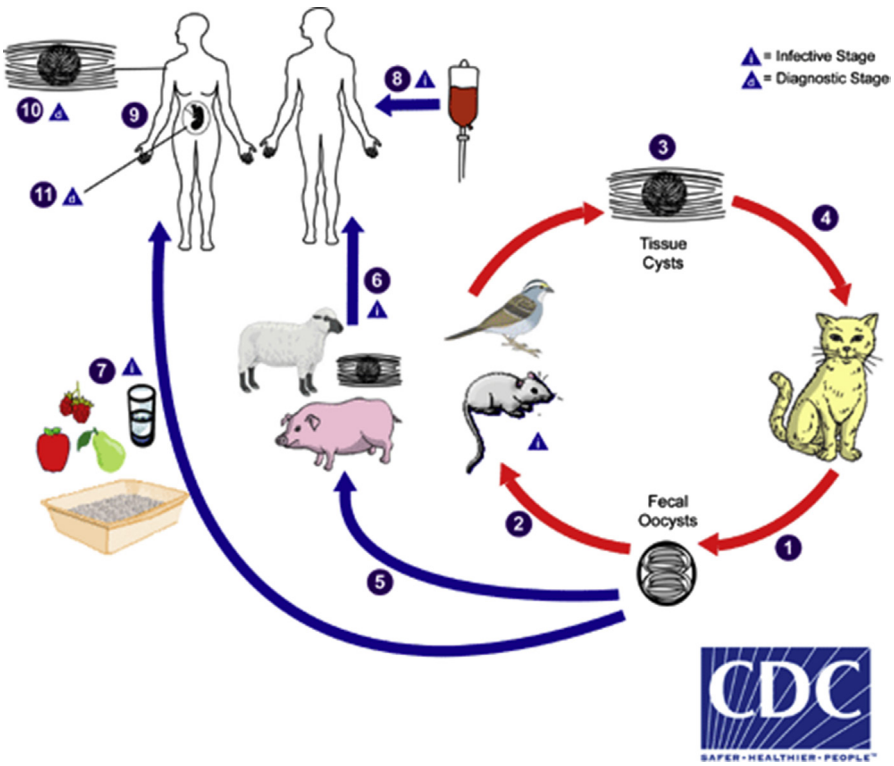

FIGURE 15.1

Life cycle of *Toxoplasma gondii* (http://www.cdc.gov/dpdx/toxoplasmosis/index.html).

abortion. Severe congenital toxoplasmosis has also been reported in cats, dogs, and pigs, but not in cattle or horses. Congenitally acquired toxoplasmosis in humans is generally more severe than postnatal toxoplasmosis. Severely affected infants suffer chorioretinitis, hydrocephalus, mental retardation, and jaundice at the same time. In adults there is a wide range of clinical signs and symptoms, depending on the host and parasite genetics, the mode of infection, immune function, and the organ involved.

Most infections in immunocompetent human beings are asymptomatic. The possible consequences of chronic toxoplasmosis at different levels, such as an increased predisposition to neuropsychiatric diseases (schizophrenia, psychosis, bipolar disorders, Parkinson's disease, etc.), allergic diseases, reduction in driving performance, are a matter of debate. However, in immunocompromised people, such as those suffering AIDS or receiving immunotherapy for cancer or to prevent organ transplant rejection, toxoplasmosis can be severe and sometimes fatal. In normal individuals, *T. gondii* rarely causes serious illness, depending on the virulence of the genotype involved. Flu-like symptoms and lymphadenopathy often occur along with fever, headache, fatigue, and muscle and joint pains. Less frequent symptoms include maculopapular rash, nausea, abdominal pain, and loss of vision following chorioretinitis.

Biological, serological, or histological methods or combinations of them can be useful for the diagnosis of *T. gondii* infection. Direct diagnosis is made by finding the parasite in host tissue, collected by biopsy or at necropsy. The sensitivity of the search for *T. gondii* is increased by inoculation of laboratory animals (generally mice), or tissue cultures, with suitable material.

However, nowadays molecular amplification techniques are preferable because of their relatively high sensitivity and short time to detection. In molecular diagnostics it is usually the B1 gene that is amplified. Most diagnoses in humans are based on indirect methods; searching for specific IgM/IgA and IgG. Currently, infection time, mainly in pregnant women, is evaluated by an IgG avidity test; some individuals show low IgG avidity that may persist for many months after infection, and so a low avidity result may not indicate recent infection in any case.

In immunocompetent individuals toxoplasmosis does not need treatment unless chorioretinitis occurs, in which case sulfadiazine (or clindamycin) and pyrimethamine should be given orally for 1 month. These drugs act on multiplying tachyzoites and have little or no effect on tissue cysts. Folinic acid should also be given to counteract the myelotoxic effects of pyrimethamine. The treatment of immunodeficient patients is more complex. In some countries, spiramycin is used as a prophylactic to minimize transmission of the parasite from the mother to the fetus; however, no regimen can absolutely prevent vertical transmission.

After handling meat, the hands should be washed thoroughly with soap and water. All materials that come into contact with uncooked meat should be thoroughly washed to eliminate the meat fragments, possibly containing tissue cysts. Cooking the meat of any animal to at least 66°C before consumption prevents infection. Particular attention should be paid by pregnant women to avoid contact with cat feces or litter and soil, because of the risk of ingesting oocysts accidentally. This can be done by wearing gloves while gardening and washing vegetables thoroughly before

eating. The risk of ingestion during pregnancy from eating raw shellfish is debatable. Travel to South America is a major risk factor because there are more virulent *T. gondii* genotypes.

To prevent infection caused by ingestion of infected tissues, cats should never be fed with uncooked meat, viscera, or bones, and only dry, canned, or cooked food should be given, which obviously precludes hunting outdoors. In the home, freezing (to −8 to −12°C) overnight can kill most *T. gondii* tissue cysts that may be present in the meat.

A reliable vaccine is not yet available.

15.2.2 *SARCOCYSTIS* SPECIES

Sarcocystis spp. are coccidian parasites with an obligatory prey–predator two-host cycle. They are responsible for a zoonotic infection called sarcocystosis, which has a worldwide distribution in intermediate hosts (pigs and cattle) and in humans. The asexual cycle develops only in the intermediate host, often represented in nature by a prey animal. Sexual stages develop only in a carnivorous definitive host, which becomes infected by eating sarcocysts containing the bradyzoites. These transform into male and female gamonts in the small intestine and, after fertilization, oocysts are produced. The oocysts, containing two sporocysts (each with four sporozoites), sporulate in the lamina propria. Since the oocyst wall is thin it often breaks, releasing sporocysts that may be eliminated along with oocysts in feces, usually 1 week after ingestion of the sarcocysts. The asexual cycle occurs initially in the vascular endothelium, then in circulating blood cells, and finally in muscles. In about 1–2 months, the sarcocysts mature becoming infectious for the carnivore host.

Of the more than 200 different species of *Sarcocystis*, humans serve as the definitive host for two (*Sarcocystis hominis* and *Sarcocystis suihominis*). However, humans also act as accidental intermediate hosts for several species of *Sarcocystis*, of which humans are dead-end intermediate hosts. For example, *Sarcocystis nesbitti* is a snake species that may accidentally cause human muscular sarcocystosis in Malaysia. Symptomatology differs depending on site of infection and varies with the species of *Sarcocystis* responsible for the infection.

Sarcocystis hominis, which is mildly pathogenic for humans, is acquired by ingestion of uncooked beef containing *S. hominis* sarcocysts. When the infection is symptomatic, diarrhea and stomach ache occur. *Sarcocystis suihominis* (which is more pathogenic than *S. hominis*) is acquired by eating undercooked pork. Symptoms include nausea, vomiting, stomach ache, diarrhea, and dyspnea within 24 h of ingestion of uncooked infected pork, and 11–13 days after ingestion sporocysts are shed. The diagnosis of intestinal sarcocystosis is easily made by fecal examination, which, however, does not allow identification of species from the sporocyst morphology. Sporocysts (or oocysts) are shed fully sporulated in feces. Sarcocysts have been found in striated muscles of human beings, mostly as occasional findings. In muscular sarcocystosis, the predominant early clinical features of fever and myalgia are nonspecific and a differential diagnosis with dengue, Chikungunya, leptospirosis, and rickettsial disease is required. When myositis occurs, viral myositis, toxoplasmosis, trichinellosis, and toxocariasis must be excluded, especially in the last two cases when eosinophilia is present.

A definitive diagnosis of sarcocystosis is obtained only after identification of sarcocysts in muscle tissue. Ideally, biopsy specimens are obtained under magnetic resonance imaging guidance; alternatively, the parasites can be sought in clinically affected areas. Findings on histological examination can be variable. The finding of immature sarcocysts with metrocytes (mother cells) suggests recently acquired infection, whereas the presence of only mature sarcocysts indicates a past infection.

Polymerase chain reaction (PCR) has been used to detect *S. nesbitti* in human muscle. However, the sensitivity of PCR detection is not very high; in fact, tissue with visible sarcocysts has been found to give negative results by this technique. Direct 18S rDNA sequencing and nested PCR have also been used in the identification of *S. nesbitti* from human muscle.

Gastrointestinal disease is normally self-limiting and does not need specific treatment. On the other hand, in muscular sarcocystosis, treatment with anticoccidial agents such as trimethoprim-sulfamethoxazole (TMP-SMX), clindamycin, and pyrimethamine has been used, but the optimal approach is not well defined. There is no vaccine to protect livestock or humans against sarcocystosis. To prevent infection humans should never consume uncooked meat.

15.2.3 *TRICHINELLA* SPECIES

Trichinella spiralis is a nematode parasite, which was first described as responsible for human trichinellosis, and is most frequently derived from domestic pork. The other species of *Trichinella* (*Trichinella nativa*, *Trichinella nelsoni*, *Trichinella britovi*, *Trichinella pseudospiralis*, *Trichinella murrelli*, and *Trichinella papuae*) are morphologically similar and can infect humans. No information is available about the ability of newly discovered species such as *Trichinella zimbabwensis* and *Trichinella patagoniensis* to infect humans. All species of *Trichinella* complete their development in a single host. In encapsulated species such as *T. spiralis*, *T. nativa, T. nelsoni, T. britovi*, *T. murrelli*, and *T. patagoniensis*, the first-stage (L1) infective larva is surrounded by a modified skeletal muscle cell called a nurse cell, and outside of that by a collagen capsule that is digested in the stomach when the infected muscle is ingested by a new host. The free larvae then move into the upper small intestine and invade the columnar epithelial cells. Within 30 h, the larvae molt four times before maturing into the adult stages, the males and females. After mating, the adult female sheds live newborn larvae (NBL), also called juvenile larvae, 5 days after infection. Adult worms may persist in the intestine of humans for many weeks. The NBL migrate throughout the body via the blood and lymph circulatory systems, circulating in the capillaries of many different tissues. However, the NBL invade and develop further in skeletal muscle cells only, where they continue to grow and develop during the first 2 weeks of intracellular life until reaching the fully developed L1 infective stage. This stage is resistant to gastric juices, allowing transmission to a new host, which ingests meat containing them. In this way the life cycle is completed.

The disease first manifests as an enteral phase with gastrointestinal symptoms such as nausea, abdominal pain, and diarrhea. Subsequently, a parenteral phase follows with facial edema, fever, and eosinophilia, and after muscle invasion by NBL

with acute muscular pain. Cardiomyopathy as well as CNS involvement (neurotrichinellosis) may complicate the picture, resulting from unsuccessful invasion of larvae into the tissues where they elicit immunopathological processes.

Diagnosis of trichinellosis may be made by either a direct or an indirect demonstration of infection. Larvae that have reached the musculature within the first 3 weeks of infection are more readily detected by compression and histological techniques; otherwise, digestion of muscle tissue is the preferred method. Circulating antibodies can be detected even in lightly infected patients 3–4 weeks after infection and as early as 2 weeks in heavily infected individuals. Among several serological tests utilized over the years the enzyme-linked immunosorbent assay (ELISA) has proven the most reliable when an excretory/secretory antigen is used. However, positive ELISA results should be confirmed with an immunoblot (IB).

Treatment comprises benzimidazoles such as mebendazole (MBZ) or albendazole (ABZ) along with corticosteroids, which should never be administered alone. MBZ is given at a daily dose of 5 mg per kg body weight (administered in two doses) (e.g., in adults, two tablets twice daily) for 10–15 days. The whole treatment cycle may be repeated after 5 days. ABZ should be used at a daily dose of 800 mg/day (15 mg/kg/day) administered in two doses per day for 10–15 days. To prevent possible side effects, blood cell counts and liver function should be regularly monitored.

To prevent *Trichinella* infections in pigs the following should be practiced: (1) strict adherence to garbage-feeding regulations, particularly cooking requirements for waste material (100°C for 30 min); (2) stringent rodent control; (3) preventing exposure of pigs to dead animal carcasses, including pigs themselves; (4) prompt and proper disposal of dead pig and other animal carcasses (e.g., burial, incineration, or rendering); and (5) construction of effective barriers between pigs, wild animals, and even domestic pets.

Meat inspection has proven to be a very successful strategy for controlling trichinellosis, but it is not used for this purpose in the United States. Inspection is currently performed by either of two direct methods: microscopical examination (trichinoscope) or muscle sample digestion. The trichinoscope method is expensive, labor intensive, and is not standardized worldwide. The practical limit of sensitivity is about three larvae per gram of diaphragm pillar muscle. The digestion method is rapidly replacing the trichinoscope method in most countries where meat inspection is mandatory. This method involves the artificial digestion (pepsin-HCl) of diaphragm tissue (or other muscles, depending on the animal species) pooled into batches with the aim of reducing the number of samples and time required for examination. Currently, serological methods for detection of Trichinella infection are not considered to be suitable for the purpose of assuring food safety during meat inspection.

Consumer risk may be reduced by using other strategies such as: (1) cooking meat thoroughly (especially game) reaching an internal temperature of 60°C for at least 1 min; (2) freezing meat to −15°C for 20 days, –23°C for 10 days, or −30°C for 6 days if the meat is less than 15 cm thick. *Trichinella zimbabwensis* and *T. papuae* can infect reptiles such as crocodiles and soft turtles. Human outbreaks have been

described following consumption of the latter infected with *T. papuae*. For this reason, raw food prepared with such meat should be avoided.

When available, a vaccine will be useful in the swine population but not in humans; in fact, education of the population to instigate correct hygiene habits is more effective.

15.2.4 *TAENIA* SPECIES

Taenia saginata ("beef tapeworm") and *Taenia solium* ("pork tapeworm") are the two main species of tapeworm, which can infect humans. In addition, biological and epidemiological studies in Southeast Asia have demonstrated the presence of a subspecies of *T. saginata asiatica* ("Taiwan *Taenia*") for which pigs rather than cattle represent the intermediate host.

The adult stage of these tapeworms resides in the human small intestine. It is composed of a chain (named strobila) of segments (proglottids), containing both male and female reproductive systems. As the segments mature and fill with eggs, they detach and pass out of the anus, either free or in the fecal bolus. The viability of an adult tapeworm may last up to 30–40 years. The worm sheds thousands of eggs from a host per day (500,000–1,000,000), thus causing high levels of environmental contamination. These eggs contain the oncosphere, i.e., the infective stage, which can mature in the environment. An individual hosting a tapeworm may suffer symptoms variable in their intensity, such as nervousness, insomnia, anorexia, loss of weight, abdominal pain, and digestive disturbances.

If *Taenia* eggs are ingested by cattle or pigs, the oncosphere stage is released in the intestine. The oncosphere penetrates the gut and migrates throughout the body via the circulatory system and is distributed to different tissues (mainly skeletal muscle or heart muscle in the case of *T. saginata*) where it develops into the cysticercus stage, a fluid-filled cyst or small bladder. When beef or pork, either raw or poorly cooked, is ingested by humans, the larval cyst is freed and it attaches to the intestinal wall by a small head (scolex) with suckers. After 2 months, the tapeworm reaches maturity and completes the life cycle, when it begins to shed eggs.

When *T. solium* eggs are ingested (in the absence of taeniasis) or released in the intestine (concomitantly with taeniasis) cysticercosis also occurs in humans, where cysticerci are mainly localized in the liver, brain, CNS (neurocysticercosis = NCC), skeletal muscle, and the myocardium. NCC is increasingly recognized as a serious public health problem, especially in developing countries where it represents the most important cause of not genetically determined epilepsia. In the United States, Canada, and western Europe, human cases are generally imported from Latin America, China, and Africa where prevalence is relatively high.

Most cases of human cysticercosis (*T. solium*) are asymptomatic. Neurological symptoms occur when the parasite degenerates and dies after many years of infection, and the inflammatory response, no more inhibited by the parasite, prevails. Symptomatic infections may be characterized as either disseminated, ocular, or

neurological. Disseminated infections may localize in the viscera, muscles, connective tissue, and bone; subcutaneous cysticerci may present a nodular appearance. These localizations are often asymptomatic, but may produce pain and muscular weakness. CNS involvement may include the invasion of the cerebral parenchyma, subarachnoid space, ventricles, and spinal cord. The larval cysts may persist for years. Symptoms of infection may include partial paralysis, dementia, encephalitis, headache, meningitis, epileptic seizures, and stroke. Ocular infections are localized most commonly in regions such as the vitreous body and subretina. During the early stage of infection, retinal edema, exudation, and hemorrhage occur. As the lesion develops, the inflammatory reaction can lead to severe uveitis, retinal and vitreous proliferation, retinal vascular occlusion, and exudative retinal detachment. When the parasite dies, a large quantity of toxins are released into the vitreous body, which causes more severe histoclasia and visual impairment.

Diagnosis of taeniasis is based primarily on identification of proglottids or eggs in the feces. A species-specific DNA probe for *T. solium* and *T. saginata* eggs has been developed for use in a dot blot assay. As regards serological diagnosis of humans with cysticercosis, it is advisable to perform an IB, which uses purified worm glycoproteins as antigens. This method affords a guaranteed 98% sensitivity and 100% specificity in proven parasitological cases. More recently, an ELISA with a low molecular weight recombinant protein (8 kDa) from *T. solium* was shown to be optimal in terms of both specificity and sensitivity.

An antigen-capture immunodiagnostic system based on the use of the monoclonal antibody HP10, specific for a repetitive glyco-residue secreted by *T. saginata* and other taeniids metacestodes, has been used with both serum and cerebrospinal fluid samples. This capture ELISA showed greatest sensitivity with patients harboring several live cysticerci or affected by severe NCC.

For taeniasis, niclosamide is the drug of choice; 2 g administered orally in a single dose is recommended for adult patients. Management of NCC involves the use of cysticidal therapy, symptomatic therapy (antiepileptics), corticosteroids to prevent allergic manifestations during cysticidal treatment, and sometimes surgery depending on the localization of cysticerci in the brain. Cysticidal therapy essentially includes two drugs: (1) ABZ used at a dose of 15 mg/kg/day (maximum 800 mg), usually for 28 days, although shorter durations of 8–14 days have also been used; side effects depend on the dose and duration of therapy; and (2) praziquantel (PZQ) used at a dose of 50 mg/kg/day. The usual duration of PZQ therapy is for a period of 15 days; side effects are dose related, although uncommon. Some authors suggest a combination of both ABZ and PZQ.

To prevent taeniasis, meat should be cooked to at least 60°C. In endemic areas, the control of cysticercosis is based on the introduction of strict hygienic rules.

Among several candidate vaccines, the oncospheral stage-specific TSOL18 antigen is a promising one. TSOL18 is able to confer almost 100% protection, and is ready to be used in control programs that are in progress in different endemic regions.

Swine and bovine cysticercosis also represents an important economic issue in certain regions because of condemnation of infected carcasses at slaughter.

15.3 FISHBORNE PARASITES

15.3.1 *CAPILLARIA PHILIPPINENSIS*

Capillaria philippinensis is a trichurid nematode that causes intestinal capillariasis. The nematode is small; males are 1.5–3.9 mm and females 2.3–5.3 mm in size. The worms reside in the small intestine and eggs produced by the females pass in the definitive host's stool and reach fresh water. The eggs are able to survive in water for months. Once the eggs are eaten by freshwater fish, they hatch, penetrate the fish intestine, and migrate to the edible tissues. When the fish is eaten by a definitive host, the larvae develop into adults in 2 weeks, and the first generation of female worms produce eggs that pass in the feces. There are always a few adult female worms that remain in the intestinal tract and continue to produce unembryonated eggs, which embryonate in the intestine and release larvae that develop into adults. This is a process of autoinfection that maintains the infection and increases the parasite population. Autoinfection is an integral part of the life cycle.

Humans are presently the only confirmed definitive host, but fish-eating birds are probably the natural hosts. Many species of small freshwater fish in the Philippines and Thailand are intermediate hosts. Human infections are reported mostly from those countries, with sporadic reports from Japan, Korea, Taiwan, Egypt, India, Iran, Italy, and Spain. Patients with intestinal capillariasis usually present watery diarrhea, weight loss, abdominal pain, borborygmus, muscle wasting, weakness and edema. Laboratory examination showed low levels of potassium and albumin in the blood, and malabsorption of fats and sugar. These patterns may result from the secretion of a proteolytic substance by *C. philippinensis*, and/or direct penetration of the intestinal wall, that causes cellular injury and dysfunction. As the number of parasites increases in the human host, symptoms become more severe. The loss of potassium, protein and other essential elements leads to weight loss and cachexia. If treatment is not started in time, the patient will die. Treatment is by restoration of potassium levels, and by administration of an antidiarrheal drug and an anthelminthic. The recommended drugs are mebendazole at a rate of 400 mg/day, administered in a series of doses for 20 days, or ABZ at a dose of 400 mg/day for 10 days. Diagnosis of *C. philippinensis* infection is by detection of eggs, larvae, and adults in the patient's feces. Immunodiagnosis may be used as a supplementary diagnostic tool, which helps to detect *C. philippinensis* infection. The indirect diagnosis of intestinal capillariasis has been performed using *T. spiralis* antigens.

15.3.2 *GNATHOSTOMA* SPECIES

Gnathostomes are nematodes belonging to the order Spirurida, which are biologically characterized by requiring one or more intermediate hosts in their life cycles. The genus has 12 species with only four recorded as infecting humans: *Gnathostoma spinigerum*, commonly found in wild and domestic cats and dogs in India, China, Japan, and southeast Asia; *Gnathostoma hispidum*, found in wild and domestic pigs in Europe, Asia, and Australia; *Gnathostoma doloresi*, found in wild boars;

and *Gnathostoma nipponicum*, found in weasels in Japan. *Gnathostoma* larvae are short and stumpy with a globose head armed with rows of hooklets. The body also has spines half way down its length. The larvae measure 11–25 mm for males and 25–54 mm for females. Adult worms live coiled up in the wall of the fish-eating mammals' stomach, producing a tumor-like mass. Eggs produced by female worms pass in animal feces, reach water, and embryonate. The larva hatches from the egg and is eaten by a freshwater copepod, where it develops into the second stage. When the infected copepod is eaten by a bird, fish, frog, turtle, or mammal, the larvae are freed in the intestine and develop into the third stage, which does not develop further. The larvae encyst in the muscles of their transport hosts, where they remain as infectious larvae. When the second intermediate host or paratenic host is ingested by the final host, the larva will penetrate the intestinal wall, migrate to the liver and abdominal cavity, and, after about 4 weeks, return to the peritoneal cavity, where it penetrates the stomach wall and provokes a tumor. Cats and dogs are definitive hosts for *G. spinigerum*, while pigs are the definitive host for *G. hispidium.* Intermediate hosts are fish, frogs, snakes, chickens, ducks, rats, etc. The major source of infection in Thailand is a snake-headed fish, *Ophicephalus* sp., which is eaten raw or fermented. In Japan, raw fish is eaten as shashimi and causes infection, and in Mexico fish prepared as ceviche is a source of infection.

Humans are not natural hosts and do not allow parasite maturation. Human gnathostomiasis is caused by migrating immature third-stage larvae (L3) after eating raw or inadequately cooked freshwater fish or other intermediate hosts such as snakes, frogs, and chickens. However, two alternative routes of infection have been suggested; ingestion of water containing infected copepods (thus taking the place of a second intermediate host) or by penetration of the skin of food handlers by L3 from infected meat. Symptoms in humans occur as the late L3 migrate through the tissues, causing intermittent symptoms of cutaneous or visceral larva migrans (VLM). The larvae have been observed to move at 1 cm/h to the skin through the subcutaneous tissue causing the typical migratory swellings (cutaneous disease) and from there they may penetrate into deeper tissues and viscera to involve the lungs, eyes, ears, gastrointestinal and genitourinary systems, and, rarely, but often fatally, the CNS (visceral disease).

The triad of eosinophilia, migratory lesions, and verified exposure risk are highly suggestive of a diagnosis of gnathostomiasis. Currently, a number of serological tests are available for the diagnosis of gnathostomiasis. The most widely used in Europe as well as in endemic countries (Japan, China, Southeast Asia, India, and more recently Central and South America) is an IB to detect the specific 24-kDa band considered diagnostic of gnathostomiasis. A definitive diagnosis is made only when the larvae are recovered from surgical specimens, urine, or vaginal discharge. In humans, high cure rates have been achieved using a dose of 400 mg of ABZ twice a day for 21 days. Larvae may emerge from the subcutaneous tissue following long-term treatment with ABZ, making them more accessible and so possibly amenable to excision. Ivermectin has been shown to be an effective treatment, either as a single dose of 0.2 mg/kg or as doses of 0.2 mg/kg on 2 consecutive days.

15.3.3 ANISAKIDAE FAMILY

Anisakidosis is a nematode larval infection caused by any member of the family Anisakidae, whereas anisakiasis is the disease caused by members of the genus *Anisakis*. *Anisakis simplex* and *Pseudoterranova decipiens* are the anisakid species most commonly involved in infection. These are parasites of marine mammals found in the stomachs of cetaceans and pinnipeds. Eggs are passed in the feces and remain on the ocean floor until a larva develops. The larva hatches from the egg and is eaten by a small crustacean (euphausid). The L3 develops in the crustacean and, when it is eaten by squid or marine fish, the larva penetrates the gut and passes into the peritoneal cavity or into the musculature. When eaten by the mammalian definitive host, the larva is then released from the fish or squid tissue and enters the animal's stomach.

Whales, dolphins, and porpoises are definitive hosts for *A. simplex*, and seals, sea lions, and walruses are the hosts for *P. decipiens.* Mackerel, herring, cod, salmon, and squid are second-intermediate hosts for *A. simplex*, and cod, halibut, flatfish, and red snapper are the second-intermediate host for *P. decipiens*.

The L3 are the stage of the parasite that is pathogenic to humans. The larvae are creamy white to yellowish brown in color, 20–50 mm long, and 0.3–1.22 mm wide.

The transmission of these foodborne pathogens is particularly associated with traditions of consumption of raw or undercooked fish. A number of fish dishes are considered high risk for the acquisition of anisakidosis, including Japanese sushi and sashimi, Filipino bagoong, Dutch salted or smoked herring, Scandinavian gravlax, Hawaiian lomi-lomi and palu, South American ceviche, and Spanish pickled anchovies. Usually within a few hours after the ingestion of infected marine fish, the L3 penetrate the tissue of the gastrointestinal tract and cause an acute and transient infection that may lead to abdominal pain, nausea, vomiting, and/or diarrhea. The L3 induces the formation of an eosinophilic granuloma. Some patients develop syndromes simultaneously exhibiting clinical manifestations of allergy and infection after eating living parasites. The *Anisakis*-associated allergic response has been identified as associated with *A. simplex* and more recently *Anisakis pegreffii* and may lead to severe clinical symptoms including anaphylactic shock. Allergic reactions to *A. simplex* antigens are associated with the production of specific IgE, although this is detected in all patients following anisakiasis, including patients without allergic symptoms. *Anisakis simplex* is more frequent in coastal populations and among 20–50-year-old males. *Pseudoterranova decipiens* has been reported infecting humans in northern Japan and along the California coast. In these areas, sea lion populations have increased to high levels along with the intermediate host. *Pseudoterranova decipiens* infection usually causes "tickle throat."

A diagnosis of infection is made by collecting the parasite surgically following the occurrence of an acute abdominal pain. However, the use of fiber-optic gastroduodenoscopy instead of surgery is now being used to recover the larvae. Immunodiagnostic methods have also been used to diagnose infection. The use of ABZ as a treatment is debatable. To prevent anisakiasis, freezing (at −35°C for 15 h or at −20°C or below for 7 days or at −15°C for at least 96 h) and cooking (at >60°C

as core temperature for at least 1 min) remain the processes recommended to guarantee destruction of the larvae, under well-defined conditions. Nevertheless, it should be noted that these treatments might not inactivate allergens.

15.3.4 *CLONORCHIS SINENSIS*

Clonorchis sinensis, the Chinese liver fluke, is widespread in Asia, being reported from China, Japan, Korea, Taiwan, and Vietnam. The adult worms measuring 10–25 mm long and 3–5 mm wide, are lanceolate, flat, and pinkish in color. The hermaphroditic adult worms reside in the distal tributaries of the bile passages. Eggs produced by the worms pass down the bile ducts to the intestines and are passed with the feces. The eggs must reach fresh water where they are eaten by the first-intermediate snail host. A broad spectrum of mollusk species belonging to five families (Assimineidae, Bithyniidae, Hydrobiidae, Melaniidae, and Thiaridae) acts as first-intermediate host for *C. sinensis*. The larval miracidium is released from the egg and enters the snail tissue to go through polyembryony producing sporocysts, rediae, and cercariae. The cercariae leave the snail and search mainly for cyprinid fish second-intermediate hosts, most commonly topmouth gudgeon (*Pseudorasbora*) or carp (*Ctenopharyngodon* spp.). However, some additional 60 fish species not belonging to the family Cyprinidae can host *C. sinensis*. The cercariae enter the skin of the fish and encyst as metacercariae. When the fish is eaten raw, pickled, or smoked by humans and animals, these acquire the infection and the metacercariae excyst in the intestine of the definitive host and migrate through the Ampulla of Vater to the bile radicles where they mature into adults after at least 1 month. Flukes have also been detected in the duodenum and stomach. In humans, flukes can survive for 20–25 years.

The parasites in the biliary tract attach to the bile duct epithelium using their suckers and provoke hyperplasia of the epithelium, leading to fibrous development of the ducts. Fever and chills may develop, and the liver may become large and tender. The magnitude of the pathology caused by *C. sinensis* depends on their number, the duration of the infection, and the susceptibility of the host. Long-term infections may lead to carcinoma of the bile ducts. The International Agency for Research on Cancer has classified *C. sinensis* and *Opisthorchis viverrini* as group 1 carcinogens.

15.3.5 *OPISTHORCHIS VIVERRINI*

Opisthorchis viverrini is found in Thailand and surrounding countries in Southeast Asia, whereas *Opisthorchis felineus* is reported from Eastern Europe, Siberia, and the European Union. The life cycles of these flukes are similar to that of the Chinese liver fluke, but the intermediate hosts are different. Three mollusk species of the genus *Bithynia* (*Bithynia inflata*, *Bithynia leachi*, and *Bithynia troscheli*) play the role of first-intermediate hosts for *O. felineus.* Larval stages of *O. viverrini* have been detected in *Bithynia funiculata* and in two subspecies of *Bithynia siamensis* (*B. siamensis siamensis* and *B. siamensis gomiomphalos*). There are many fish second-intermediate hosts for these species, but the most important hosts of *O. viverrini*

are cyprinoid fish of the genera Cyclocheilichthys, Hampala, and Puntius. On the other hand, in Europe, *O. felineus* metacercariae have been detected in *Alburnus alburnus*, *Abramis brama*, *Abramis ballerus*, *Blicca bjoerkna*, *Idus idus*, *Rutilus rutilus*, *Scardinius erythrophthalmus*, and *Tinca tinca* at prevalence rates of up to 95%. Human infections are acquired by eating improperly cooked, preserved, or fermented fish. Diseases associated with these species are similar to those caused by *C. sinensis*. The diagnosis of the liver fluke infection is by detection of eggs in feces or duodenal aspirates, or by immunological methods. Currently, the drug of choice to treat patients with liver flukes is PZQ.

According to the WHO (1995) the recommended dose for the treatment of liver fluke infections is 40 mg/kg body weight. PZQ should be administered at a daily dose of 75 mg/kg body weight divided into three subdoses of 25 mg/kg body weight at 4–5 h intervals. This treatment gives 100% and 80–85% cure rates for *O. viverrini* and *C. sinensis* infection, respectively. To prevent clonorchiasis and opisthorchiasis, freshwater fish, which can act as a host for the parasite, should be cooked until the core reaches 65°C for at least 1 min (EFSA, 2010). *Opisthorchis felineus* metacercariae in fish fillets can be killed by freezing at −28°C for 20 h, at −35°C for 8 h, or at −40°C for 2 h. Metacercariae may also be killed by freezing at −10°C for 5–70 days, depending on the size of the fish. Devitalization of *O. felineus* metacercariae present in tench muscles has been demonstrated at −18°C for 96 h.

15.3.6 OTHER FISHBORNE FLUKES

Intestinal trematodes, although less commonly associated with mortality than other groups of fishborne flukes, are responsible for significant morbidity. The most common species belong to two families; Heterophyidae and Echinostomatidae. Fertilized eggs are released into the environment with the feces of their host, and the eggs may reach water sources such as ponds, lakes, streams, or rivers. Either eggs, each containing a miracidium, are eaten by an appropriate snail and they hatch inside the snail, as species of Opisthorchiidae (Heterophyidae) do, or the eggs hatch and a free-living miracidium is released into the water (Echinostomatidae).

Metagonimus yokogawi and *Heterophyes heterophyes* are two tiny intestinal heterophid parasites that pass eggs into the feces. *Pirenella* spp. of snails are the first-intermediate host for *H. heterophyes*, and *Semisulcospira* spp. are snail hosts for *M. yokogawi*. *H. heterophyes* is endemic in Egypt and China, and *M. yokogawi* in Japan and Korea. Both species of flukes are very small, only 1–2 mm long and 0.3–0.7 mm wide. Adult flukes inhabit the mucosa of the middle part of the small intestine. Attachment of the fluke may cause small ulcers in the mucosa leading to abdominal pain, diarrhea, and lethargy. The severity of symptoms depends on the number of worms involved. Eggs released by the worms may penetrate the gut and migrate to vital organs via the circulatory/lymphatic system. In fact, there are reports of eggs of these worms in ectopic locations such as the brain and heart. Infections are often reported in sports fishermen who eat the fish raw shortly after catching them in the cool mountain streams in northern Japan. The diagnosis of infection is based on finding eggs in the feces.

Echinostomes are intestinal fluke parasites that are acquired by eating a variety of aquatic animal life, especially freshwater fish in Asia. They are generally parasites of other animals, and humans are accidental hosts. The miracidium hatches from the egg and enters a susceptible snail, *Gyraulus* spp.; cercariae produced may enter other snails or other aquatic animals. *Echinostoma lindoensis*, *Echinostoma ilocanum*, and *Echinostoma misyanum* are endemic in certain parts of Indonesia, the Philippines, and Malaysia. The parasites have a characteristic collar of spines around the oral sucker, are spindle shaped, and measure 4–7 mm by 1.0–1.35 mm. The severity of symptoms is generally worse than with heterophyids. Mechanical irritation by the flukes and allergic reactions caused by their toxic metabolites constitute important elements of the pathogenesis, which also depends on the intensity of infection. Diagnosis is based on the presence of eggs in the feces. Treatment of heterophyid and echinostome infection makes use of 10–20 mg/kg PZQ in a single oral dose.

15.3.7 *DIPHYLLOBOTHRIUM LATUM*

Diphyllobothrium latum is a tapeworm. There are several species of fish tapeworms, but the most important is *D. latum*, which is widespread in the temperate and subarctic regions of the Northern Hemisphere and has also recently been found in South America (Chile). Recent cases of diphyllobothriosis caused by *Diphyllobothrium dendriticum* have been recorded in European countries where it has not previously been observed (the Netherlands, Switzerland, and the Czech Republic). In Europe, *D. latum* seems to be emerging in subalpine lakes, with more than 100 human cases having been reported in the last decade in the French, Swiss, and Italian regions bordering these lakes. This cestode typically parasitizes piscivorous birds (such as gulls) and mammals (such as foxes or bears); human infections have usually been considered accidental. Fish-eating mammals are definitive hosts. The worm resides in the small intestine and produces eggs, which pass in the feces into water. A ciliated larva (coricidium) develops in the egg and, when released from the egg, swims in the water until ingested by a copepod. A procercoid larva develops in the copepod and, when eaten by a fish second-intermediate host, the larva develops into a plerocercoid larva, or sparganum, in the fish flesh. When the fish is eaten uncooked, the plerocercoid larva attaches to the intestinal mucosa and develops into an adult tapeworm. The worm is the largest parasite to infect humans and ranges in length from 2 to 15 m with a maximum width of the gravid proglottids of 20 mm. The scolex is 2 mm long and 1 mm wide, and has a dorsal ventral groove or bothrium. The mature proglottid has a rosette-shaped uterus that fills with eggs.

There is little disease associated with fish tapeworm infections. The worms may compete with the host for vitamin B12 and cause megaloblastic anemia. This has been observed more frequently in Finland, where patients experience fatigue, weakness, the desire to eat salt, diarrhea, epigastric pain, and fever. Diagnosis of infection is made by finding eggs in the stools. A single dose of PZQ (25 mg/kg) is usually sufficient to eliminate the tapeworm, although as an alternative, niclosamide (2 g in a single dose) can also be effective.

Various species of fish serve as the second-intermediate host, including pike, perch, turbot, salmon, and trout. Infection occurs if the fish is poorly cooked, pickled, or smoked. The Japanese eat the fish as shashimi or sushi, which can lead to a risk of infection. Definitive hosts, other than dogs, include foxes, bears, mink, seals, and sea lions.

Occasionally, humans may acquire infections with the spargana of *Spirometra* spp. This is a diphyllobothrolid tapeworm of felines and canines. The plerocercoid larva produces a larva migrans syndrome that causes transient migratory swellings.

15.4 PARASITES TRANSMITTED BY FRESHWATER-RAISED PLANTS

Freshwater-raised plants, such as watercress, are part of the diet of many cultures, and are the route of transmission of the fluke parasites that cause human fascioliasis, which is an important health problem in many regions both for humans and livestock. The disease is caused by species belonging to the family Fasciolidae, in particular *Fasciola hepatica*, which shows worldwide distribution and *Fasciola gigantica*, which is limited to regions of Africa and Asia.

Fasciolids cause major human health problems in Andean countries, the Caribbean, North Africa, the Near East, Southeast Asia, and Western Europe. In human hyperendemic areas, children and females are the most affected. Human fascioliasis shows a marked heterogeneity of epidemiological situations and transmission patterns. Variations in climatic factors, and anthropogenic environmental modifications, give rise to variations in the seasonality of fascioliasis and in long-term disease risk trends. The rapid and potent ability of fasciolids to suppress the immune response explains why hosts do not develop resistance to them, and also the frequency of coinfection by other pathogens. The two-host life cycle is similar in both species, and includes specific freshwater snails, such as species of the genera *Galba*, *Fossaria*, and *Pseudosuccinea*, as vectors. The expansion of *F. hepatica* from Europe to other continents has been attributed to the parasite adapting to new snail species and also to the spread of the main intermediate host snail to other continents. In contrast, the intermediate host snails of *F. gigantica* (*Radix natalensis* and *Radix auricularia*) seem to have a poorer diffusion capacity and hence a smaller geographic distribution. *Fasciola* parasites develop into adult flukes (*F. hepatica* can be up to 30 mm by 13 mm; *F. gigantica* can be up to 75 mm) in the large bile ducts of infected mammals, which pass immature *Fasciola* eggs in their feces. In fresh water, and after several weeks, the eggs hatch and the miracidium reaches and infects a snail host. Under optimal conditions the development process in the snail may be completed in 5–7 weeks; cercariae are then shed into the water and they lose their tails when they encyst as metacercariae (infective larvae) on water plants. In contrast to cercariae, metacercariae have a hard outer cyst wall and can survive for prolonged periods in wet environments. In humans, maturation from metacercariae into adult flukes takes approximately 3–4 months. The disease is mainly confined to the liver, where

parasites digest the tissue, causing extensive parenchymal destruction with intensive hemorrhagic lesions and immunological and inflammatory reactions. Juvenile flukes may cause ectopic fascioliasis. Clinical manifestations are evident in both invasive and biliary periods. Anemia, caused by blood loss in the bile ducts, is one of the most characteristic signs, especially in heavier infections. Liver fibrosis characterizes the late phase of the disease but, unlike clonorchiasis or opisthorchiasis, no association with biliary carcinoma has been reported. Diagnosis is mainly made by coprological and serological techniques. Among the drugs that are used, triclabendazole is presently the drug of choice (a single dose of 10 mg/kg, or in the event of treatment failures, the dosage can be increased to 20 mg/kg split into two doses given within 12–24 h). Unfortunately, a human vaccine is not yet available.

15.5 PARASITES DISSEMINATED IN FECALLY CONTAMINATED FOOD AND WATER

15.5.1 *CYSTOISOSPORA BELLI* (PREVIOUSLY *ISOSPORA BELLI*)

Cystoisospora belli is a coccidian protozoan responsible for intestinal coccidiosis in humans. Its oocysts are elongate and ellipsoidal, measuring 20 × 19 μm. After sporulation, they contain two ellipsoidal sporocysts without a Stieda body (which is an organelle located at the polar region of other coccidia). Each sporocyst (measuring 9–14 × 7–12 μm) contains four crescent-shaped sporozoites and a residual body. Sporulation occurs within 5 days, both within the host and in the external environment. Thus both unsporulated and sporulated oocysts may be shed in the feces.

Infection occurs by the ingestion of food contaminated by oocysts. Merogony and gametogony occur in the epithelial cells of the upper small intestine, from the level of the crypts to the tips of the villi. In AIDS patients, the parasite may be disseminated to extraintestinal organs including mesenteric and mediastinal lymph nodes, liver, and spleen. Single zoites surrounded by a capsule (cyst wall) have a prominent refractile or crystalloid body indicating that the encysted organisms are sporozoites. Organisms with a cyst wall are found only in extraintestinal organs.

Cystoisospora belli can cause severe symptoms with an acute onset, particularly in AIDS patients. Infection has been reported to cause fever, malaise, cholecystitis, persistent diarrhea, weight loss, steatorrhea, and even death.

The stools during infection are fatty and at times very watery. Diagnosis can be established by finding characteristic bell-shaped oocysts in the feces or coccidian stages in intestinal biopsy material. Affected intestinal portions may have a flat mucosa similar to that which occurs in sprue (celiac disease). *Cystoisospora belli* oocysts can be detected using the innovative FLOTAC dual technique, which uses flotation solutions, but also fluorescence microscopy can aid diagnosis, because of its autofluorescence. A real-time PCR assay targeting the internal transcribed spacer 2 region of the ribosomal RNA gene was developed to detect *C. belli* DNA in fecal samples.

Cystoisosporiasis in immunocompetent patients is usually self-limiting and does not require treatment but only symptomatic support, such as antimotility drugs to control diarrhea. In immunocompromised patients the most effective therapy is a combination with dihydrofolate reductase inhibitors and sulfonamides, specifically TMP-SMX.

15.5.2 *CYCLOSPORA CAYETANENSIS*

Cyclospora cayetanensis is an intestinal coccidian protozoan of humans. Its oocysts are approximately 8 μm in diameter and they contain two ovoid sporocysts (4 × 6 μm). Each sporocyst has two sporozoites. Unsporulated oocysts are excreted in feces and sporulation occurs outside the body. No other stage in the life cycle is known. Outbreaks of cyclosporosis in humans have been associated with ingestion of fruits, salads, and vegetables contaminated with oocysts. Both immunocompetent and immunosuppressed patients of all ages may suffer diarrhea, fever, fatigue, and abdominal cramps. Infection has been reported in several countries.

Diagnosis can be made by fecal examination. Oocysts, remarkably uniform, contain a sporont (inner mass) that occupies most of the oocyst. They are autofluorescent and acid-fast stained, thus imposing a differential diagnosis with those from *Cryptosporidium*. Unlike cryptosporidial oocysts, *C. cayetanensis* oocysts have a much thicker wall and their contents are more granular.

Treatment with TMP-SMX is considered effective in relieving symptoms. Irradiation is one proposed method of killing coccidian oocysts on fruits and vegetables. In water and food matrices, oocysts cannot sporulate if heated or frozen, therefore losing their infectivity. Oocysts are resistant to chemical treatments and pesticides commonly used by the food industry.

15.5.3 *CRYPTOSPORIDIUM* SPECIES

Cryptosporidium parvum is a coccidian protozoan parasite that represents a major cause of intestinal, parasitic disease in humans. Its oocysts are approximately 5 μm in diameter and contain four sporozoites. After ingestion of food or water contaminated with sporulated oocysts by a host, sporozoites excyst and penetrate the surface of the microvillus border of a host cell. Asexual development leads to first- and second-generation meronts. Merozoites released from second-generation meronts form male (micro) and female (macro) gamonts. After fertilization, oocysts are formed. Oocysts sporulate in the host, then pass in the feces. As many as 79 species of mammals are considered to be hosts for *C. parvum*. In addition to those *C. parvum* capable of interspecies transmission, there is now thought to be a human-specific genotype, which has been called *Cryptosporidium hominis*, and even new species (e.g., *Cryptosporidium canis*) that were once thought to be *C. parvum*.

Infection with this parasite is common but fortunately disease is rare in immunocompetent humans. Infections in immunosuppressed patients (particularly, such as those with AIDS) can be life threatening and even fatal. The most characteristic

symptom is profuse watery diarrhea, cramps, abdominal pain, vomiting, and low-grade fever. In rare cases, cryptosporidiosis of extraintestinal organs, including gall bladder, lungs, eyes, and vagina occur.

Diagnosis is made by fecal examination, including direct examination of fecal floats, staining of fecal smears using acid-fast and other stains, direct immunofluorescence or immunochromatographic tests. There is, however, no specific therapy to date; nitazoxanide [2-acetyloloxy-*N*-(5-nitro-2-thiazolyl) benzamide] is the only drug approved by the American Food and Drug Administration to treat cryptosporidiosis in immunocompetent patients. Removal of oocysts during filtration and sedimentation of municipal water is a task for engineers. Boiling of water kills all coccidian oocysts.

15.5.4 *GIARDIA DUODENALIS*

Giardia duodenalis is a flagellated protozoan parasite with bilateral symmetry. Trophozoites and cysts are the two stages in its simple fecal–oral cycle. Trophozoites are pear shaped, 10–20 μm long, and 5–15 μm wide; they contain two nuclei and eight (four lateral, two ventral, and two caudal) flagella. The dorsal surface is convex and the ventral surface is usually concave with a sucking disc at the broader end. Cysts are round to ellipsoidal (8–19 μm by 11–14 μm in size). They contain two hints of trophozoites with four nuclei and no flagella. After excystation, trophozoites attach to the intestinal epithelium by the adhesive disc, and increase in number dividing by longitudinal binary fission. Cysts and trophozoites may be passed in the feces. Although trophozoites do not survive for long periods outside the host, cysts can survive in the external environment.

The molecular taxonomy of *G. duodenalis* describes several genotypes or assemblages, among which A1, A2, and B can infect humans as well as animals, suggesting a possible zoonotic route of transmission. Other assemblages (C, D, E, F, G, and H) infect animals, but not humans.

Humans are infected by ingesting food (vegetables, strawberries, but also mussels) or water contaminated with cysts, especially in low-income countries where water is not adequately treated. It is calculated that about 280 million clinical cases are reported annually. The exact mechanism of pathogenesis is unknown because the parasite is extracellular; it probably causes an initial mechanical injury that drives an inflammatory response, which amplifies the damage. Giardiasis can cause severe intestinal disorders resulting in diarrhea, nausea, steatorrhea, and weight loss. Diagnosis is made by fecal examination. To find trophozoites, smears of freshly passed stool should be made in isotonic saline (not water). Both flotation in zinc sulfate and the Ritchie method are useful in detecting cysts in feces. The discharge of *Giardia* in the stool is not always regular; therefore multiple stool examinations may be necessary. Immunodiagnostic methods including an ELISA, or immunochromatography or a direct immunofluorescence test, to detect *Giardia* in stools are now commercially available.

Metronidazole is the drug recommended for the treatment of giardiasis; however, in some cases drug resistance has been described. Because of the potential for wild

and domestic animals to act as reservoirs for infection, good personal hygiene is necessary to prevent infection with *Giardia*. Boiling will kill *Giardia* in water.

15.5.5 SOIL-TRANSMITTED NEMATODES AND VISCERAL LARVA MIGRANS

Several nematodes can be transmitted through ingestion of contaminated soil.

The clinical picture is represented by VLM, which usually derives from the migration of nematode larvae in the accidental human host. *Toxocara cati* from cats, *Ascaris suum* from pigs, and *Ancylostoma* from dogs and cats can all cause VLM or cutaneous larva migrans, but VLM is most frequently caused in humans by *Toxocara canis* and *Baylisascaris procyonis*. The definitive hosts of these parasites are respectively dogs and other canids, and raccoons.

Adult worms live in the stomach and small intestine and after mating, female worms shed unembryonated eggs that are eliminated with the feces. First-stage larvae develop inside the egg and undergo a molt to form second-stage larvae. *Toxocara* and *Bayliascaris* eggs are sticky and survive in the environment for many years. Humans, by ingesting embryonated eggs in contaminated water or food, or by ingestion of contaminated soil, become infected. In the accidental human hosts, second-stage larvae can migrate extensively through many organs but cannot mature. Even one or a few larvae in the eye and brain can cause extensive damage, and symptoms vary depending on the organs involved. In the definitive host (dog), *T. canis* larvae return to the gut and mature into adult worms where they can lay a large number of eggs for a long period. *T. canis* is transmitted transplacentally and via milk to pups. Three-week-old pups can shed *T. canis* eggs in feces, which represent a health threat for humans, especially children. *Baylisascaris procyonis* is only transmitted fecally, and eggs in the feces deposited by raccoons transmit the infection, especially to children.

Diagnosis of VLM in humans is difficult because of variable symptoms. Eosinophilia can arouse suspicion of helminth infection. Serological tests can aid diagnosis; in the case of a positive ELISA reaction the result should be confirmed by IB. MBZ and ABZ have been used to treat VLM in humans. Dogs should be dewormed regularly to reduce environmental contamination by eggs.

15.6 SUMMARY

This chapter has reviewed information on diseases associated with foodborne (meat- and fishborne) parasites, but also parasites disseminated in fecally contaminated food and water, including their etiologic agents, life cycle, symptoms, diagnosis, treatment, prevention, and control. Parasites covered include species of the genera *Anisakis*, *Baylisascaris*, *Capillaria*, *Clonorchis*, *Cryptosporidium*, *Cyclospora*, *Diphyllobothrium*, *Fasciola*, *Giardia*, *Gnathostoma*, *Cystoisospora*, *Opisthorchis*, *Sarcocystis*, *Taenia*, *Toxocara*, *Toxoplasma*, *Trichinella*, and others.

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