Intestinal Nematodes: Biology and Control

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KEYWORDS

• *Toxocara* • *Toxascaris* • *Ancylostoma* • *Uncinaria* • *Trichuris*

A variety of nematodes occur in dogs and cats. Several nematode species inhabit the small and large intestines. Important species that live in the small intestine are roundworms of the genus *Toxocara* (*T canis, T cati*) and *Toxascaris* (ie, *T leonina*), and hookworms of the genus *Ancylostoma* (*A caninum, A braziliense, A tubaeforme*) or *Uncinaria* (*U stenocephala*). Parasites of the large intestine are nematodes of the genus *Trichuris* (ie, whipworms, *T vulpis*).

After a comprehensive description of their life cycle and biology, which are indispensable for understanding and justifying their control, current recommendations of nematode control are presented and discussed thereafter.

BIOLOGY OF INTESTINAL NEMATODES

Ascaridae

Toxocara canis

Life cycle The most frequent and important roundworm of dogs is the zoonotic parasite *Toxocara canis*. The adult stages live in the lumen of the small intestine. Eggs produced by mature female worms pass through the intestine and are deposited in the environment via feces as unembryonated and not infective eggs.

Depending on soil type and climatic conditions, such as temperature and humidity, eggs will develop to an infective stage (L3) within a period ranging from 3 weeks to several months. These embryonated and infective eggs can survive for several years under optimal conditions. After oral uptake of these stages, development continues during and after a typical blood-liver-lung migration pathway. A few hours after infection, L3 reach the liver, and pass on to the lungs where they molt to the L4 stage. These larvae penetrate the blood-air-barrier, migrate upward to the trachea, pass the larynx and pharynx, and are swallowed down the esophagus, to reach the lumen of the duodenum as immature adults or, in older descriptions, the fifth larval stage.[1,2]

Alternatively, infective larvae (L3) as somatic stages can also be transmitted via paratenic hosts or vertically between dam and puppies. When paratenic hosts ingest infective eggs, development occurs only to a resting L3 in various tissues, so that

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the L3 are protected from the environment and can wait until the host, usually prey of canids like rodents, will be eaten by the definitive host, the dog.

After ingestion of infective *Toxocara* eggs, larval development depends on the immune status of the host. Either adults form in the duodenum after tracheal migration, or in older immunocompetent animals, somatic larvae are found after passive hematogenic distribution to various peripheral organs like the musculature, kidneys, liver, and the central nervous system.

These dispersing and later resting (hypobiotic) somatic larvae—still L3—were shown to have epidemiologic importance in the pregnant dam. These stages are released and reactivated in the last third of pregnancy, when they migrate transplacentally into the fetuses' organs as vertical infection. This host-finding strategy of *Toxocara* is further enhanced by lactogenic transmission of larvae to newborn puppies. Both transmission types happen independently of whether the dam is patently infected or not. Additionally, infective larvae can infect paratenic hosts where they are stored for infection after predation of *Toxocara*-infected paratenic hosts by dogs. There, the larvae develop in most cases directly to adult worms in the intestinal tract without further migration.

Pathogenesis The larval migration through the liver leads to an increase of specific enzymes such as glutamate dehydrogenase (GLDH) and alanine aminotransferase (ALT).[3] Also, pneumonia caused by the migration of larvae in the lung is described within the first days of life. Severe infections cause signs beginning in the second week of life including ascites, anorexia, and anemia and a dilatation of the proximal duodenum is reported. On necropsy, multiple petechiae and intestinal ruptures or perforations were seen with parasites penetrating the small intestinal wall into the peritoneal cavity, followed by peritonitis or massive blood loss into the peritoneal cavity.[3–5]

Clinical signs Clinical signs are dependent on the age of the animal and on the number, location, and stage of development of the worms.[1,6,7] After birth, puppies can get acute toxocarosis from pneumonia owing to tracheal migration and die within 2 to 3 days. At an age of 2 to 3 weeks, puppies can show digestive disturbances and emaciation, caused by mature worms in the stomach and intestine. They can show diarrhea, vomiting, coughing, constipation, and nasal discharge at clinical examination. Distension of the abdomen (''potbelly'') can occur as a result of a heavy worm burden but more probably from gas formation caused by dysbacteriosis. Mortality is possible because of obstruction of the gall bladder, bile duct, and pancreatic duct and rupture of the intestine, but is rather rare in this stage.

Toxocara cati

Toxocara cati is the most common gastrointestinal helminth of the cat worldwide. It plays an important role not only by infecting young kittens but also as a zoonotic parasite that can cause human toxocarosis.[8–10] Following the oral uptake of eggs containing infective L3, these undergo a tracheal migration via the liver and lungs until they finally reach the small intestine. During and after this migration the larvae develop to the adult stage, and patency starts 8 weeks post infection. Some of the larvae reach the muscle tissue where they are encysted and retain infectivity.[11,12] Although the life cycle is very similar to that of *T canis*, a different adaptation of the host-parasite-relationship can be observed: lactogenic transmission of larvae occurs only after acute infection of the queen during late pregnancy but not during chronic natural infection. There is no evidence for the existence of arrested somatic larvae in the adult cat as an important host-finding strategy in the life cycle of *T cati*. Following milk-borne