Mouth

Protozoa
- *Tetratrichomonas felistomae* (Trichomonads)

Stomach

Nematodes
- Ollulanus tricuspis (Trichostrongyles)
- Physaloptera praepulialis (Spirurids)
- Physaloptera rara (Spirurids)
- Aschotheca putorii (Adenophorean)

Small Intestine

Protozoa
- Giardia felis (Giardia)
- Cryptosporidium felis (Coccidia)
- Isospora felis (Coccidia)
- Isospora rivolta (Coccidia)
- Toxoplasma gondii (Coccidia)
- Hammondia hammondi (Coccidia)
- Sarcocystis (Coccidia)

Nematodes
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- Toxascaris leonina (Ascarids)
- Strongyloides planiceps (Rhabditoids)
- Strongyloides felis (Rhabditoids)
- Ancylostoma tubaeforme (Hookworms)
- Ancylostoma braziliensis (Hookworms)
- Uncinaria stenocephala (Hookworms)

Trematodes
- Alaria marcianae (Alaria)

Cestodes
- Spironema species (Spirometra)
- Spirometra mansonoides (Spirometra)
- Mesocestoides lineatus (Mesocestoides)
- Dipylidium caninum
- *Taenia taeniaeformis*
- *Echinococcus multilocularis* (Echinococcus)

Large Intestine

Protozoa
- *Pentatrichomonas hominis* (Trichomonads)

Nematodes
- Strongyloides tumefaciens (Nematode)

Mouth

Protozoa - *Tetratrichomonas felistomae* (Trichomonads)

**Parasite Identification** - Trophozoites are about 10 µm long and 3 to 4 µm wide. An undulating membrane (three undulations) runs along one side of the body, and in the original description, the undulating membrane ends in a free flagellum that is about equal in length to that portion attached to the undulating membrane.

**Life Cycle** - Trichomonads move with a rapid and jerking motion caused by flagellar movement. There is no cyst stage, and transmission from cat to cat is by direct contact.

**Disease** - Gothe et al., [1] found trichomonads only in the mouths of FIV, FeLV, or FIP positive cats. The organisms were found only in cats that had gingivitis and one of these viral infections. Cats with stomatitis due to other causes and clinically normal cats did not harbor these parasites.

**Treatment** - Improved oral hygiene should reduce the possibility that trichomonads are present in the mouths of cats.

Stomach

Nematodes - *Ollulanus tricuspis* (Trichostrongyles)

**Parasite Identification** - The adult worms are small (Fig. 1). The male is 0.7 to 0.8 mm long by 0.35 mm wide and has a
The female is 0.8 to 1.0 mm long by 0.4 mm wide. The tail of the female has 3 cusps that may be accompanied by two minor ones. The vulva is in the posterior part of the body. Eggs in the female are large and few in number. The egg develops and hatches within the uterus. The first-stage larva inside the female is about one-third the length of the female worm. After further development, the female will give birth to third-stage larvae.

**Antemortem diagnosis** is difficult. Adults and larvae of *Ollulanus tricuspis* that enter the intestine are usually destroyed before being passed in the feces. Thus, only rarely is a diagnosis made by direct fecal smears or fecal flotation methods [2]. Hasslinger [3] recommends diagnosis by induction of vomiting and stomach irrigation. Examination of induced vomitus can be successful in making a diagnosis in about 70% of infected cats [2]. The saline collected from stomach irrigation should be examined after centrifugation or after larvae are collected using a Baermann apparatus.

At necropsy, a diagnosis can be made from washings or scrapings of the stomach mucosa or by peptic digestion of the stomach wall [3]. To aid in the examination of scrapings, KOH may be added to aid in the dissolution of the mucosal portion of the scraping. For digestion, the opened and everted stomach is pinned to a cork placed on the top of a funnel containing digestion fluid and maintained at 37°C for 4 to 8 hours. Then the clamp on the bottom of the funnel is opened and the collected sediment examined.

**Life Cycle** - Transmission from cat to cat is through the consumption of vomitus. Third-stage larvae are found in the adult female and free within the lumen of the cat’s stomach (Fig. 2) [4]. Larvae within the female are 0.4 mm long, and those in the stomach are 0.5 mm long. Fourth-stage larvae are found on the surface of the gastric mucosa, and are around 0.65 mm long. Wittmann [5], like Cameron, failed to produce infections in mice fed third or fourth-stage larvae or adults. Wittmann [5] transmitted the infection between cats by the feeding of the adult males and females, fourth-stage larvae, or third-stage larvae and showed that the parasites survive in the vomitus for up to 12 days. Thus, stages passed in the vomitus can infect another host. After the feeding of third-stage larvae, the prepatent period is from 33 to 37 days.

**Disease** - Cats with *Ollulanus tricuspis* generally have a history of chronic vomiting [8,9]. A Persian cat died due to chronic gastritis caused by *Ollulanus tricuspis* [10]. The three-year old cat had been imported from England about a year before. The cat lived with ten other pets. The cat stopped eating, continued to drink milk, and prior to death was dehydrated with a subnormal temperature. Three cases of chronic infections in cats resulted in two of the cats being euthanized due to their wasted condition and chronic vomiting; the third cat died shortly after the appearance of blood in its vomit and feces [11]. The stomachs of these cats had signs of chronic inflammation very similar to that reported by Hanichen and Hasslinger [10]. The infection can cause chronic gastritis in infected cats. Cameron [8] felt that the worms caused only moderate local erosion of the mucosa and increased mucus secretion. The pathological changes can be more severe, with hyperplasia, inflammation, cellular infiltration, and sclerosis of the of the stomach epithelium [10]. The worms live under a layer of mucus or partially embedded in the gastric glands [11]. The infection causes significant increases in mucosal fibrous tissue and mucosal lymphoid aggregates that have large germinal centers [12].

**Treatment** - Tetramisole (a 2.5% formulation administered at 5 mg/kg body weight) is efficacious and without side effects [3].
Physaloptera praeputialis (Spirurids)

Parasite Identification - These pink worms have a cuticular preputial-like sheath that covers the posterior end of the body of both sexes (Fig. 3). The males are 1 - 4.5 cm long. Females are 1.5 - 6 cm long with the vulva slightly anterior to midbody. The larvated eggs, 45 to 58 µm long and 30 to 42 µm wide, have a thick, clear shell (Fig. 4 and Fig. 5). The eggs are so clear that they are easy to miss in sugar flotations. The Physaloptera spp. described from cats fall into two groups. Physaloptera praeputialis, Physaloptera pseudopraeputialis, and Physaloptera brevispiculum (not actually reported from the domestic cat) have preputial-like sheaths. Physaloptera rara and Physaloptera pacitae are without sheaths. Physaloptera praeputialis and Physaloptera rara are the species commonly reported from cats.

Life Cycle - An arthropod (German cockroaches (Blatella germanica), camel crickets, (Ceutophilus spp.), and field crickets) are a required part of the life cycle of Physaloptera praeputialis [13]. The insects are infected by eating the eggs in cat feces. In crickets (Acheta assimilis), infective larvae are present in the body cavities by about 3 weeks after egg ingestion [14,15]. In cats fed infective larvae, the third and fourth molts occur 45 and 95 days after infection, respectively. After ingestion of insects containing larvae, the prepatent period is 131 - 156 days. Cats can also be infected by the ingestion of paratenic hosts. Cats fed larvae from naturally infected lizards (Varanus griseus) and the long-eared hedgehog (Hemiechinus aratus) developed patent infections 60 days after being fed the third-stage larvae [16]. Cats would be more likely to be infected by the ingestion of these paratenic hosts than by the ingestion insects.

Disease - A typical presentation of infection with Physaloptera praeputialis is vomiting with vomitus containing one or more worms. Two infected cats with anemia, eosinophilia, and melena had intermittent vomiting of several months duration with adult worms detected in the vomitus [17]. In 6 of 32 cats in Monterrey, Mexico that were infected with this parasite, necropsies revealed moderate catarrhal gastritis and multiple pseudogranulomas; these six cats were not noted to have signs of gastritis before being euthanized [18].

Treatment - Ivermectin (200 µg per kg bodyweight) will remove the clinical signs of Physaloptera praeputialis infection [17]. Smith found that two of 44 cats that were infected with a Physaloptera spp. prior to treatment expelled their worms within 24 hours after treatment with levamisole solution injected subcutaneously at a dosage of 8 mg/kg body weight [19]. About 5% of the treated cats had side effects of vomiting and salivation for one to two hours after treatment with the levamisole.

Physaloptera rara (Spirurids)

Parasite Identification - The difference between Physaloptera rara and Physaloptera praeputialis in that there is no sheath
over the posterior portion of the body of the males and females. The males are 2.5 to 3 cm long; the length of the females is 3 cm to 6 cm. The color of these worms tends to be white. The male has pedunculate papillae and caudal alae on its tail. The female’s vulva is anterior to the middle of the body. The eggs are thick shelled, 42 μm to 53 μm long, 29 μm to 35 μm wide, ellipsoid and contain a larva when passed in the feces. Like the eggs of Physaloptera rara, the eggs of Physaloptera preaugutialis are quite clear and difficult to see, especially in sugar flotations.

**Life Cycle** - The German cockroach, Blatella germanica, and grain beetles, Tribolium confusum, can serve as intermediate hosts of Physaloptera rara [20]. Petri and Ameel [14] added crickets, Acheta assimilis and ground beetles, Harpalus sp., to the list of intermediate hosts. Grasshoppers, Melanoplus femurrubrum, were added to the list of intermediate hosts by Olsen [21]. Widmer found Physaloptera larvae attached to the mucosa of the stomachs of rattlesnakes in Colorado, and he used these larvae to experimentally infect cats [22,23]. Olsen infected cats with larvae from rattlesnakes and found the prepatent period to be 75 to 79 days [21]. Olsen also showed that frogs and mice could serve as paratenic hosts, with the larvae persisting attached to the mucosa of the gastro-intestinal tract. A cat developed a patent infection 156 days after infection when larvae recovered from frog feces 21 days after infection were used.

**Disease** - Clinical signs due to this parasite are rare. Santen et al. [24] report on signs including vomiting and diarrhea in a seven-month-old cat that was infected with Toxocara cati and Physaloptera rara.

**Treatment** - Santen et al. [24], who treated an infected cat with pyrantel pamoate (5 mg pyrantel pamoate per kg body weight), provided the only description of treatment in the cat. Examination of the feces of the cat six weeks after treatment revealed the continued presence of eggs of Toxocara cati and Physaloptera rara in the feces. Two oral doses of 5 mg pyrantel pamoate per kg body weight were then used to treat the cat three weeks apart. On repeat fecal examinations, no additional eggs in the feces were revealed.

**Aonchotheca putorii (Adenophorean)**

**Parasite Identification** - The male Aonchotheca putorii range in size from 2.5 to 5.3 mm (average 4 mm); the females range in size from 3.5 to 7.4 mm (average 5 mm). The capillarids recovered from cats in New Zealand were approximately 8 mm long [25]. Body width at the posterior end of the stichosome is 24 to 38 μm (mean 33). Maximum body width ranges from 31 to 41 μm (average 33) and 34 to 51 μm (average 41), for males and females, respectively. The distance from the vulva to the anterior end is 2.01 to 3.18 mm (average 2.5). The vulva of Aonchotheca putorii may or may not be covered by a cuticular flap; the vulva is surrounded by varying amounts of corrugation. The males demonstrate a characteristic structure of the tip of the spicule and a distinct form of the lateral and caudal alae. The single spicule in the male ranges in length from 162 to 276 μm with an average of 211 μm [26]. Males of this genus lack spines on the cirrus, the sheathe surrounding the spicule. The eggs of this capillarid range from 57 to 66 μm (average 61 μm) in length and 21 to 28 μm (average 23 μm) in width. The eggs have a dark shell with thickened lengthwise ridges on their surfaces (Fig. 6).

![Figure 6. Aonchotheca putorii. Egg showing surface with the dark striae typical of this eggshell. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

**Life Cycle** - Cats in New Zealand are thought to become infected by eating infective eggs. The eggs are believed to be in soil contaminated by hedgehog feces because these capillarids reach maturity in the stomachs of cats and dogs as well as in hedgehogs [27].

**Disease** - There are few reports on the clinical signs of infection in cats. Physical examination of a domestic cat with a 3-week history of partial anorexia and intermittent bloody vomitus and tarry feces revealed lethargy, dehydration, pale mucus membranes and signs of pain upon palpation of the cranial abdomen [28]. Multiple fecal examinations revealed no helminth eggs. A complete blood cell count revealed a normocytic, normochromic anemia, and serum biochemistry tests revealed hyperglycemia, hypocalcemia, and hypokalemia. Contrast radiography revealed delayed gastric emptying, and an exploratory laparotomy revealed a 0.5 cm diameter perforation in the caudal aspect of the pylorus. There were adhesions between the pylorus and the surrounding structures. A gastroduodenostomy was performed, and histology of the resected tissue revealed adult Aonchotheca putorii and eggs in the pyloric mucosa near the area of perforation. Histological examination of the gastric tissue showed chronic, hyperplastic pyloric gastritis with dilation of numerous pyloric glands, regions of superficial mucosal fibrosis, and a perforated ulcer in the caudal aspect of the pylorus. The worms were surrounded by mucus containing many red blood cells and a few neutrophils. Some parasites were in the lamina propria of the mucosa. Smaller worm, hypothesized to be larvae, were noted in the deeper layers of the mucosa, within glandular lumens, and between the basement membrane and the epithelial cells of the glands. Cellular necrosis and regeneration surrounded the worms. Eggs were observed in the
pyloric mucus and in the lumens of the pyloric glands.

Treatment - Levamisole (2 doses: 7.5 mg/kg at 2-week intervals, the first dose split 12 hours apart and the second dose given singly) treatment of an infected cat caused the eggs of *Aonchotheca putorii* disappeared from the feces [26]. Curtsinger et al. [28] administered ivermectin (300mg/kg BW) per os one week following surgery and again two weeks later. Seven months later, the cat had a normal appetite, normal feces, and a PCVC of 42%.

Small Intestine Protozoa -

*Giardia felis* (Giardia)

Parasite Identification - Within cats, *Giardia* exists as a binucleate, flagellate trophozoite (Fig. 7). The trophozoite is small (10.5 to 17.5 µm in length by 5.25 to 8.75 µm in maximum width), and has eight trailing flagella [29]. The two large nuclei are just posterior to midbody and these nuclei are about 3 µm long and 1.5 µm wide. About one-third to one-half of the anterior surface is occupied by a sucking disc borne on the ventral surface of the trophozoite. The trailing flagella propel the organism and also create a vacuum for the sucking disc by pumping fluid out from under it. Movement of the trophozoite is along the surface of the intestinal epithelium.

Figure 7. *Giardia* sp. Cultured trophozoites showing the typical tennis-racket shape and attenuated posterior end. - To view this image in full size go to the IVIS website at www.ivis.org . -

The cyst stage occurs in the large bowel as the trophozoite prepares to enter the external environment. Cysts are about 7.4 µm wide and 10.5 µm long, and have a width-to-length ratio of about 4:1 (Fig. 8). Trophozoites produce the resistant cyst wall as they pass from the small to the large intestine in response to an undefined stimulus. The trophozoite then divides, which results in two trophozoites within the mature cyst. The cyst stage is expelled in the feces.

Figure 8. *Giardia* sp. Cyst showing the ovoid shape in which two nuclei are visible. This is a phase microscope image which makes the nuclei more obvious than with a typical light microscope. - To view this image in full size go to the IVIS website at www.ivis.org . -

If a cat has diarrhea and an accompanying infection with *Giardia*, a confirmatory diagnosis will often require a direct saline examination of a small quantity of fresh feces. This examination will allow the identification of the trophozoite stage. As only trophozoites are found in stools of this type examination of diarrheic feces for cysts is often inconclusive. In human medicine, fixed fecal smears are often prepared and stained using a trichrome staining method or iron hematoxylin for the examination of protozoa. The many kinds of protozoa that must be differentiated are the major reason for the examination of fixed smears of human feces. It is typically not necessary to perform such procedures in cats, as few fecal protozoans are present. However fixed fecal samples do have the advantage of producing a permanent slide that can be examined at a later date.

The detection of *Giardia* in fecal smears using fluorescein-conjugated antibodies to *Giardia*, and subsequent examination under a fluorescence microscope is another method now used routinely. This method increases the probability of finding the trophozoites and cysts, because they will fluoresce when examined, but the equipment is costly, and other procedures are still be required to concentrate the organisms in many samples.

The detection of *Giardia* antigens within fecal samples is a method that eliminates some of these problems. Antigen detection tests developed for use with human feces are capable of detecting infections in diarrheic fecal samples in which, due to the degeneration of non-viable trophozoites, there may be no detectable organisms. Such tests work by detecting antigens of the *Giardia* organism that are passed in the feces [30]. These tests, such as ProSpecT®/Giardia™ (a product of Alexon Inc., Mountain View, CA), have not yet been approved for use in cats, but there are several indications that they are likely to work very well. Unlike the duodenal aspirate method used for examination for *giardiasis* in dogs, cats should not be examined for giardiasis using this method, because the organisms may live further posterior in the intestine in the cat than they do in the dog.

Centrifugal flotation procedure can usually be used to detect cysts and the zinc-sulfate method is preferred. As cyst excretion has been shown to be sporadic by [31], it may be best to examine the samples by both flotation and antigen detection.
Although the cysts can be observed in sugar flotations of fecal matter, the cysts will rapidly collapse. Collapsed cysts look like small crescent moons rather than the ovoid forms that are observed in zinc-sulfate preparations or direct smears soon after they are made. Cysts can also be detected by antigen detection assays.

**Strains, Phenotypes and Genotypes** - Various methods have been used to show that there are potentially different groups of *Giardia*. *Giardia* isolates from humans, cattle, dogs, sheep, cats, and guinea pigs have been compared using isozymes, [32-37]. Overall, this work appears to be somewhat contradictory with some groups finding the different isolates to be very similar to each other, while other groups seem to find a good deal of heterogeneity between isolates. It would appear, however, that the two major types that are found in humans also are occasionally found in cats and dogs, and some work has suggested that cats are a major source. Other work has found that the canine form is sometimes difficult to obtain in culture, and that dogs may be hosts to the two major types found in humans, as well as, a third form in Western Australia that might be restricted to canine hosts [38]. It was found that the isolates fell into two groups when restriction endonuclease analysis of DNA from 15 *Giardia* isolates from humans, beaver, a cat, and a guinea pig used [39]. The cat and one of the two beaver isolates were similar to the pattern produced by most of the human isolates, but the other beaver isolates and guinea pig differed from the majority of the human isolates as did some of the other human isolates. Using DNA polymorphisms has not cleared up the continuing separation between groups although it appears that groups I and II appear to hold the majority of the genotypes examined so far [40,41]. Examination of the ribosomal RNA sequencing of these genotypes also supported the infection of Western Australian dogs with certain isotypes that appear to be restricted to canine hosts [42].

**Life Cycle** - The cyst is passed in the feces of the host and a new host is infected by direct fecal-oral contamination. Cysts may also be transmitted to a next host through contaminated drinking water. On ingestion of a cyst by a host, the trophozoites leave the cyst within the small intestine and take up residence on the intestinal mucosa. Repeated division of the trophozoites occurs until they have populated the intestines of the new host. Some trophozoites are periodically carried in the fecal stream towards the anus, encysting on the way. A cat is capable of excreting thousands of cysts in the environment within five to sixteen days after the cat has ingested a cyst.

**Disease** - In cats, clinical signs from infections with *Giardia felis* can and do develop. Diarrhea is the typical sign [43]. Cats may also undergo weight loss, and kittens may fail to gain weight. Maintenance of normal appetites and clinical values are usually observed in cats with signs of diarrhea. Periods of vomiting are not usually a symptom in cats infected with *Giardia*. The diarrhea that is observed tends to be due to problems with malabsorption and steatorrhea, and for this reason, the feces tends to have increased levels of neutral fats, and be soft and pale in color. It has been shown that when experimentally infected lambs were compared to uninfected controls, infection with *Giardia lamblia* was associated with decreased weight gain and feed efficiency [44].

**Treatment** - Activity against *Giardia* infections by several formulations of benzimidazoles has been shown [43]. Albendazole cleared eighteen of the twenty dogs that had been shedding cysts at the beginning of therapy. In cats, a similar treatment (25 mg/kg body weight orally twice daily for two days) did not work. However, increasing the number of treatments (25 mg/kg body weight orally twice daily for five days) successfully cleared five cats of the cysts they were shedding in their feces. This compound is currently not approved for use in dogs or cats, although it is used as an anthelminthic in cattle. Further, albendazole has been shown to be associated with bone marrow aplasia in one cat when used to treat *Giardia* infection [45]. Fenbendazole has been shown to prevent beagles from shedding cysts in their feces at the dosage routinely applied for anthelminthic therapy (50 mg/kg body weight orally once a day for three days) [46]. This compound is an approved and routinely used anthelminthic in dogs, but is not approved for use in cats. Fenbendazole has been administered to cats as an anthelminthic with no apparent detrimental effects [47]. Febantel is approved for use in cats, but has not been examined for its efficacy in treating giardiasis in this species, although a compound containing febantel has been found to be effective against *Giardia* in dogs [48].

Other drugs used to treat *Giardia* infections in cats have included metronidazole, quinacrine, and furazolidone [49]. Oral metronidazole at 10 or 25 mg/ kg body weight given twice a day for 5 days was observed to cure cats of their infections with *Giardia*. Quinacrine was given orally at 2.3 mg/kg body weight once a day for 12 days to 5 cats, but four continued to pass cysts. Furazolidone given orally twice a day at 4 mg/kg body weight has also been shown to be effective.

**Cryptosporidium felis** (*Coccidia*)

**Parasite Identification** - The oocysts of *Cryptosporidium felis* can be distinguished from those of *Cryptosporidium parvum* in that they are smaller. The diameter of oocysts of *Cryptosporidium felis* measures 4.3 μm (3.5 to 5.0 μm). The mean diameter of those of *Cryptosporidium parvum* tends to measure 5.0 μm.

**Life Cycle** - In cats, ingestion of an oocyst causes infection. Four sporozoites are contained in each oocyst. When various aspects of the new host are stimulated, the sporozoites excyst from the oocyst and penetrate cells of the mucosa. Like other coccidians, the sporozoites induce phagocytosis; however, unlike with other coccidians, the small sporozoites appears to remain on the surface of the cell, that is, the cell membrane bulges out around the small parasite. A highly convoluted membrane like structure that is called the "feeding organelle" or "apical organelle" develops between the host cell and the...
Two sporocysts are contained in each sporulated oocyst. Sporozoites are 10 - 15 µm and contain a single nucleus and a refractile globule. These then proceed to infect other cells. The next phase of the infection is the development of sexual stages, microgametocytes and macrogametocytes. Although the microgametes are aflagellar, they are capable of movement and they will fuse with a macrogamete. The macrogamete deposits an oocyst wall after fusion, to become an oocyst. The oocyst undergoes a process of sporulation to produce oocysts that contain four infective sporozoites while still within the host. Iseki [50] described that within the intestinal material he examined, these sporozoites were sometimes seen to be undergoing spontaneous excystation and he felt that autoinfection was a distinct possibility. The prepatent period was 5 to 6 days in experimentally infected cats, and the patent period was 7 to 10 days.

**Potential Transmission** - The potential transmission of *Cryptosporidium* from cats to people is unknown. There have been reports that link feline cryptosporidiosis to human infection [51,52]. At the same time it would seem that the infections are acquired from other human as many of the human isolates are neither from cats nor cattle. There have also been studies showing that for HIV-infected individuals pet ownership is not a risk factor for cryptosporidiosis [53].

**Disease** - There have been no reports of *Cryptosporidium felis* causing disease in cats, but it is very unclear as to whether cats are routinely infected with this species or with isolates of *Cryptosporidium parvum*. Asahi et al. [54] showed that experimentally infected cats shed oocysts for up to three to five months. Three of these cats were held for a year, and then prednisolone inclusions were initiated. These cats again shed large numbers of oocysts in their feces after about a week of prednisolone treatment. Even though these cats shed large numbers of oocysts, none of the cats developed significant diarrhea or weight loss during the infections.

On occasion cats have been experimentally infected with *Cryptosporidium* isolated from calves and considered to be *Cryptosporidium parvum* [55,56], but cats seem rather refractory to such infections. We performed a trial at Cornell University where two virus-free kittens were each fed 10 million oocysts, and observed that only a very few oocysts were shed in the feces of these cats, and they never developed signs of infection.

No matter whether the species *Cryptosporidium felis* or *Cryptosporidium parvum* is involved, cats do sometimes present with severe disease due to cryptosporidiosis. A cat that has an underlying immunosuppressive disorder such as a feline leukemia virus infection is a typical presentation [57]. There are cases, however, where cats develop severe disease and persistent cryptosporidiosis where there is no apparent underlying condition [58]. Also, the recent development of serological tests that detect antibody in the blood of cats that have been infected would suggest that throughout the United States somewhere around 15% of cats have been or are currently infected with *Cryptosporidium* [58,59].

Recurring bouts of diarrhea will occur in the cat that presents with cryptosporidiosis. A water-losing diarrhea caused by the development of the parasites within the epithelial cells of the mucosa is the disease caused by *Cryptosporidium* infection. Histologically, infection causes a blunting of the villus of the intestine and crypt hyperplasia that is accompanied by an intense neutrophilic response [60]. It has been found that net water, sodium, and chloride movement in AIDS patients with cryptosporidiosis was the same as that in healthy controls [61]. These authors concluded from this work, that the diarrhea may be due to the secretion of electrolytes and water efflux distally to the site of infection or as to some yet undefined feature or the infection. It has been shown using monolayers of polarized colonic epithelial cells and the experimental infection of these cells with *Cryptosporidium parvum* that there is an increased macromolecular permeability of the monolayer, and it was felt that disruption of the epithelial cell barrier played a role in the observed diarrhea [62]. Additional work using the cell monolayer system has shown rather conclusively that infection of the epithelial cells will ultimately result in significant changes in the host cell permeability, the entire monolayer permeability, and ultimately. the death of the infected cells [63].

**Treatment** - Cases known to be caused by *Cryptosporidium felis* have not been treated, and no attempts have been made to treat cats experimentally infected with this species. Treatment of cryptosporidiosis in cats is as difficult as treatment is in humans. The relief of symptoms and increased fluids is the basic therapy. Paromomycin has been used to treat cats with some success [64]; but this therapy has potential complications that can include renal failure [65].

**Isospora felis (Coccidia)**

*Parasite Identification* - Oocysts measure 38 - 51 by 27 - 39 µm with a mean of 41.6 by 30.5 µm. The length width ratio is 1.3 - 1.4 with a mean of 1.35. *Isospora felis* oocysts are the largest of the coccidial oocysts observed in cats (Fig. 9) (Table 1). Two sporocysts are contained in each sporulated oocyst. Sporozoites are 10 - 15 µm in length, lie lengthwise in the sporocyst, and contain a single nucleus and a refractile globule.
Life Cycle - Several authors have described portions of the life cycle of *I. felis* [66-71]. Sporozoites excyst in the small intestine, and developmental stages are located in enterocytes of the distal portions of the villi in the ileum, and rarely of the duodenum and jejunum. The first developmental cycle is probably by endodyogeny. Mature first-generation meronts with 16 - 17 merozoites were first observed 4 days PI. Mature second-generation meronts with about 10 merozoites were first observed 5 days PI. Third-generation meronts with 36 to 70 merozoites were first observed 6 days PI, and were in the same host cell as the second-generation meronts. Sexual stages were observed 6 days PI, and oocysts were first observed 7 days PI. The prepatent period is 7 - 11 days. The patent period is 10 - 11 days. Oocysts are excreted unsporulated, and, sporulate in 40 hr at 20ºC, 24 hr at 25ºC, 12 hr at 30ºC and 8 hr at 38ºC [72]. Sporulation does not occur above 45ºC. Mice (*Mus musculus*), Norway rats (*Rattus norvegicus*), golden hamsters (*Mesocricetus auratus*), cows (*Bos taurus*), and dogs (*Canis familiaris*) will maintain sporozoites within their tissues and can serve as paratenic hosts [73,75-77]. The sporozoites present in the tissues of these paratenic hosts will infect to cats if they are ingested.

Disease - *I. felis* appears moderately pathogenic for 6 week old to 13 week old kittens given 1 to 1.5 x 10^5 oocysts. Soft, mucoid, feces are observed in kittens 8 days after infection but severe disease did not occur. Microscopic lesions in kittens examined about 6 days after being given oocysts are mild and consist of erosion of the superficial epithelial cells. In kittens examined 7 to 9 days after infection; congestion, mild neutrophilic infiltration, and hypersecretion of the mucosa are observed [68]. Epithelial hyperplasia was also noted in some kittens. *Isospora felis* is more pathogenic for younger kittens. Four week old kittens may develop severe enteritis, emaciation, and may die if given 1 x 10^6 oocysts [79].

Treatment - Coccidiosis in cats can be treated with various sulfonamides and quinacrine.

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<thead>
<tr>
<th>Species</th>
<th>Length (mean)</th>
<th>Width (mean)</th>
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<tbody>
<tr>
<td><em>I. felis</em></td>
<td>38 to 51</td>
<td>27 to 39</td>
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<tr>
<td><em>I. rivolta</em></td>
<td>18 to 28</td>
<td>16 to 23</td>
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<tr>
<td><em>T. gondii</em></td>
<td>11 to 13</td>
<td>11 to 13</td>
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<tr>
<td><em>H. hammondi</em></td>
<td>10 to 13</td>
<td>10 to 13</td>
</tr>
<tr>
<td><em>B. darlingi</em></td>
<td>11 to 13</td>
<td>11 to 13</td>
</tr>
<tr>
<td><em>B. wallacei</em></td>
<td>16 to 19 (17)</td>
<td>10 to 13 (11)</td>
</tr>
<tr>
<td><em>C. parvum</em></td>
<td>4 to 5</td>
<td>4 to 5</td>
</tr>
<tr>
<td><em>Sarcocystis ssp.</em></td>
<td>11 to 14</td>
<td>7 to 9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Antiprotozoal Agent</th>
<th>Treatment Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfadimethoxine (SDM)</td>
<td>50 mg/kg for 10 days or 55 mg/kg for 1 day and 27.5 mg/kg until signs disappear</td>
</tr>
<tr>
<td>SDM plus Ormetoprim (OM)</td>
<td>55 mg/kg SDM plus 11 mg/kg OM for up to 23 days</td>
</tr>
<tr>
<td>Sulfaguanidine</td>
<td>150 to 200 mg/kg for 5 days</td>
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</table>
**Isospora rivolta** (Coccidia)

**Parasite Identification** - Sporulated oocysts measure 23 - 29 by 20 - 26 μm with a mean of 25.4 by 23.4 μm (Fig. 10). The ratio of length to width is 1.08. The mid-range of coccidial oocysts that are passed in the feces of cats are represented by the oocysts of *Isospora rivolta* (Table 1).

**Life Cycle** - In experimentally infected kittens three types of meronts that differ structurally were observed [80]. Type I meronts with 8 merozoites were observed first 0.5 days PI and divided by endodyogeny. Type II meronts were observed first 2 days PI, and produced an undetermined number of merozoites. Type III meronts with 2 to 30 merozoites were first observed 3 days PI. Sexual stages and oocysts were first observed 5 days PI. The prepatent period is 4 to 7 days. The patent period is 2 weeks or greater. At 24°C sporulation occurs within 24 hr, at 30°C within 12 hr, and at 37°C within 8 hr. Mice (*Mus musculus*), golden hamsters (*Mesocricetus auratus*), Norway rats (*Rattus norvegicus*), cows (*Bos taurus*), and opossums (*Didelphis virginiana*) have been found to serve as paratenic hosts in the life cycle of *Isospora rivolta* [75]. In kittens fed mouse tissues containing *I. rivolta* stages, the developmental cycle was delayed 0.5 to 2 days in the appearance of the different stages [81].

**Disease** - Experimental studies indicate that for newborn but not weaned kittens *I. rivolta* is pathogenic [80]. Three to 4 days after inoculation of 1 x 10^4 to 1 x 10^6 oocysts to newborn kittens, diarrhea occurs. In these kittens microscopic lesions consisting of congestion, erosion, villous atrophy, and cryptitis occurred. Deaths did not occur. Clinical signs were absent in 10 to 13 week old kittens given 1 x 10^6 oocysts.

**Toxoplasma gondii** (Coccidia)

**Parasite Identification** - Unsporulated *T. gondii* oocysts are spherical to subspherical measure 11 to 13 μm in diameter, and...

<table>
<thead>
<tr>
<th>Antiprotozoal Agent</th>
<th>Treatment Regimen</th>
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<tbody>
<tr>
<td><strong>Sulfadiazine (SD) &amp; Trimethoprim (TRI)</strong></td>
<td>25 to 50 mg/kg SD plus 5 to 10 mg/kg TRI for 6 days for cats over 4 kg 12.5 to 25 mg/kg SD plus 2.5 to 5 mg/kg TRI for 6 days for cats over 4 kg</td>
</tr>
<tr>
<td><strong>Amprolium HCl (AMP)</strong></td>
<td>300 to 400 mg/kg for 5 days 110 to 220 mg/kg for 7 to 12 days 20 to 40 mg/kg for 10 days</td>
</tr>
<tr>
<td><strong>AMP plus SDM</strong></td>
<td>150 mg/kg AMP plus 25 mg/kg SDM for 14 days</td>
</tr>
<tr>
<td><strong>Quinacrine</strong></td>
<td>10 mg/kg for 5 days</td>
</tr>
<tr>
<td><strong>Furazolidone</strong></td>
<td>8 to 20 mg/kg BWT once or twice daily</td>
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**Cryptosporidiosis**

<table>
<thead>
<tr>
<th>Antiprotozoal Agent</th>
<th>Treatment Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Paromomycin</strong></td>
<td>165 mg/kg every 12 hour for 5 days</td>
</tr>
</tbody>
</table>
contain a single mass called a sporont (Fig. 11 and Fig. 12). Sporulated *T. gondii* oocysts are ellipsoidal to subspherical, and each contains 2 ellipsoidal sporocysts that enclose 4 sporozoites.

**Figure 11.** *Toxoplasma gondii*. Three oocysts in a direct smear of a cat fecal (oocysts indicated by arrows). - To view this image in full size go to the IVIS website at www.ivis.org.

**Figure 12.** *Toxoplasma gondii*. Unsporulated oocyst. - To view this image in full size go to the IVIS website at www.ivis.org.

**Life Cycle** - Based on the developmental stage that the cat ingests, the life cycle of *T. gondii* varies [82-85]. Bradyzoites are the infective stage when cats ingest tissue cysts. Some of the released bradyzoites will penetrate enterocytes and begin the enteroepithelial cycle that will terminate in oocyst production [84]. Some bradyzoites, however, will penetrate into the intestinal lamina propria and begin development as tachyzoites. As early as 8 hours after tissue cysts are ingested, infectious stages of *T. gondii* are present in the liver and mesenteric lymph nodes and chronic infections are produced by these stages. Prior to the formation of sexual stages at 3 - 4 days after infection, five structurally distinct types of schizonts are produced in the enterocytes of the small and large intestine [82,84]. For tissue-cyst-induced infections, the prepatent period is 3 to 10 days. Oocysts are excreted in the feces for 7 to > 20 days, with most being excreted between days 5 and 8. Oocyst excreting infections are a result of the ingestion of sporulated *T. gondii* oocysts or tachyzoites in only 16 to 20% of cats as compared with 97% of cats that fed tissue cysts [83,85,86]. In oocyst infected cats, the prepatent period is greater, 18 days or more in these cats as compared to 3 to 10 days in cats that are fed tissue cysts [86]. The reason for the extended prepatent period is that the tachyzoites or sporozoites must first produce tissue cysts that contain bradyzoites. Then, these bradyzoites will find their way back to the intestine to produce the enteroepithelial cycle that results in oocyst production. Lactogenically or transplacentally infected kittens will excrete oocysts but the prepatent period is usually 3 weeks or more because the kittens are infected with tachyzoites [87]. The greatest numbers of *T. gondii* oocysts are produced by domestic cats under 1 year of age. Cats born and raised outdoors are usually infected with *T. gondii* shortly after they are weaned. If *Toxoplasma gondii* naive adult domestic cats are fed tissue cysts they will excrete oocysts but they usually will excrete fewer numbers of oocysts and excrete oocysts for a shorter period of time than recently weaned kittens.

Sporulation of oocyst is dependent on temperature and moisture [88]. Sporulation is asynchronous and sporulation of some oocysts occurs before others. Completely infectious oocysts are present at 25ºC (room temperature) within 24hr; at 15ºC in 5 days, and at 11ºC in 21 days [89]. Unsporulated oocysts do not survive freezing but can remain viable for several months at 4ºC and become infectious if placed under the appropriate conditions. Unsporulated oocysts die if kept at 37ºC for 24 hours and are killed if exposed to 50ºC for 10 minutes. Sporulated oocysts are more resistant to chemical and environmental stresses than are unsporulated oocysts. Viable oocysts of *T. gondii* have been isolated from soil samples [90-92] and experimentally they can survive in the soil for over 18 months [93]. Sporulated oocysts can not survive freezing or temperatures of 55ºC or higher [94,95]. Sporulated oocysts survive at 4ºC for several years in liquid medium [95]. In cats that have excreted oocysts, intestinal immunity to *T. gondii* is strong [96-98]. Primary *T. gondii* infection does not cause immunosuppression [99,100]. A significant role in resistance to intestinal infection is not played by serum antibody and intestinal immunity is most likely cell mediated.

Oocysts begin to be excreted in the feces before IgG, IgM or IgA antibodies are present in the serum [101-103]. Enteroepithelial stages partially develop in the intestines of immune cats but oocyst production is prevented [100]. Most cats that have excreted oocysts once and are challenged within 6 months to 1 year do not re-excrete oocysts. In about 55% of cats intestinal immunity will last up to 6 years [98].

Immunosuppression with high doses of corticosteroid (10 to 80 prednisone orally daily or 10 to 80 mg/kg methylprednisolone acetate IM weekly) will cause some chronically infected cats to re-excrete oocysts [104]. Clinically relevant doses of 5 to 20 mg/kg corticosteroid given weekly for 4 weeks did not cause recently or chronically infected cats to re-excrete *T. gondii* oocysts [261]. Oocyst excretion in chronically infected cats will not be caused by doses of 5 mg/kg cortisone acetate for 7 days [105].

Cats that undergo a primary feline immunodeficiency virus infection when they are chronically infected with *T. gondii* demonstrate an increase in *T. gondii* antibody titers suggesting some reactivation of encysted stages. Experimental studies,
However, indicate that there is no reactivation of *T. gondii* oocyst excretion or development of clinical toxoplasmosis [106-110]. In FIV positive cats, clinical disease has rarely been associated with re-activated toxoplasmosis. Cats do not appear to be predisposed to acute toxoplasmosis by prior experimental FeLV infection, and there is no apparent effect on oocyst excretion [111].

There exists an interesting relationship in cats between the intestinal coccidium *Isospora felis* and *T. gondii* [112,113]. Cats that have previously recovered from a *T. gondii* infection will re-excrete *T. gondii* oocysts if they then obtain a primary *I. felis* infection. Cats acquiring a primary *T. gondii* infection following a primary *I. felis* infection develop strong immunity to *T. gondii* and will not re-excrete *T. gondii* oocysts if challenged with *I. felis* [114].

**Disease** - There is little to no disease associated with enteric toxoplasmosis in cats. Occasionally kittens shedding oocysts may have mild diarrhea but often the infection occurs without signs.

**Treatment** - Once oocysts appear in the feces, most have already been formed within the enterocystes. Thus, treatment will typically not stop oocyst shedding.

### Hammondia hammondi (Coccidia)

**Parasite Identification** - *H. hammondi* oocysts are excreted unsporulated in the feces. The oocysts are colorless, subspherical to spherical, and measure 10.5 - 12.5 by 11.2 - 13.2 µm with a mean of 10.6 by 11.4 µm. Two ellipsoid sporocysts each with 4 sporozoites are contained in the sporulated oocysts. Sporocysts measure 6.0 - 7.5 by 8.0 - 10.7 µm with a mean of 6.5 by 9.8 µm, lack a Stieda body, and contain a sporocyst residuum composed of dispersed or compact granules. The sporozoites are curved and elongate within the sporocyst had a crystalloid body. Sporulation occurs at room temperature (22 to 26º C) in 2 to 3 days.

Diagnosis based on oocyst structure alone is impossible because of the similarity in oocyst size and structure between *T. gondii* and *H. hammondi*. For a definitive diagnosis to be obtained animal inoculation is needed. Identification is also difficult of *H. hammondi* in tissues. The tachyzoites of *H. hammondi* and *T. gondii* are indistinguishable from each other. The tissue cysts of *H. hammondi* resemble those of *T. gondii* and can also be confused with sarcocysts of some thin-walled *Sarcocystis* species [115].

**Life Cycle** - The only known feline definitive hosts are cats. The life cycle is described as being obligatorily heteroxenous. Four and 5 days after tissue cysts are ingested two types of schizonts are found in the cat’s small intestines. Five days after infection the sexual stages first appear. The prepatent period is 5 to 6 days, and the range of the patent period is 12 to 28 days. Oocyst excretion or latent infections in the tissues of cats do not occur when cats are orally inoculated with *H. hammondi* oocysts. Natural intermediate hosts of *H. hammondi* include goats (*Capra hircus*), rats (*Rattus rattus* and *R. norvegicus*), and roe deer (*Capreolus capreolus*). Experimental intermediate hosts include long-tailed field mice, white mice, deer mice, yellow-necked field mice, rats, multimammate rats, guinea pigs, hamsters, bank voles, European voles, field voles, dogs, goats, sheep, monkeys, and pigs [116]. Infection of chickens with *H. hammondi* does not occur [118]. After excystation in the intestine of an intermediate host, the sporozoites penetrate the intestinal mucosa and multiply by endodyogeny in the lamina propria, submucosa muscularis, Peyer's patches, and mesenteric lymph nodes. By 11 days after infection, tissue cysts are present in the muscles of mice. The tissue cysts are initially small but in about a month can be over 300 µm in length. Viability of the tissue cysts is 1.3 years and probably longer. Intermediate hosts cannot be infected by tissue cysts. Congenital transmission of *H. hammondi* does not occur in cats [117] or mice [118]. Immunity to *H. hammondi* is less stable than immunity to *T. gondii* in cats. When challenged, about 50% of cats will re-excrete oocysts [119]. In the absence of reinfection some cats will spontaneously re-excrete small numbers of oocysts. The course of a primary *H. hammondi* infection in cats is not affected by immunosuppression and immunosuppression does not cause cats to re-excrete oocysts. Cats are not made immune to infection with *T. gondii* when infected with *H. hammondi*. Cats do not develop antibodies to *T. gondii* after they have recovered from a *H. hammondi* infection.

**Disease** - In cats *Hammondia hammondi* does not cause disease. Up to 30% of mice die if they are inoculated with 105 to 106 oocysts. Lesions due to multiplication of tachyzoites are present in the intestinal tracts of these mice. A transient myositis in the skeletal muscles of infected mice may also be present.

**Treatment** - There have been no attempts to treat cats for *Hammondia* infections.

### Sarcocystis (Coccidia)

**Parasite Identification** - The stage of *Sarcocystis* passed in the feces of cats is a sporulated sporocyst containing 4 sporozoites. The sporocysts are oblong and around 10 µm in length (Fig. 13).

![Figure 13. Sarcocystis sp. Sporocyst as it appears when passed in feces of the carnivore host.](https://www.ivis.org)
**Life Cycle** - All *Sarcocystis* species have an obligatory two-host life cycle. Cats are important as definitive hosts for at least 11 named species (Table 3) and are the intermediate host for one species, *Sarcocystis felis*. The intermediate hosts become infected by ingestion of sporocysts from the environment. The sporozoites excyst from the sporocysts in the intestinal tract. The sporozoites leave the intestinal tract and undergo first-generation merogony in endothelial cells of arteries usually in mesenteric lymph nodes [120]. A second generation of merogony occurs in capillaries or small arteries in many tissues throughout the body. The meronts are usually most numerous in glomeruli of the kidneys. The merozoites from the last generation are released into the circulation and can occasionally be found intracellularly in unidentified mononuclear cells [120]. Limited multiplication may occur at this stage of infection. Eventually these merozoites will penetrate cells and develop into the sarcocyst stage that contains bradyzoites (Fig. 14). The first- and second-generation meronts develop directly in the host cell cytoplasm, whereas the bradyzoites develop within a parasitophorous vacuole.

![Figure 14. Sarcocystis sp. High magnification of a muscle cyst (the stage in the intermediate host) showing the myriad bradyzoites. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

<table>
<thead>
<tr>
<th>Species of <em>Sarcocystis</em></th>
<th>Intermediate Host</th>
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<tbody>
<tr>
<td><em>S. cuniculi</em></td>
<td>European rabbit (<em>Oryctolagus cuniculus</em>)</td>
</tr>
<tr>
<td><em>S. cymruensis</em></td>
<td>Norway rat (<em>Rattus norvegicus</em>)</td>
</tr>
<tr>
<td><em>S. fusiformis</em></td>
<td>Water buffalo (<em>Bubalus bubalis</em>)</td>
</tr>
<tr>
<td><em>S. gigantea</em> (syn. <em>S. ovifelis</em>)</td>
<td>Sheep (<em>Ovis aries</em>)</td>
</tr>
<tr>
<td><em>S. hirsuta</em> (syn. <em>S. bovifelis</em>)</td>
<td>Cow (<em>Bos taurus</em>)</td>
</tr>
<tr>
<td><em>S. leporum</em></td>
<td>Cottontail rabbit (<em>Sylvilagus floridanus, Sylvilagus nuttalli, Sylvilagus pallistris</em>)</td>
</tr>
<tr>
<td><em>S. medusiformis</em></td>
<td>Sheep (<em>Ovis aries</em>)</td>
</tr>
<tr>
<td><em>S. moulei</em> (syn. <em>S. caprifelis</em>)</td>
<td>Goat (<em>Capra hircus</em>)</td>
</tr>
<tr>
<td><em>S. muris</em></td>
<td>House mouse (<em>Mus musculus</em>)</td>
</tr>
<tr>
<td><em>S. odoi</em></td>
<td>White-tailed deer (<em>Odocoileus virginianus</em>)</td>
</tr>
<tr>
<td><em>S. porcifelis</em></td>
<td>Pig (<em>Sus scrofa</em>)</td>
</tr>
</tbody>
</table>

Table 3. Names and Intermediate Hosts of *Sarcocystis* Species Transmitted by Cats

The developing sarcocyst contains a stage called a metrocyte that divides by endodyogeny to produce the bradyzoites. A mature sarcocyst can contain thousands of bradyzoites and be grossly visible. The presence of grossly visible sarcocysts of *S. gigantea* in sheep and *S. hirsuta* in cattle is a cause for condemnation of the carcass [121,122].

**Disease** - The enteric stages of *Sarcocystis* in cats cause little to no disease.

**Treatment** - None required.

**Nematodes** -

*Toxocara cati* (*Ascarids*)

**Parasite Identification** - The adult worms are cream-colored to pinkish and are up to 10 cm in length. Warren [123] reports males as 3 to 7 cm in length and females as 4 to 10 cm in length. The adults have distinct cervical alae that are wide and short giving the anterior end the distinct appearance of an arrow. The esophagus is about 2% to 6% of the total body length and terminates in a glandular ventriculus that is about 0.3 to 0.5 mm in length. The vulva of the female is 25% to 40% behind the
Sprent [124] described the development of *Toxocara cati* then underwent considerable growth. When kittens were fed mice given 10,000 infective eggs of *Toxocara cati*, larvae were recovered from the muscle tissues of the cat. The larvae that make the liver-lung migration and return to the stomach via the stomach wall. After the tenth day of infection, larvae were in the lungs and stomach wall, and a number of larvae began to be and there were also larvae in the stomach wall. Five days after infection, larvae were in the lungs, tracheal washings, and form the alimentary tract before commencing development. By three days after infection, larvae were in the liver and lungs, that had been orally infected with eggs. After giving kittens 10,000 embryonated eggs, the larvae were found to migrate away to produce the infective third-stage larva. The egg is passed containing a single cell, and after a period of time in the environment, two molts occur within the eggshell to produce the infective third-stage larva.

**Toxocara cati**. Eggs in cat feces. Not the thickened mammilated shell and the dark zygote within. - To view this image in full size go to the IVIS website at www.ivis.org.

**Life Cycle** - The adult worms live in the small intestine (Fig. 16) and the female produces eggs that are passed in the feces. The egg is passed containing a single cell, and after a period of time in the environment, two molts occur within the eggshell to produce the infective third-stage larva.

**Figure 15.** *Toxocara cati*. Eggs in cat feces. Not the thickened mammilated shell and the dark zygote within. - To view this image in full size go to the IVIS website at www.ivis.org.

**Figure 16.** *Toxocara cati*. Intestine of a cat at necropsy showing adult worms. - To view this image in full size go to the IVIS website at www.ivis.org.

Sprent [124] described the development of *Toxocara cati* in the feline host following oral infection with eggs and with mice that had been orally infected with eggs. After giving kittens 10,000 embryonated eggs, the larvae were found to migrate away from the alimentary tract before commencing development. By three days after infection, larvae were in the liver and lungs, and there were also larvae in the stomach wall. Five days after infection, larvae were in the lungs, tracheal washings, and stomach wall. After the tenth day of infection, larvae were in the lungs and stomach wall, and a number of larvae began to be recovered from the muscle tissues of the cat. The larvae that make the liver-lung migration and return to the stomach via the trachea then undergo considerable growth. When kittens were fed mice given 10,000 infective eggs of *Toxocara cati*, almost all larvae were found to develop without a liver-lung migration, and larvae were only rarely recovered from muscle tissues. The larva in the egg measures 0.31 mm to 0.42 mm in length. Within the stomach wall of the cat, the larvae grow to about 1.3 mm in total length. The molt from third stage to fourth stage occurred when the larvae measured 0.999 to 1.235 mm in length. Fourth stage larvae were found in the stomach contents, intestinal wall, and intestinal contents. In egg-infected cats, fourth-stage larvae were first observed 19 days PI; whereas, in mouse-infected cats, fourth-stage larvae were first observed 10 days PI. The molt to the young adult stage occurred within the intestinal lumen when the larvae were 4.3 to 6.5 mm in length. The fourth-stage larvae differ from the young adults of similar length by the much thinner cuticular annulations on the adults. The smallest female observed to have eggs in its uterus was 55 mm long. The prepatent period was 56 days. In four older cats that were each experimentally infected with 500 embryonated eggs, the observed prepatent period was 38, 39, 39, and 40 days (personal correspondence from Dr. Stoye to Dr. J.C. Parsons).

Transplacental transmission does not occur. During the last 4 weeks of gestation, a pregnant queen was given three inocula of 10,000 embryonated eggs [124]. Larvae were not recovered from the tissues of the kittens after birth. Transmammary transmission commonly occurs with *Toxocara cati* [125]. The examination at birth of 78 kittens from 20 queens that were naturally infected with *Toxocara cati* and 14 kittens from 7 queens that were experimentally infected with 300 to 2,000 eggs of *Toxocara cati* per day from day 2 to 56 prepartum revealed no larvae in the organs of the kittens when they were examined. When 12 kittens were examined 15 to 22 days after natural delivery from 5 queens that had been orally infected with 2,000 eggs of *Toxocara cati* for 1 to 10 days prepartum, a total of 7,959 larvae were recovered, with most of the larvae being recovered from the gastrointestinal tract. Larvae were not found in the 5 littersmates (one from each litter) that had not been allowed to nurse. Larvae were also found in the mammary glands and milk of these 5 queens. Nineteen kittens that were derived by caesarian section from 6 queens and raised colostrum-free to maturity remained free of *Toxocara cati*. The life cycle of *Toxocara cati* probably routinely involves paratenic hosts. The larvae can persist in the tissues of cockroaches [124], earthworms [126]; mice [127], chickens [124,126], dogs [124], and lambs [124]. Beaver et al. [128] showed that both *Toxocara cati* and *Toxocara canis* produce lesions in white mice that are similar to those observed in human cases of visceral larva migrans. The larvae undergo a liver-lung migration in the mouse [129-131], and then later, the larvae of *Toxocara cati* are mainly found in the somatic musculature [124].

**Disease** - Kittens often have no clinical signs due to *Toxocara cati* infection. However, kittens are capable of displaying signs similar to puppies with moderate worm burdens, i.e., a pot-bellied appearance and a general failure to thrive. On occasion it may be possible to palpate thickened intestines.

In a 7 year old domestic male cat that had anorexia, vomiting, and an enlarged abdomen [132] a laparotomy revealed perforated gastric ulcer and an adult *Toxocara cati* in the abdominal cavity. The next day, acute perforations of the stomach
occurred again, and during a second surgery, two new gastric perforations were repaired and four adult *Toxocara cati* were removed from the abdominal cavity. The cat died during recovery from the second emergency surgery, and it is difficult to determine whether the ascaridoids were the cause of the gastric perforations or were simply migrating into lesions from some other underlying cause.

**Treatment** - Treatment of gastrointestinal infections with *Toxocara cati* is straightforward. Approved compound include piperazine, pyrantel, dichlorvos, febantel formulated with praziquantel, and pyrantel formulated with praziquantel. Ridley et al., [133] reported on the use of pyrantel pamoate for the treatment of *Toxocara cati* in kittens experimentally infected by the feeding of infected mice; at 20 mg of base per kg body weight, this compound was 100% effective in removing the worms from these cats. The febantel formulated with praziquantel has been shown to be 100% effective in removing *Toxocara cati* from cats [134]. Ivermectin (200 µg per kilogram body weight) has been found to remove adult *Toxocara cati* from infected cats [135]. Milbemycin oxime (500 µg per kilogram body weight) is also effective against the adults of *Toxocara cati*. There has been no work published on the pharmacological prevention of the transmammary transmission of larvae.

*Toxascaris leonina* (Ascarids)

**Parasite Identification** - The adults of *Toxascaris leonina* are cream colored to pinkish worms. The females are 6 to 10 cm in length, and the males are about 5 cm long [136]. The cervical alae of the adults are longer and considerably narrower than those of *Toxocara cati*, and the head of *Toxascaris leonina* resembles a spear while (Fig. 17) the head of *Toxocara cati* resembles an arrow head [137].

![Figure 17. *Toxascaris leonina*. Anterior end showing the alae that five the head of this worm a spear-shaped appearance. (The alae on *Toxocara cati* appear much broader making the head of that worm look like an arrowhead.) - To view this image in full size go to the IVIS website at www.ivis.org.](www.ivis.org)

There is no ventriculus on the esophagus of *Toxascaris leonina* [138]. The vulva of the female *Toxascaris leonina* is about 33% from the anterior end of the worm [136]. The males of *Toxascaris leonina* have tails that gradually taper to a point. The eggs of *Toxascaris leonina* have a smooth shell and are about 70 µm by 80 µm; Warren [139] reported ex utero eggs as having dimension of 54 by 74 µm (Fig. 18). The cell in the egg of *Toxascaris leonina* appears lighter than the zygote in the eggs of *Toxocara cati* (Fig. 19).

![Figure 18. *Toxascaris leonina*. Egg showing the thick shell that is smooth unlike that of *Toxocara cati*. - To view this image in full size go to the IVIS website at www.ivis.org.](www.ivis.org)

![Figure 19. *Toxascaris leonina*. Two eggs in the same field as a single egg of *Toxocara cati*. Not the T. leonina eggs have smooth shells, appear lighter internally, and one has already undergone a single division. - To view this image in full size go to the IVIS website at www.ivis.org.](www.ivis.org)

**Life Cycle** - The adult worms live in the small intestine, and eggs are passed in the feces. The eggs of *Toxascaris leonina*, unlike those of *Toxascara canis*, are capable of developing to the infective stage at 37ºC [140]. Around 95% of eggs will contain infective-stage larvae after four days of culture at 25ºC [136]. At 17ºC to 22ºC, it takes 6 days for 99% of the eggs to reach the infective stage; at 30ºC, it takes five days, and they also found that 97% of the eggs reached the infective stage when held at 37ºC. At 40ºC the eggs were incapable of completing their development even after being returned to 25ºC. Cats can be infected by the ingestion of embryonated eggs. After ingestion of the eggs, the larvae enter the wall of the small intestine, and grow to a length of 0.5 to 0.6 mm [142]. The larvae then molt to the fourth stage, grow to around 6 mm, then molt to the adult stage. A characteristic feature of infections with *Toxocaris leonina* in the cat is the persistence of small fourth-stage larvae in the wall and intestinal lumen of the cats for weeks to months after infection [141]. Adults appear as
avoid confusion with larvated hookworm eggs. The tail of the parasitic female of Strongyloides planiceps has a narrower tail and the free-living female of Strongyloides stercoralis has a longer tail and narrows more slowly to the tip of the tail [146].

**Parasite Identification** - The differences between *Strongyloides felis* and *Strongyloides stercoralis* are that the parasitic female of *Strongyloides felis* has a narrower tail and the free-living female of *Strongyloides felis* has a post-vulval constriction of the body which is lacking in the free-living female of *Strongyloides stercoralis*. The parasitic females of *Strongyloides felis* and *Strongyloides stercoralis* have ovaries that are straight, while the parasitic female of *Strongyloides planiceps* has ovaries that spiral. Also, infections with *Strongyloides felis* and *Strongyloides stercoralis* result in larvae being passed in the feces of the infected cat, while infections with *Strongyloides planiceps* produce eggs that are found in feces that are freshly deposited. *Strongyloides tumefaciens* is different from the other species of *Strongyloides* present in the cat by having a longer parasitic female (5 mm long) that is found in tumors in the mucosa of the large intestine.

The Baermann technique is best for the diagnosis of infection. Larvae can be detected using a direct smear of feces, but the sensitivity of this method is low because most cats shed less than 50 larvae per gram of feces. It is necessary to perform fecal cultures that will generate the free-living adult stages to determine whether a cat is infected with *Strongyloides felis* or *Strongyloides stercoralis*.

**Life Cycle** - Speare and Tinsley [148] described the life cycle of this parasite. The adult parthenogenetic female lives in the mucosa of the small intestine and produces eggs that hatch to produce first-stage rhabditiform larvae in the small intestine that are passed in the feces. The larvae passed in the feces will typically develop into male and female free-living adults. The free-living adults typically produce eggs that hatch to yield larvae that mature to the infective filariform third-stage larva in about six days at 20°C.

Occasionally, in about two days the larvae passed in the feces will develop directly to infective-stage larvae. There is only one generation of the short-lived free-living adults; within ten days after cultures are established most of the adults die. The cat is infected by the penetration of the skin by infective-stage larvae. After entering the skin, the infective larvae are carried to the lungs, break through the alveolar spaces, migrate up the trachea, and down the esophagus to the small intestine. The prepatent period is 11 days with a range of 9 - 14 days. The fact that kittens of infected queens remain uninfected and that infections are found almost exclusively in older cats would suggest that transmammary infection with this species does not occur. Infections in experimentally infected cats have been shown to persist for over two years.
Disease - No pathognomonic clinical signs of infection with this parasite are present [148]. Diarrhea is not a typical feature of infection, although acute watery diarrhea that cleared after treatment occurred in some cats with naturally acquired infections (see below). The experimental infection of cats with 500 larvae produced no signs other than unkempt coats and a slight unthriftiness. In experimentally infected cats, alertness and appetite remained normal. An inflammatory response associated with the intestinal phase of the infection does not appear to occur, but some worms have been found associated with an adenomatous metaplasia of the glandular epithelium of the intestinal crypts (Fig. 20) [148]. Subpleural inflammatory plaques, focal granulomas, and a vasculitis is associated with the migration of the larval stages through the lungs of experimentally infected cats.

Figure 20. *Strongyloides tumefasciens*. Histologic section showing adult in the colonic mucosa. - To view this image in full size go to the IVIS website at www.ivis.org.

Treatment - In three cats, thiabendazole (25 mg/kg BID for two days) has been found to be 100% efficacious. Fenbendazole (20 mg/kg SID for three days) caused no change in the numbers of larvae shed by two cats but in a third cat caused a transient disappearance of larvae. Only slight larval reductions are caused by oxendazole (8 to 9 mg/kg SID for two days). In two cats levamisole (10 mg/kg) in combination with niclosamide (200 mg/kg) caused a temporary drop in the production of larvae. Pyrantel emboate (12.5 mg/kg) and pyrantel pamoate (25 mg/kg), both were without effect following a single treatment. Praziquantel (25 mg/kg) also had no effect [148].

Ancylostoma tubaeforme (Hookworms)

Parasite Identification - The *Ancylostoma tubaeforme* adults are 7 to 12 mm long. Adult specimens of hookworms found in the cat can be distinguished on the basis of the shape of the buccal capsule. First, by determining whether or not there are ventral teeth in the buccal capsule, members of the genus *Ancylostoma* can be separated from those of *Uncinaria*. Specimens of *Ancylostoma* have large teeth within the buccal capsule while specimens of *Uncinaria* are recognized by having cutting plates. They can be differentiated from the adults of *Ancylostoma braziliense* and *Ancylostoma ceylanicum* by the presence of three teeth on either side of the ventral midline (*Ancylostoma braziliense* and *Ancylostoma ceylanicum* each possess only two such teeth).

The eggs of the different *Ancylostoma* species are apparently indistinguishable from each other. *Ancylostoma tubaeforme* eggs have been measured to be 55 - 76 by 34 - 45 µm with means of 61 by 40 µm (Fig. 21). The eggs of *Uncinaria* are larger than those of *Ancylostoma* being 70 to 90 µm in length by 40 to 50 µm in width. In mixed infections the two eggs are easy to distinguish [149].

Figure 21. *Ancylostoma tubaeforme*. Eggs in a cat fecal specimen. IN a fresh specimen, the eggs are usually in a four-cell to eight-cell stage. If specimens sit at room temperature overnight, the eggs may contain first stage larvae or hatched first-stage larvae. - To view this image in full size go to the IVIS website at www.ivis.org.

Life Cycle - The life cycle has been studied in some detail [150-153]. 68°F (20°C) is the optimal temperature for larval development, which is lower than the optimal temperature for developing larvae of *Ancylostoma caninum*. The infective third-stage larvae of *Ancylostoma tubaeforme* can infect cats both orally or through penetration of the skin. After oral inoculation, the larvae enter the stomach wall and proximal small intestine where they remain for 10 to 12 days while developing to the adult stage. Then the adults reenter the lumen. The prepatent period following oral administration of *Ancylostoma tubaeforme* is 18 to 28 days which is longer that the prepatent period of the other cat hookworms. About one month after the cat is infected, the worms will have reached their maximum length. After penetration of the skin by *A. tubaeforme larvae*, they migrate through the lungs, up the trachea, and down the esophagus. The larvae then spend very little time within the gastrointestinal tract wall. In larvae that have penetrated the skin development of fourth-stage larvae is more rapid, but there is no apparent growth of the larvae before they reach the intestinal tract. Following skin penetration the prepatent period is between 19 to 25 days. Penetration of the skin is through the secretion of proteolytic enzymes, although, an early investigation failed to reveal any in the examination of the larvae of *Ancylostoma tubaeforme* [154].

Rodents can be paratenic hosts. Larvae are found concentrated in the cranial aspect of their murine host after both oral and percutaneous infection, where they have remained alive for up to 10 months [155]. Larvae in the tissues of mice have been
shown capable of orally infecting other mice. Cats have not apparently been experimentally infected by the feeding of infected mice.

It is assumed that there is neither transmammary nor transplacental transmission of hookworms from the queen to her kittens. This assumption is consistent with the lack of hookworm disease seen in young kittens and the small number of larvae that have been recovered from the viscera and musculature after being infected with larvae either by the percutaneous or oral route [153]. This question, however, has never actually been carefully addressed experimentally.

In experimentally infected cats the life expectancy of the adult worms has been observed to be 18 months to 2 years [152]. Relatively few adult worms tend to be harbored by cats. In 235 cats from New Jersey with naturally acquired adult worms, there were 1 to 123 worms per cat (with a mean of 20). There has been no examination as to the number of eggs produced by a female each day or the effects of the age of cats on the ability of worms within the intestine to mature to the adult stage.

**Disease** - Clinical signs associated with an infection of *Ancylostoma tubaeforme* are anemia and weight loss (Fig. 22) [156]. When cats were given 1000 to 2000 infective-stage larvae, they lost weight when compared to cats that received 0, 100 or 500 larvae. The cats receiving 1000 or 2000 larvae died, and in these cats, hemoglobin levels fell to 4 g/dl and packed cell volumes fell to 20% after the cats were infected for a month. In the cats receiving fewer larvae, the hemoglobin levels and packed cell volumes also fell, but then after six weeks the levels seemed to stabilize.

![Figure 22. Ancylostoma tubaeforme. Intestine of a cat infected with about 100 adult worms showing the amount of blood present on the mucosa at necropsy. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

Cats have died from experimental infections with *Ancylostoma tubaeforme*. Rohde [157] gave cats infective larvae, and 16 of them died within 12 to 47 days after the cats were infected. Examination of the intestines after death revealed that between 7 to 290 adult worms (mean of about 100 worms per cat) were harbored by these cats. Within 46 days of the initiation of the infection nine Siamese cats given 2000 larvae, and six of nine Siamese cats given 1000 infective-stage larvae died. The small intestines of these cats were found to harbor between 183 to 213 adult worms.

**Treatment** - Several products are marketed for the treatment of *Ancylostoma tubaeforme* in cats. Oral treatments with febantel and a febantel-praziquantel mixture (10 mg febantel/kg in cats and 15 mg febantel/kg in kittens), dichlorvos (11.1 mg/kg body weight), and n-Butyl chloride (400 mg/kg) are marketed for cats as well as disophenol sodium which is to be administered subcutaneously at 10 mg/kg body weight [178]. Ivermectin is now available as a chewable for cats with an efficacy that has been shown to be 90.7% in cats for treatment of *Ancylostoma caninum* [159]. A combination praziquantel and pyrantel product (Drontal) is also now available.

### Ancylostoma braziliensis (Hookworms)

**Parasite Identification** - The adults of *Ancylostoma braziliense* are 4 to 10.5 mm in length. Members of the genus *Ancylostoma* can be distinguished from *Uncinaria* by determining whether or not there are ventral teeth present in the buccal capsule. Specimens of *Ancylostoma* have large teeth within the buccal capsule while *Uncinaria* specimens are recognized by the presence of cutting plates. The adults of *Ancylostoma braziliense* and *Ancylostoma ceylanicum* possess two teeth only on the ventral aspect of the buccal cavity with the lateral tooth being large and the median tooth quite small. The *Ancylostoma tubaeforme* adults have three teeth on each side of the buccal capsule. *Ancylostoma braziliense* can be differentiated from *Ancylostoma ceylanicum* by careful examination of the teeth present within the buccal cavity. In *Ancylostoma braziliense*, the medial teeth are smaller then they are in *Ancylostoma ceylanicum*. Careful examination of the copulatory bursa of the male is another means of separating these two species. The lateral lobes of the bursa are relatively shorter in *Ancylostoma braziliense* than they are in *Ancylostoma braziliense* and *Ancylostoma ceylanicum* the branching of the externo-dorsal rays occurs more posterior than it does in *Ancylostoma brasiliense*. Finally, Yoshida [160] showed that if the adults are killed in hot water (149°F or 65°C) prior to fixation that about 90% of the females of *Ancylostoma braziliense* are noted to have a distinct bend of 20 degree in the body at the level of the vulva (about two-thirds back on the body from the anterior end). This bend does not occur with adults of *Ancylostoma ceylanicum*.

In the cat, the eggs of the different *Ancylostoma* species found are apparently indistinguishable from each other. Most workers [181] (e.g., Sarles, 1929) consider the eggs of *Ancylostoma braziliense* to be slightly smaller (55 µm x 34 µm) than those of *Ancylostoma caninum* (62 µm x 38 µm). The eggs of *Uncinaria* are considered larger than those of *Ancylostoma* being 70 to 90 µm in length by 40 to 50 µm in width. In mixed infections the two eggs are easy to distinguish [162]. Yoshida [163] showed that the infective-stage larvae of *Ancylostoma braziliense* average 662 µm in length, which made them recognizable shorter than the larvae of *Ancylostoma ceylanicum* which averaged 712 µm long. The larvae of *Ancylostoma braziliense* are thus slightly longer than the larvae of *Ancylostoma caninum* which average 630 µm in length [164] and slightly longer than the *Ancylostoma tubaeforme* larvae that have been reported to be 630 µm long [165]. The infective-stage
laurae of the *Ancylostoma* species are all longer than the *Uncinaria stenocephala* larvae which measure only 500 to 580 \( \mu \text{m} \) in length. Thus, based on length, the infective-stage larvae of the four major feline hookworms rank as *Ancylostoma ceylanicum* (712 \( \mu \text{m} \)), *Ancylostoma braziliense* (660 \( \mu \text{m} \)), *Ancylostoma tubaeforme* (630 \( \mu \text{m} \)), and *Uncinaria stenocephala* (<600 \( \mu \text{m} \)) in length.

**Life Cycle** - Infection of cats can occur by the ingestion of larvae or by the larvae penetrating the skin. When cats are infected orally with third-stage larvae, these infective larvae enter the mucosa of the intestine. These larvae develop to the fourth stage within the mucosa. After the second day of infection the larvae then appear in the intestinal lumen. The prepatent period of the infection following oral infection is 14 to 16 days. If infection in cats is through the skin, the larvae migrate via the bloodstream to the lungs, migrate up the trachea, and are then swallowed. Most of the larvae are still young third-stage larvae when they reach the small intestine. The prepatent period is 13 to 27 days following cutaneous infection [166].

Ingestion of paratenic hosts can also cause infection of cats. After oral or percutaneous infection in mice, the larvae migrate via the bloodstream to the lungs and then proceed to the area of the head of the mouse, where they persist for up to 18 months. In mice, most larvae are found within the nasopharyngeal epithelium or within salivary glands [167]. Larvae recovered from infected mice have been used to experimentally infect cats [168].

On the basis of a single trial, it would appear that transmammary and transplacental transmission of *Ancylostoma braziliense* does not occur in dogs [169]. This has not been studied in the cat.

*Ancylostoma braziliense* adults have been reported to live about four to eight months [161]. Between 200 to 6000 eggs per day are produced by a single female worm [161]. The number of eggs produced by a single female decline as the infection matures. The infecion of younger cats can be more easily obtained than the infection of older cats [161].

**Disease** - It was noted that *Ancylostoma braziliense* was not as pathogenic as *Ancylostoma caninum* when this hookworm was originally described [170]. Very little hemorrhage occurs at the site of adult attachment or larval development. It has been shown that the blood loss due to infections of adults in kittens is about 1 to 2 ul of blood/worm/day using 51 Chromium-labelled erythrocytes [171]. Blood loss was first detected in cats 10 days after infection, and the experimentally infected kittens had hematocrit values, hemoglobin levels, and weight gains that were comparable to uninfected age-matched control kittens.

**Treatment** - Products for the oral treatment of *Ancylostoma braziliense* [172]: toluene (in a dichlorophen-toluene mixture with a dose of 264.5 mg toluene per kilogram body weight), dichlorvos (11.1 mg/kg), febantel (as a febantel-praziquantel mixture at the dose of 10 mg febantel per kilogram body weight for cats and 15 mg of the febantel per kilogram body weight in kittens), n-Butyl chloride (400 mg/kg), and praziquantel and pyrantel in Drontal is approved for this species. Ivermectin as a chewable product that is to be administered monthly at 24 \( \mu \text{g/kg} \) is reported to reduce infections by 98.1% as compared to untreated controls [159]. Disophenol sodium is approved in a formulation at 10 \( \mu \text{g/kg} \) that is to be administered subcutaneously.

**Uncinaria stenocephala (Hookworms)**

**Parasite Identification** - The adults of *Uncinaria stenocephala* are 3 mm to 12 mm long. They can be distinguished from the other hookworms found in the cat by the presence of cutting plates within the buccal capsule (Fig. 23), instead of the teeth that are present in species of *Ancylostoma*. The eggs of this worm can also be differentiated from *Ancylostoma* eggs by their larger size. The eggs of *Uncinaria stenocephala* are approximately 70 to 90 \( \mu \text{m} \) in length by 40 to 50 \( \mu \text{m} \) in length, and are easy to differentiate from the eggs of *Ancylostoma* when present in mixed infections [173].

![Image](https://www.ivis.org)

**Figure 23. Uncinaria stenocephala.** Anterior end of an adult showing the cutting plates in the buccal capsule. - To view this image in full size go to the IVIS website at www.ivis.org . -

**Life Cycle** - Cats are relatively refractory to *Uncinaria stenocephala* infection [157,174]. Only three cats produced patent infections when six cats were experimentally infected with larvae cultured from dog feces. The number of eggs produced in these cats was few, and they were present in the feces only for a short period. However, cats were readily infected with larvae cultured from dog feces in another study done in Istanbul, Turkey [175].

The course of infection with *Uncinaria stenocephala* has been described in dogs [176-178]. When larvae are orally administered, they undergo a limited somatic migration where they enter the crypts of the gastric glands in the pyloric region of the stomach and the glands of the duodenal mucosa for the first two days after infection. The larvae then reenter the...
intestinal tract, and as the worms develop, they move within the intestine in a caudal direction. The mature worms are found in the third quarter of the small intestine, and infections are patent in 13 to 21 days. Application of larvae to the skin results in lower rates of infection, and the larvae migrate to the lungs before reentering the gastrointestinal tract through the trachea and esophagus. The prepatent period following skin application is 15 to 17 days. Transmammary and transplacental transmission apparently does not occur with Uncinaria stenocephala. The infection of two bitches, each at the time of conception with 20,000 larvae failed to induce infection in the puppies. The infection of four bitches with 20,000 larvae at the time of whelping, also failed to produce infections in the nursing pups. The necropsies of these bitches 28 days after infection revealed adult Uncinaria stenocephala in the intestines, but the organs of the body contained no larvae.

The adults of Uncinaria stenocephala live for four months in the dog. A source dog used for experimental infections was infected for approximately one year. Others have reported that patent infections will persist about 6 months. The number of eggs produced by a single female per day has been calculated to be 16,000 to 19,000 per female per day or 3,000 to 5,000 eggs per female per day. Uncinaria stenocephala larvae will persist within the musculature of orally or percutaneously infected mice. As with the dog, more larvae are in the tissues following oral infection than following percutaneous infection. If the mother mice are percutaneously infected on the day of parturition, very low numbers of larvae are transmitted from the mothers to the mouse pups.

**Disease** - The infection is rare in cats. Uncinaria stenocephala is the least pathogenic of the hookworm infections in dogs. Blood loss caused by adults of Uncinaria stenocephala within the intestine has been calculated to be 0.3 ul per worm per day. This is only about 1% to 2% of the amount of blood lost in a dog due to the presence of a single Ancylostoma caninum. The oral inoculation of 1,000 larvae caused no signs of disease in beagle puppies. However, The oral inoculation of infective larvae has been reported to induce severe diarrhea and a 10% reduction in plasma protein levels. Infections of greyhounds or beagles with 87 to 1,850 adults caused suboptimum growth and protein-losing enteropathy. The oral inoculation of adult beagle bitches with 20,000 larvae at the time of conception or whelping caused only slight diarrhea that was on occasion accompanied with bloody mucous and a slight peripheral blood eosinophilia two weeks postinfection. Examination of the tissues of animals within a few days after infection have indicated that there are minimal lesions associated with the larvae that are found in the glands of the duodenum and stomach the first couple days after infection and that the appearance of the fourth-stage larvae in the ileum is marked by the appearance of petechial mucosal hemorrhages. There is marked inflammation around larvae that penetrate the skin, and within the lungs the larvae are found in focal areas of inflammation.

**Treatment** - Uncinaria stenocephala is more refractory to treatment with certain compounds than Ancylostoma caninum, the canine hookworm. Treatment with ivermectin in dogs at a dose of 6 µg/kg was 27% to 51% and 57% to 90% effective against the adults of Uncinaria stenocephala and Ancylostoma caninum, respectively. Similarly, milbemycin oxime was at 0.5 mg/kg body weight was 100% efficacious of adult Ancylostoma caninum, but had no effect on populations of adult Uncinaria stenocephala. Vaccination has been used to prevent infections of dogs with Uncinaria stenocephala. Dogs that received normal larvae infections were refractive to infection when challenged 200 days after the primary infection. Dogs receiving larvae irradiated with 40 Krads of gamma irradiation developed patent infections after being inoculated with irradiated larvae, but with infections producing much lower egg counts as compared to dogs inoculated with normal larvae. When these dogs that had been vaccinated with irradiated larvae were challenged with normal larvae, there was a marked reduction in the number of adults that developed in these animals compared to the unvaccinated controls.

**Trematodes**

Alaria marcianae (Alaria)

**Parasite Identification** - These are very small trematodes that are 1.2 to 1.6 mm in length with a distinct forebody and hindbody (Fig. 24). On each side of the small oral sucker are two tentacles directed anterior and associated with pseudosuckers that are about 100 µm long.

Figure 24. Alaria marcianae. Several adults recovered at necropsy showing the anterior alae that protrude form the anterior end. At necropsy, the worms are firmly attached to the mucosa and must be pulled free in many cases. - To view this image in full size go to the IVIS website at www.ivis.org. -

The oral sucker is about 100 µm in width. The forebody is concave ventrally, and the lateral margins fold in and slightly overlap the ventral surface of the body. The ventral sucker has about the same diameter as the oral sucker and is located in the middle of the forebody. The tribocytic organ is just posterior to the ventral sucker, and is approximate 150 to 200 µm
Life Cycle - The *Alaria marciana* life cycle was initially elucidated by Johnson [189] and then expanded by Shoop and Corkum [190,191]. The eggs in the feces are passed in an undeveloped state. The eggs contain miracidia that possess two pigmented eye spots after development for a period in water. Then the miracidia hatch and seek out a snail host of the genus *Helisoma* that they penetrate and in which they develop into sporocysts. From the sporocysts in the snail, longifurcous cercariae that possess both oral and ventral suckers, a pharynx, unpigmented eyespots, and well-developed penetration glands are released. The cercariae penetrate tadpoles and develop there. The stage in the tadpole is termed a mesocercaria because of its resemblance to an enlarged cercarial body without the tail. *Mesocercariae* can be passed between carnivorous paratenic hosts. On ingestion of the *mesocercaria* by the final host, the *mesocercariae* typically migrate through the lungs where they develop through the metacercarial stage before returning to the intestine to develop to adults. Nineteen days after infection, eggs are produced in the feces. The work of Shoop and Corkum showed that it is possible for *mesocercariae* to be transmitted by transmammary transmission between mammalian paratenic hosts. Also, if queens become infected with *mesocercariae* while nursing, they can pass the infection through the milk onto their offspring.

Disease - In cats there are no reports of *Alaria marciana* causing clinical disease. The migration, however, through the lung could cause migration tracts through the lung parenchyma.

Treatment - Praziquantel would probably be efficacious.

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**Cestodes**

*Spirometra species* (*Spirometra*)

The taxonomy of the genus *Spirometra* is very confused for reasons ranging from inadequate descriptions to assumptions made that specimens of larval forms recovered from intermediate or paratenic hosts in Europe are the same as those recovered from related hosts in Asia. It is also apparent that authors had initially tried to assume that the same final host would indicate specimens belonging to the same species. It is recommended that those interested in reading about the history of this confusion examine the section on *Spirometra* in Wardle and McLeod [192].

Two major species of *Spirometra* are recognized for the purpose of the following text. *Spirometra erinaceieuropaei* (Rudolphi, 1819) is considered to represent specimens that are mainly from Asia and Europe, although this species has also been reported from the Americas. *Spirometra mansonoides* Mueller, 1935 is discussed as the representative of specimens mainly from the Americas. This classification represents a beginning that can be used to allow information to be gained on these parasites although it is likely to be less than perfect. Very little attention in recent years has been given to the actual identity of the species of *Spirometra* present in cats in any given area, and it is hoped that further examination of specific characters will allow the identification of the important species infecting cats.

Four other species have been described from felids. One species, described by Southwell [193] is *Spirometra felis* for specimens recovered from *Felis tigris* and *Felis pardus* in the Calcutta Zoological Gardens, and Southwell believed them the same as the *Spirometra felis* described as *Bothriocephalus felis* by Creplin [194] from a domestic cat. Diesing [195] originally described a second species from a "cat-like" animal in Brazil as *Spirometra decipiens*. Supposedly, Chandler [196] "rediscovered" *Spirometra decipiens* in a domestic cat and a *Felis nebula* in the Calcutta Zoological Gardens. Wolffhügel and Vogelsang [197] described *Spirometra decipiens* in Uruguay from forms obtained by feeding dogs larvae from frogs, and these authors claimed they were the same as *Spirometra longicolle* from a *Felis jaguarondi* in Argentina. Faust et al. [198] recovered specimens identified as *Spirometra decipiens* from a leopard, a cat, and a dog in China and obtained the same form by feeding larvae from frogs to dogs. Saleque et al., [199] described a case of *Spirometra* that was not assigned to any certain species in a cat in India. In the Americas, two other species have been reported from non-domestic cats. In Brazil *Spirometra gracile* was described from small specimens recovered from *Felis macrura*. *Spirometra urichi* was described by Cameron [200] and recovered from an ocelot in Trinidad.
*Spirometra mansonoides* (Spirometra)

Parasite Identification - The original description of this tapeworm was provided by Mueller [201]. He stated that the maximum length of the strobila was 60 cm with a maximum width of 7 mm. Following almost forty years of study, he later determined that this tapeworm may attain a length of 1.5 meters with a maximum width of a centimeter or more in a large dog [202]. This tapeworm has serrate margins. The strobila appears to be delicate in the neck region and robust in the posterior (Fig. 26).

Figure 26. *Spirometra mansonoides*. Adult from cat in a petri dish with a US nickel for size comparison. - To view this image in full size go to the IVIS website at www.ivis.org.

*Spirometra mansonoides* selectively absorb large quantities of vitamin B12 like the adult *Diphyllobothrium latum* [203]. The adult tapeworms have a characteristic pinkish color due to this absorption [202]. The scolex of *Spirometra mansonoides* lacks suckers, but possesses two shallow longitudinal grooves called bothria [201]. It varies from 0.2 mm to almost 0.5 mm in diameter with the bothria being approximately 1.0 mm in length. The bothria are broad, shallow, and flat bottomed. Each proglottid of *Spirometra* possesses a spiraled centrally located uterus and associated uterine pore through which eggs are released. The uterus consists of an anterior series of heavy "outer" coils and a posterior series of narrow "inner" coils. A narrow duct that will accommodate only three or four eggs joins these two regions. The genital and uterine apertures open on the ventral surface of the proglottid. Characteristically these tapeworms release their eggs until they become exhausted of their uterine contents. Until groups of segments have shed all their eggs and are passed as "spent" segments, segments are not discharged into the feces [204]. In the proglottids of *Spirometra mansonoides*, the uterus terminates in the anterior of the proglottid in a distinct "U"-shaped uterus packed full of eggs. In *Spirometra erinaceieuropaei* this distinct uterine formation does not occur.

The egg of *Spirometra* resembles that of a digenetic trematode: it is yellow-brown, oval, and possesses a distinct operculum at one pole (Fig. 27). The eggs measure 60 µm by 36 µm. The eggs of *Spirometra* have an asymmetric appearance and tend to be pointed at one end. The abopercular end may have a slight bump. When the eggs are passed in the feces they are unembryonated. It is possible that cats will have negative fecal samples for extended periods and these fecals will be followed by periods when eggs are present.

Figure 27. *Spirometra mansonoides*. Egg passed in cat fecal. - To view this image in full size go to the IVIS website at www.ivis.org.

Life Cycle - Domestic and wild felids serve as the principal definitive hosts for *Spirometra mansonoides*, although raccoons and dogs may also harbor the adult cestodes; Mueller believes the bobcat, *Lynx rufus*, to be the natural host in the Americas. Unembryonated eggs pass through the adult cestode's gravid proglottids' uterine pore and are discharged with the cat's feces to the external environment. Unembryonated eggs can be stored in water for at least a year at 4°C without any significant decreases in viability. The eggs develop to the infective stage in the presence of aeration at room temperature, and can then also be stored at 4°C for at least a year. Eggs are induced to hatch by cold temperature shock and exposure to direct sunlight. The first developmental stage in fresh water, the ciliated coracidium, emerges from the egg and is eaten by the first intermediate host, a freshwater crustacean of the genus *Cyclops*. The procercoid, the second developmental stage, develops within the copepod. When the second intermediate host, a frog, water snake, or rodent ingests procercoid, it develops into a plerocercoid or sparganum, the third developmental stage. These ribbon-like, white, spargana are primarily found in subcutaneous sites. Cats become infected with *Spirometra mansonoides* by preying on frogs, fish, water snakes, birds, or rodents containing the infective plerocercoids. The tapeworm develops to the mature stage within ten to thirty days in the cat's small intestine. Some of the plerocercoids may develop in the intestinal wall and Mueller hypothesized that they may eventually migrate back into the lumen of the gut to begin an infection with the adult tapeworm. The adult tapeworm may survive in the cat for as long as 3.5 years [202]. Infection of cats can also occur by the larval form of *Spirometra*. 
Disease - Most of the reports of infection with *Spirometra* species in domestic cats have been from results of fecal assays or parasitologic surveys. Muller [205] described the signs of infection as causing marked signs in cats with adult worms. Animals that are infected lose weight but remain hungry. The intestinal wall of the infected cat became thickened, particularly in the layers of circular muscles. Recovery is rapid if a cat is given an anthelminthic. If animals have been infected for long periods, stunted growth may occur where deworming fails to result in the animals reaching target weights. There is a marked retardation in growth if nursing kittens become infected. Muller believed that in infected cats a severe anemia developed, but no specific parameters were reported to substantiate this claim. In the United States, one cat infected with adult *Spirometra* did exhibit an intermittent watery diarrhea of two months duration, which resolved following therapy [204].

Treatment - It is expected that treatment of *Spirometra mansonoides* in cats with praziquantel at the elevated dosage of 30 to 35 mg/kg body weight would cause the elimination of these parasites as they do *Spirometra erinaceieuropaei* [206]. The *Spirometra* reported by Kirkpatrick and Sharninghausen [204] in a domestic cat was likely to be *Spirometra mansonoides*. This tapeworm was refractory to treatment with albendazole at 25 mg/kg BID for 6 consecutive days and with 1500 mg of niclosamide following an overnight fast. A single treatment of the cat with 100 mg of bunamidine HCl appeared to remove the worms from this cat based on a postmortem performed a month later after death by other causes. Bunamidine (Scolaban) is administered to cats orally at a rate of 25 to 50 mg/kg body weight up to a maximum of 600 mg. Bunamidine irritates the oral mucosa and thus the tablets should not be broken, crushed, mixed with food or dissolved in liquid because. Bunamidine should be administered on an empty stomach, and following medication, food should be withheld for 3 hours. Treatment with bunamidine should not be repeated within a 2 week period. Also, it should not be concurrently administered with butamisole (Styquin), to unweaned kittens, or to cats with hepatic or cardiac disease. Cats should not be allowed to become excited or exercise immediately after treatment with bunamidine. Diarrhea, vomiting, and ventricular fibrillation are the most frequent side effects [207].

*Mesocestoides lineatus* (Mesocestoides)
Parasite Identification - The gravid proglottids of *Mesocestoides lineatus* are smaller than those of *Taenia taeniaeformis* and *Dipylidium caninum* and are easily recognized by the large parauterine organ present in the segments that contain the eggs of this worm that have the common hexacanth embryo of cestodes. The anterior end of the worm has a scolex with four distinct suckers that bears neither a scolex nor a rostellum (Fig. 28). The neck of the tapeworm contains immature segments.

Figure 28. *Mesocestoides lineatus*. Anterior end showing the muscular suckers and the lack of any rostellum or hooks on the anterior end. - To view this image in full size go to the IVIS website at www.ivis.org . -

Life Cycle - Segments are shed by the adult cestode and they are passed in the feces (Fig. 29 and Fig. 30).

Figure 29. *Mesocestoides lineatus*. Segments collected form a fecal specimen. - To view this image in full size go to the IVIS website at www.ivis.org . -

Figure 30. *Mesocestoides lineatus*. Segment from feces that has been stained brown by fecal pigments. - To view this image in full size go to the IVIS website at www.ivis.org . -

Loos-Frank [208] described the behavior of the proglottids passed in the feces noting that "Gravid proglottids have a conspicuous behavior. On freshly produced feces they stand more or less upright and their elongated anterior part makes slow
waving movements. After a few minutes they start crawling away from the feces and can be found quite a distance away from the pellets in moist conditions. In a meadow they were observed on grasses all around freshly deposited feces of a wild fox". She also described the fact that the strobila of *Mesocestoides lineatus*, like that of *Spirometra mansonioides*, is often shed in a relatively cyclical manner wherein they may be shed for a month and then no segments are shed during the next month. Examination of the animals at necropsy revealed that during the periods of segment shedding these cestodes were shedding rather large portions of their strobila causing the populations of worms present to become markedly shortened. This is supported by the observations of Witenberg [209] that in infected animals there were often cestodes of varying states of maturity recovered.

The development of the presumed first-stage larval form of *Mesocestoides lineatus* has not been described. Loos-Frank [210] examined this question with Dr. Ebermann, who had experience with the infection of oribatid mites with other cestode larvae, and they tried, as have others, to reproduce the work of Soldatova [211] who claimed that in a few of the mites that were fed on the eggs of this parasite that development occurred. Other arthropod hosts that have been fed eggs without the development of tetrathyridia include cockroaches and the maggots of *Musca domestica* and *Calliphora* sp. Loos-Frank [210], as have many other authors, also tried feeding the eggs directly to rodents without the development of larvae. Similarly, Witenberg [209] did not obtain production of any tetrathyridia when he fed eggs to various lizards. Thus, the hosts that serve as the first host of this parasite have never been determined, although it is in believed to be an arthropod. That the parasite is so universally found around the world suggest that they first intermediate host must be relatively common.

The second-larval stage, the tetrathyridium, occurs in the peritoneal cavity and musculature of numerous animals including amphibia, birds, reptiles, and mammals (including the cat). Large numbers of these larvae can be harbored by some animals. Ingestion of an intermediate host that is in infected with tetrathyridia causes cats to become infected with the adult tapeworm. Asexual multiplication does not occur by the tetrathyridia of *Mesocestoides lineatus* within the intermediate host [212,213]. Each tetrathyridium develops into a single adult cestode in cats [208]. Kawamoto et al. [213] demonstrated that after ingestion by the cat of tetrathyridia from snakes that were maintained in the peritoneal cavities of mice prior to use, the majority of the tetrathyridium was shed leaving only the scolexes and necks attached to the mucosa of the intestine. The strobila reached lengths of 23 to 52 cm and gravid proglottids with eggs occupied about 20% of the terminal portion of the strobila by 10 days after infection of the cats. Twenty-one days after the cats were infected, shedding of proglottids began. The larvae of a related species which was found in the peritoneal cavity of a population of fence lizards in southern California by Specht and Voge [214], *Mesocestoides vogae* (synonym = *Mesocestoides corti*), does undergo proliferation when inoculated into other hosts, such as the laboratory mouse; however, this is the only isolate of *Mesocestoides* that has displayed this property. This tapeworm also undergoes proliferation in the intestine of dogs and cats that are fed the tetrathyridial stage [213], but it has never been determined that the cat or dog are the natural final host of this species of tapeworm.

The adult worms develop in about two to three weeks after the cat ingests the tetrathyridial stage [208,209]. Also, prepatent periods as long as 56 days have been reported [215,216]. Witenburg believed that when the cat was not the typical natural host of the adult stage that the longer prepatent periods occurred. Not all tetrathyridia ingested by cats develop to the adult stage; some mature to adults while others migrate through the wall of the intestine to continue as the tetrathyridial stage in the cat [209,217,218]. In the infected cat, the adults of *Mesocestoides lineatus* can probably live for around a year [208].

**Treatment** - Praziquantel has been used to treat dogs experimentally infected with *Mesocestoides vogae* (synonym = *Mesocestoides corti*), and was found at 5 mg/kg to be 100% efficacious [219]. A dose of 2.0 mg/kg has also successfully treated dogs with *Mesocestoides vogae* [220]. In cats naturally infected with this parasite, nitazoxanide (at 100 mg/kg) was found to be 100% effective against *Mesocestoides lineatus* [209].

**Dipylidium caninum**

**Parasite Identification** - The scolex of the adult *Dipylidium caninum* is tiny, having a diameter less than 0.5 mm. It possesses four muscular suckers that aid in locomotion and attachment. At the apex of the scolex is a dome-shaped projection called the rostellum (Fig. 31). In *Dipylidium caninum* the retractable rostellum is armed with four to seven rows of tiny, backward facing, rose-thorn-shaped hooks. This tapeworm may attain a length of from 15 to 70 cm and be 2 to 3 mm in width. The tapeworms can appear very white, yellow, or a light reddish yellow color (Fig. 32).

![Figure 31. Dipylidium caninum. Anterior end showing suckers and rostellum with hooks. - To view this image in full size go to the IVIS website at www.ivis.org . -](image-url)
The typical adult is composed of 60 to 175 segments [221]. Each proglottid contains two sets of male and two sets of female reproductive organs with each set of genital apertures opening medially on the lateral edges of the proglottid (Fig. 33). Proglottids of *Dipylidium caninum* have two genital pores for fertilization, but no opening to allow for the escape of eggs. *Dipylidium caninum* is sometimes referred to as the "double pored tapeworm" because of these bilateral genital pores. Within each proglottid eggs accumulate until the proglottid becomes packed like a ripe seed pod [222]. Gravid proglottids are creamy white, 10 to 12 mm in length and resemble the seeds of a cucumber. Thus, *Dipylidium caninum* is sometimes also referred to as the "cucumber seed tapeworm" [223]. Gravid tapeworm proglottids are filled to capacity with egg packets or egg capsules (Fig. 34), each of which contain from 5 to 30 hexacanth ova [222].

The terminal tapeworm proglottids are passed in the feces [223]. Since the tapeworm proglottids possess both longitudinal and circular smooth musculature [224], they have the ability to move about the cat's perianal region, on the feces, on the bedding or across any surface where they may be deposited [223]. In the external environment these proglottids will desiccate; they shrivel up as they lose moisture, often resembling uncooked rice grains [221].

**Life Cycle** - The life cycle of *Dipylidium caninum* is in perhaps recounted by veterinarians more than any other parasite due to its ease of infectivity. As mentioned previously, the adult parasite is found in the small intestine, and the gravid terminal segments are passed in the feces. The larval stages of the cat flea (*Ctenocephalides felis*) savor these segments and will descend actively upon a freshly passed proglottid to eat it [225]. The flea larva has mandibulate mouthparts that allow it to ingest the *Dipylidium caninum* eggs. Larvae of *Pulex irritans*, *Ctenocephalides canis*, and the dog louse, *Trichodectes canis*, are also capable of serving as intermediate hosts for *Dipylidium caninum*.

With the flea, the hexacanth embryo develops into a tailless cysticercoid, stage that causes infection in the cat. The rate of development of the larval tapeworm is determined by the ambient temperature. The flea becomes infected as a larva, however until the adult flea has emerged from its pupal case, the hexacanth embryo does not develop to an infective cysticercoid. Development is completed on the last day only in response to the host's body temperature [225]. The flea may contain an average of 10 cysticercoids with a range of 2 to 82. Ingesting the flea during the grooming process infects the cat [226]. Venard [227] fed a cat *Dipylidium caninum* infected fleas, and 23 days later he recovered tapeworms from the cat. Hinaidy [228] also reported 2 to 4 weeks to be the prepatent period.

**Disease** - Unless adult tapeworms are present in large numbers, they cause little harm or inconvenience to the feline definitive host. Convulsions and epileptiform seizures occasionally occur in cats with severe infections [221]. Heavy infections in young animals can produce non-specific abdominal symptoms including diarrhea or constipation. The animal may exhibit a pot-bellied, unthrifty appearance. In rare cases intestinal obstruction may occur. However, most clients
consider the sight of proglottids of *Dipylidium caninum* crawling about the cat's haircoat, on the client's bedclothes, or on the recently passed feces of the cat disgusting.

**Diagnosis** - The client may observe tapeworm segments crawling on or about the cat, although the laboratory diagnostician will fail to demonstrate the characteristic egg packets on fecal flotation. Egg packets within proglottids are best demonstrated by taking a gravid proglottid and teasing it open to disperse the egg packets in a small amount of physiologic saline or tap water. Inspection with a hand lens or the naked eye is usually sufficient for the identification of segments of *Dipylidium caninum*. The pathognomonic indicators are the characteristic cucumber-seed shape coupled with the double pore. In the vicinity of their cat's resting places, pet owners often find dehydrated, shrunken objects. These desiccated objects bear little resemblance to segments of *Dipylidium caninum* but they will assume their former cucumber seed appearance if they are rehydrated in water.

**Treatment** - Praziquantel is the anthelmintic with the broadest spectrum of cestocidal activity. In cats a single oral or subcutaneous dose (5 mg/ kg body weight) of this anthelmintic eliminates 100% of both immature and adult *Dipylidium caninum*. Epsiprantel is an alternative cestocide administered at a single oral dose of 5.5 mg/kg of body weight. A vigorous flea control program is an important adjunct to the treatment of dipylidiasis in cats. The cat's owner should be informed of the potential for reinfection via the flea intermediate host whenever a dose of cestocidal medication is dispensed.

**Taenia taeniaeformis**

**Parasite Identification** - The most robust of the tapeworm parasites found in the cat is *Taenia taeniaeformis*. This is also the only species of *Taenia* reported around the world from the domestic cat. The worm tends to be white, thick bodied, and around 15 to 60 cm long (Fig. 35). The scolex has two rows of hooks having the typical claw hammer shape of the *Taeniidae*. There tends to be approximately between 30 to 50 hooks per scolex (Fig. 36).

![Figure 35. Taenia taeniaeformis. Adult worms showing the stocky nature of this helminth. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

![Figure 36. Taenia taeniaeformis. Anterior end of adult showing the anterior rostellus, suckers, and the lack of a neck. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

Typically there is no neck, i.e., a portion of narrow segments, posterior to the scolex. Each of the mature segments possess a single lateral genital opening that randomly occurs on either one lateral side of a segment of the other. The terminal, gravid segments that are shed in the feces tend to be packed full of eggs and can easily be recognized by examination under a microscope as those of a *Taenia* by the typical brown-shelled taeniid egg containing a 6-hooked larva. The eggs of *Taenia taeniaeformis* are spherical and have a diameter of between 31 to 36 μm (Fig. 37).

![Figure 37. Taenia taeniaeformis. Egg passed in feces showing the three pairs of larval hooklets. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

Many species of *Taenia* are found in carnivores; Verster [229] recognized 29 valid species and several of questionable validity. Many species are found in the dog as the final host, including, *Taenia pisiformis*, *Taenia multiceps* and *Taenia ovis*. Some species are found in human beings, *Taenia solium* and *Taenia saginata*, or large cats, including, *Taenia gonyamai*, *Taenia ingwei*, *Taenia omissa* and *Taenia macrocystis*. Other final hosts included hyaenas, mustellids, foxes, and viverids. The separation of the species is based on the size and shape of the hooks on the rostellum along with various other characters, such as the type of larval stage, position of the genital ducts relative to the longitudinal excretory canals, etc. *Taenia*
*Taeniaeformis* was considered by Verster as the representative species for this groups of species and is considered to be most like the species found in mustellids and viveridus.

**Life Cycle** - In the cat adult tapeworms live within the small intestine and shed their terminal segments into the feces. These segments are capable of exiting at times other than during the passage of feces through the sphincter of a cat, and they may be found crawling near the cat or on the cat's fur. The segments are capable of crawling a considerable distance. The intermediate host is a small rodent where the larvae migrate through the wall of the intestine and develop to a *strobilocercus* stage in the liver (Fig. 38).

![Figure 38. Taenia taeniaeformis. Strobilocerci from experimentally infected rat. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

The *strobilocercus* is a larval stage that has a rather long segmented body that is crowned with the scolex that looks very similar to that found on the adult form. It seems that an age of about 2 months must be attained by the strobilocercus before it is in infective to a cat [230]. The posterior portion of the larva is digested away, and the anterior portion begins to grow into a tapeworm [231]. Between 32 to 80 days after the strobilocerci are ingested, patent infections develop [232]. Patent infections have been maintained by cats for 7 months to greater than 34 months. Each day, the cats produce about 3 to 4 segments, although the majority of segments appear to contain only 500 eggs or less (some can contain up to 12,180 eggs). Throughout the patent period destrobilization may occur sporadically without the termination of infection. If cats are reinfected with *strobilocerci* soon after their prior infections with the adult stage has terminated, cats can be infected with adult tapeworms again [232]. It has also been shown that by feeding cats *strobilocerci* in the presence of existing mature worms that cats can be superinfected with young tapeworms [233].

**Disease** - Infection of *Taenia taeniaeformis* asymptomatic as there are no signs associated with this infection.

**Treatment** - In cats praziquantel (5 mg/kg body weight) and epiprantel have both been shown to be efficacious in the treatment of *Taenia taeniaeformis*. Similarly, fenbendazole and febantel are approved for the treatment of this parasite in most of the world. Nitroscanate and mebendazole are also products that have efficacy against *Taenia taeniaeformis*.

**Diagnosis** - Diagnosis of infection in cats is by finding the distinctive segments in the feces or by finding the eggs upon fecal flotation. A segment can be crushed on a glass slide and examined for eggs to identify it. However, sometimes segments have shed most of their eggs, and they will contain very few eggs or perhaps none at all. The shedding of segments in the feces will typically insure that the cat is infected with *Taenia taeniaeformis* and not *Echinococcus multilocularis* in those areas where cats could also potentially be infected with *Echinococcus multilocularis* (see below), although it is possible for cats to be hosts of both parasites.

**Echinococcus multilocularis** (Echinococcus)

**Parasite Identification** - *Echinococcus* adults are all very small forms; the total length of all species is in usually less than a cm, with typical sizes being 2 to 11 mm. Also, there are typically very few numbers of segments (ranges between 2 and 7 in the strobilas) of these different species. The typical *Echinococcus multilocularis* has two rows of taeniid (claw-hammer shaped) hooks present on the scolex. The first row of hooks measures between 25 to 34 µm long, and the small hooks in the second row measure 20 to 31 µm in length. Typically the body has 2 to 6, although commonly 5 segments. Typically the total length of the body is in 1.2 to 4.5 mm. The genital pore opening tends to be anterior to the middle of each segment. *Echinococcus multilocularis* needs to be differentiated from three other species of *Echinococcus*. It appears that in the cat *Echinococcus granulosus* does not develop to the adult stage; thus, it is not expected that this species will be recovered from cats. However, in the gravid proglottid the genital opening in *Echinococcus granulosus* is posterior to the middle of each segment. *Echinococcus oligarthrus* could perhaps develop in cats and has been reported from wild felids in South and Central America. The hooks range from 28 to 60 µm in length on the scolex of *Echinococcus oligarthrus*, or about twice the size of those in *Echinococcus multilocularis*. Also in South and Central America is *Echinococcus vogeli* which is typically found as adults in the bush dog, *Speothos venaticus*, and it is not known if the cat could serve as a host for this species. The hooks of *Echinococcus vogeli* are similar in length to those of *Echinococcus oligarthrus*.

**Life History** - Rausch [234] describes the natural cycle of *Echinococcus multilocularis* as involving rodents, *Microtus*, *Lemmus*, and *Clethrionomys* and the arctic fox. Other fox species and coyotes serve as final hosts in other parts of the range. Cycles develop wherein dogs around villages become infected with the adult tapeworm and pose a threat to the humans living in the villages. Vogel [235] suggested that on farms in central Europe a cycle involving cats and house mice might be present. Leiby and Kritsky [236] suggested that in North Dakota, USA, this might occur where cats and deer mice (*Peromyscus maniculatus*) were found naturally infected.
After the ingestion of an infected rodent, the final host, the fox or dog, will produce eggs beginning 28 to 35 days. These environmentally resistant eggs are shed and contaminate the soil [237]. When a suitable rodent intermediate host ingests the egg, the 6-hooked larva hatches, penetrates the intestinal wall, and moves to the liver where it establishes the hepatic larval stage, as the alveolar hydatid cyst (Fig. 39 and Fig. 40).

The alveolar hydatid cyst allows the cestode to asexually proliferate in the intermediate host. The cyst contains a germinial membrane and develops hundreds to thousands of small stages, each of which is capable of developing into an adult worm and termed a protoscolex. It takes about 60 days for the protoscolices to become infective in the rodents, and sometimes the rapidly forming cyst can overwhelm rodents and kill them within weeks of infection [237]. When a suitable final host ingests infective protoscolices, the protoscolices embed themselves within the crypts of Lieberkühn where they begin development. The *Echinococcus multilocularis* adults localize in the posterior portion of the small intestine [238]. Apparently, the cat is a relatively poor host for *Echinococcus multilocularis*. Vogel [239] noted that the worms in the cats were smaller and produced fewer eggs when he succeeded in infecting 5 of 6 cats with *Echinococcus multilocularis* from foxes in southern Germany. Neither of two cats infected by Thompson & Eckert [238] with the European strain developed worms containing eggs. Zeyhle and Bosche [240] inoculated 10 cats and two red foxes with protoscolices, and found large numbers of cestodes in two cats, very few cestodes in 6 cats, and in two of the cats there were no cestodes. It was found that the worms from cats had an average of 106 eggs per gravid proglottid while the worms from foxes had an average of 300 eggs per gravid proglottid. Using *Echinococcus multilocularis* from a red fox in Minnesota, Crellin et al. [241] found that infections developed in 12 dogs that were inoculated with protoscolices while only 11 of 12 cats became infected; the infected dogs harbored more adults (mean of 875) than the cats (mean of 102). The worms recovered from dogs were also longer. Using *Echinococcus multilocularis* originally isolated from Alaska and maintained in rodents by intraperitoneal passage for 30 years, Kamiya et al. [242] found that the worms developed poorly in cats relative to dogs. They recovered very few worms from the cats, and the cat that was examined on day 30 of the study, contained no worms at necropsy. That the cat is probably a poor host is also indicated by natural infections. In cats the prevalence of infection is often low even when the surrounding levels of infection in foxes is in quite high [241,243]. Often, there are also few worms recovered from the infected cats [243,244], although in North Dakota, USA, Leiby & Kritsky [236] recovered 26 gravid worms from one cat and about 500 worms (50% gravid) from a second cat.

**Disease** - No signs associated with infection have been described. Dogs and foxes have been observed to harbor thousands of worms without signs.

**Treatment** - In the treatment of *Echinococcus multilocularis* praziquantel (5 mg/kg body weight) has been shown to be efficacious.

**Diagnosis** - *Echinococcus multilocularis* eggs are typical taeniid eggs with a brown eggshell, a six-hooked embryo, and being 27 to 38 µm wide and slightly ovoid in shape. Antemortem diagnosis is difficult because cats can be host either *Echinococcus multilocularis* or *Taenia taeniaeformis* and the eggs of these two worms are virtually indistinguishable. Thus, the risk is great of having an infection with *Echinococcus multilocularis* being misidentified as an infection with the more common *Taenia taeniaeformis*. The small proglottids of *Echinococcus* will not be recognized as such in the feces, and thus, an infection with *Echinococcus multilocularis* should be suspected if a cat routinely sheds eggs but never appears to shed segments.
Large Intestine
Protozoa -
*Pentatrichomonas hominis* (Trichomonads)

**Parasite Identification** - In cats, intestinal trichomonads have been described that differs from that found in the mouth [245]. Some authors consider the parasite from the intestine of the cat as a species unique to the cat [246,247]; however, others feel that it is the same species as in man and the dog. The organism in the feces of humans has been shown capable of causing infection in cats that are orally inoculated with trophozoites [248,249]. Reports of trichomoniasis from cats have been reported from the United States [249-252], South America [247], Europe [246,253,254], and China [252,255].

**Life Cycle** - The cycle is direct fecal-oral transmission. There is no cyst stage and the trophozoite is easily destroyed by dehydration or osmotic shock.

**Disease** - Most have considered this organism not to cause disease in the cat, rather being a commensal that is found in the feces of cats with diarrhea due to other causes. Romatowski [256] on the other hand, describes disease in four infected kittens. One cat had severe colitis that did not respond to treatment, and after the diarrhea the trichomonads appeared in the feces. A second cat had loose feces that persisted for several months after the trichomonads disappeared from the feces, but as long as the cat received treatment with enrofloxacin, fecal consistency was normal. A third cat responded well to metronidazole treatment. The fourth cat was euthanized without treatment. All these cats were quite young, varying in age from 2 months to 4 months. Romatowski [257] had previously presented a series of cases in three kittens. Gookin et al [258] examined the effect of pentatrichomoniasis in a large number of cases in another recent study. These authors found most of the infections in cats that were less than or equal to a year of age. The feces were typically malodorous, pasty to semiformed, and often contained fresh blood and mucus. Defecation was associated with tenesmus and flatulence. The soft stools tended to persist from days to years, but lasted about 5 months typically.

Whether or not the organism is the cause of the associated diarrhea or another manifestation of some underlying problem still remains a question. As Romatowski stated: "Diarrhea in cats infected with *P. hominis* can be difficult to treat, and disappearance of trophozoites from fecal smears does not necessarily correlate with improvement in clinical signs". Gookin et al. suggest that in young kittens *P hominis* may be a cofactor in the development of diarrhea. That there is some other cause of the underlying diarrhea and that the presence of a different microbiological flora in the colon when large quantities of fluid material are present is such that *P. hominis* multiplies in number still remains a possibility. Arguing against this was the inability of Gookin et al., to establish cultures of organisms from normal feces of 100 random-source cats while they did isolate it from the normal feces of cats that had been treated with furazolidone and fenbendazole.

**Treatment** - There is not a specific efficacious treatment for the removal of these protozoa from the colon of infected cats. Romatowski [256] used either enrofloxacin or metronidazole and suggested that the long-term daily administration of enrofloxacin was a means of suppressing the soft stools associated with this infection. Gookin et al., [258] used fenbendazole, paromomycin, or furazolidone, with mixed success. The consistency of the stools would often improve, however, the organisms often appeared to be present at low levels since they could be detected by protozoal culture of the feces even in stools that had normal consistency. Gookin et al., [258] also reported that many of the cats they saw came to the hospital after having been treated by the referring veterinarians with a wide assortment of compounds: metronidazole, fenbendazole, pyrantel pamoate, sulfadimethoxine, trimethoprim - sulfadiazine, albendazole, furazolidone, tylosin, enrofloxacin, amoxicillin, clindamycin, and erythromycin.

**Diagnosis** - Finding the trophozoite in a direct fecal smear of fresh feces prepared with saline. Also, it is possible to culture fecal material to facilitate the diagnosis [258].

Nematodes -
*Strongyloides tumefaciens* (Nematode)

**Parasite Identification** - Parthenogenic females dissected from formalin fixed nodules are about 5 mm in length [259]; the species of *Strongyloides* present in the small intestine of the cat tends to be around 3.5 mm or less in length. Eggs from females of *Strongyloides tumefaciens* are embryonated and measure 114 to 124 long by 62 to 68 µm wide. At room temperature, fecal cultures will result in third-stage larvae with a "split tail" appearance (typical for *Strongyloides* spp.) and a filariform esophagus [260].

**Life Cycle** - The life cycle of *Strongyloides tumefaciens* is unknown. Infections are probably acquired by skin penetration or oral ingestion by third- stage larvae. Parthenogenic females are found in the large intestine in grossly visible tumor-like nodules. Eggs and larvae are also present in these nodules. Eggs hatch in the nodules and larvae are passed in the feces. No parasitic males exist.
Disease - Abdominal palpation of a cat with *S. tumefaciens* revealed a colon that was fibrotic and firm [260]. In the large intestine *Strongyloides tumefaciens* produces characteristic tumor-like nodules that are white and glistening on the mucosal surface, elevated 1 to 3 mm above the mucosa, and measure 2 to 3 mm in diameter. Also a central depression may be present. Hyperplastic nodules of crypt epithelium are present in the submucosa when examined microscopically. The nodules are surrounded by a connective tissue capsule. The mucosal epithelium covering the nodules may be degenerative and infiltrated by lymphocytes and neutrophils.

Treatment - Thiabendazole will treat cats with *S. tumefaciens* when administered orally for 3 days at 125 mg daily [260]. By day 4 post-treatment, the feces became normal and larvae were eliminated.

References

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