Elegant is the word that describes the complex and successful life cycle of the biliary trematode of cats, *Platynosomum concinnum*. First described at the beginning of this century, it has been referred to as *Dicrocoelium concinnum*, *Platynosomum planicipitus*, and *Platynosomum fastuosum*. The adult *P. concinnum* is also capable of parasitizing civet cats, opossums, ferrets, and white mice. The fluke requires three other animal hosts to complete its life cycle, and these are simultaneously indigenous only in the tropical and subtropical climates of the world (Figure 1).  

**LIFE CYCLE**

A cat excretes the fluke egg (Figure 2) to the soil. The egg is ingested by the snail *Subulina octona* or, in Hawaii, *Eulota (Bradybaena) similis*. The asexual phase of the digenetic trematode begins in the snail. In the snail’s crop, in 15 minutes or less, the miracidium extrudes through the operculum of the egg. The miracidium must quickly penetrate into the connective tissue of the snail, preferably near respiratory tissue. During the next 28 days, the miracidium develops into a mother sporocyst. This sporocyst creates numerous migratory daughter sporocysts, each laden with approximately 18 cercariae. Exactly 60 days after ingestion of the fluke egg, the daughter sporocysts begin exiting through the mantle of the snail to the soil. Daughter sporocyst emergence is influenced by high humidity and increased light.  

These factors affect snail activity and accommodate a rainy summer fluke infection season.

Terrestrial isopods, such as pill, sow, or dung bugs (Figure 3), become the second intermediate hosts by ingesting the cercariae-laden daughter sporocysts. Maturation to metacercariae apparently occurs in the isopod. The isopods are ingested by such lower vertebrates as lizards, frogs, or toads (see Third Intermediate Hosts). Insectivorous birds may also be involved. The metacercariae encyst in the gallbladder and common bile duct of the lower vertebrate, waiting for the final, definitive host to complete the life cycle. The natural predatory instinct of cats ensures the completion of the *P. concinnum* life cycle. A cat ingests the lizard (Figure 4), consuming the en-
DEFINITIVE HOST
Domesticated cat with adult fluke in biliary system

Cat eats lizard

THIRD INTERMEDIATE HOST
Lower vertebrate with encysted metacercariae

Egg with miracidium in soil

FIRST INTERMEDIATE HOST
Snail with sporocysts

Lizard eats isopod

SECOND INTERMEDIATE HOST
Isopod with metacercariae

**Figure 1**—Life cycle of Platynosomum concinnum.

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cysted metacercariae. Within hours after ingestion, the metacercariae migrate up the major duodenal papilla of the cat and into the common bile duct. Twenty percent of cats have a minor duodenal papilla; it is possible but rare for the metacercariae to migrate into this papilla and finally into pancreatic tissue via the accessory pancreatic duct. Once in the feline biliary system, metacercariae mature into egg-laying adult flukes in four to five weeks. This completes the life cycle and commences the disease process in cats.

**CLINICAL INFECTION**

In tropical and subtropical settings, the prevalence of fluke infection has been reported to be 15% to 85% of the feral cat population. The variation probably reflects the imprecise nature of fecal diagnosis and the local variation in the snail–isopod–lower vertebrate population necessary for a high fluke burden. A typical patient with asymptomatic parasitism is a young, hungry, 6- to 24-month-old, feral cat of either gender. The cat feeds on native fauna, such as lizards and birds. The fluke egg is usually an incidental finding on fecal examination. Cats with small fluke burdens (less than 125 adult flukes) are often clinically asymptomatic. There is no correlation between fluke parasitism and feline immunodeficiency virus status or feline leukemia virus status. In my practice, house cats diagnosed with *P. concinnum* infection are usually older than two years of age and have a history of hunting lizards.

Overt clinical cases of *P. concinnum* parasitism typically occur in adult outdoor or indoor-outdoor cats. Cats present with a panorama of clinical signs. The severity of the signs is proportional to the number of adult flukes and the duration of parasitism. Clinical signs may be as vague as lethargy, anorexia, unthrifty haircoat, or slight weight loss. More extreme signs in-
The hematologic changes that are evident with *P. concinnum* infection are best summed up by stating that no disease level is pathognomonic for parasitism. Compared with normal feline values, hematologic values may change. The biliary fluke induces circulating eosinophilia beginning three weeks after infection. Eosinophilia is proportional to the number of adult flukes, peaks at 8 to 18 weeks, and persists for months. A practitioner can expect a 50% to 100% increase in the number of eosinophils relative to normal feline values in the practice.

A 10% to 20% increase in serum alanine transaminase and aspartate transaminase is expected in the first four to five months of parasitism. The enzymes eventually return to normal. Serum alkaline phosphatase is usually unaffected and is normal even if hepatomegaly or jaundice is present. All liver enzymes increase when cholangiocarcinoma accompanies the parasitism. In rare cases, pancreatic enzymes may be elevated.

Total serum bilirubin values depend on the degree of biliary tract inflammation, hyperplasia, and resultant bile stasis. Total biliary values as high as 20 mg/dl are possible. Bilirubinuria accompanies jaundice (Figure 5). In early cases of parasitism, before severe periductal fibrosis occurs, there are no signs of anemia, hypoalbuminemia, altered blood ammonia, or altered renal function.

Ultrasonographic examination of a parasitized cat might demonstrate distention of the gallbladder and/or common bile duct. Hepatomegaly and distention of the hepatic biliary tree may be evident. Radiography might demonstrate hepatomegaly, which is usually palpable.

Liver biopsy and necropsy findings exhibit consistent hepatic disease. Inflammation and edematous hyperplasia of the bile ducts with infiltration of eosinophils and plasma cells are present. Also apparent is an increase in the size and tortuosity of the common, cystic, and hepatic ducts (Figure 6). The bile is thickened and may be inspissated within the gallbladder. The liver may be enlarged and yellow. Viscera are jaundiced in proportion to the serum bilirubin level. Trematodes and eggs may be seen anywhere in the biliary system. Pancreatic atrophy is evident in cases in which the fluke has migrated into the minor duodenal papilla and accessory pancreatic duct.

The flukes are easily visible and readily identifiable (Figure 7). Specimen flukes must be allowed to die in
Formalin Ether Sedimentation Technique

1. Suspend one gram of feces in 25 milliliters of 0.9% saline solution.
2. Filter through fine mesh (e.g., a tea strainer or gauze).
3. Centrifuge filtrate for five minutes at 1500 rpm, and discard supernatant.
4. Suspend centrifuged pellet in seven milliliters of 10% buffered formalin for 10 minutes.
5. Layer three milliliters of cold ether on the suspension, and shake well for 60 seconds.
6. Centrifuge mixture for three minutes at 1500 rpm, and discard supernatant.
7. Resuspend pellet in 0.9% saline solution, and examine for eggs. The number is equivalent to eggs per gram.

Older cats that have been parasitized for extended periods present with emaciation, cachexia, and liver cirrhosis. Chronic cholangiohepatitis is replaced by periductal fibrosis that affects connective tissue. An affected cat can have hundreds of adult flukes in the biliary system. I have seen four cases of cholangiocarcinoma in parasitized cats (Figure 9). The cats were middle-aged (6 to 11 years old) and had gross hepatomegaly and elevated serum alanine transaminase, aspartate transaminase, and alkaline phosphatase levels. All of the patients were markedly icteric and had total bilirubin levels exceeding 8 mg/dl. The four cases represent less than 2% of all parasitized cats seen. Feral cats usually have a short life span and are seldom examined when ill; the true incidence of carcinoma may be much higher. In humans, the liver fluke Opisthorchis induces hepatic carcinoma in 55% of the individuals that it parasitizes. 17

The adult flukes stimulate minimal immune response, which is detected by the presence of humoral antibodies in the bile. 14 The flukes do not suppress the function of T or B lymphocytes. 9 Immune suppression is not a primary ailment but can be secondary to debilitation.

TREATMENT AND PREVENTION

The cesticidal drug praziquantel is the only effective agent available for treating patients with P. concinnum. The drug is administered parenterally at 20 mg/kg (four times the recommended dose level of 5 mg/kg for cats). 18 Extralabel use with client consent is necessary. As a precaution, treated cats should be observed. The action of praziquantel on flukes is unknown. The drug is excreted in the bile; perhaps trematodes, like cestodes, lose their ability to resist digestion by the host.

After treatment, fluke eggs may be evident in the feces for as long as nine weeks because of incomplete killing of the trematode or the sequestering of eggs in the biliary system. 18 Ideally, praziquantel therapy is repeated at 12-week intervals. I have also used a three-day oral praziquantel regimen. Using the canine dose rate (10 mg/kg), a cat is treated for three days. The results compare favorably and should involve a reversal of signs in mildly to moderately ill cats. Vomiting subsides, appetite returns, weight is restored, and jaundice resolves. The cat returns to normal activity, which often includes more lizard hunting.

Supportive treatment of parasitized cats must be tailored to the signs of the verminous cholangiohepatitis. The attending veterinarian must address nutritional needs and concomitant bacterial, viral, or parasitic infection. Associated bacterial infections, such as with Yersinia, may be worse than the effects of the trematode. 19 I administer a copious amount of 0.9% saline solution for rehydration and, if there is ob-
structive cholangitis, dexamethasone (1 mg/kg) parenterally twice daily. The antiinflammatory agent usually restores patency to the bile duct, alleviating the jaundice by eliminating bile through the feces.

Preventing infection in an attempt to maintain a healthy cat is a challenge. Avoidance of lower vertebrate hosts is essential and is more easily accomplished in house cats. Outdoor cats are thus more likely to be reinfected and progress to irreversible liver disease.

CONCLUSION

*Platynosomum concinnum* is a parasite of tropical and subtropical climates worldwide. A cat living in or traveling through such climates could become parasitized by ingesting an infected invertebrate host. The modern mobility of companion cats explains the occasional reports of infections outside the tropics.\(^{20,21}\) It also places all veterinarians on alert for these elusive trematodes in feline patients that travel.

The primary source of infection in cats is the ubiquitous tropical lizard. Frogs also may be a source. The *Bufo* toad (*Bufo marinus*) has been incriminated as a potential fluke source.\(^{3,8-10}\) I doubt this hypothesis. In nature, the *Bufo* species are well protected by their cutaneously excreted bufotoxin. Unlike dogs, cats evidently shun interaction with *Bufo* toads.

Definitive diagnosis of *P. concinnum* infection is accomplished by finding operculated fluke eggs in feces. Serial fecal examinations and additional clinical pathologic evaluation may be needed in occultly parasitized or severely ill animals. Praziquantel at extra-label doses is the preferred treatment agent. Preventing parasitism in companion cats means housing cats indoors and avoiding contact with third intermediate hosts, such as during vacations to Florida or the Caribbean. Outdoor or feral cats that live in the subtropics may require quarterly treatment with praziquantel.

In cats with evidence of eosinophilia, hepatomegaly, and jaundice, veterinarians should consider the possibility of fluke infection. Close inspection of cats with liver carcinoma may demonstrate underlying *P. concinnum* parasitism.

### About the Author

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