

Baylisascariasis

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Importance

Baylisascaris procyonis, an intestinal nematode of raccoons, can cause severe neurological and ocular signs when its larvae migrate in humans, other mammals and birds. Although clinical cases seem to be rare in people, most reported cases have been serious and difficult to treat. Severe disease has also been reported in other mammals and birds. Other species of *Baylisascaris*, particularly *B. melis* of European badgers and *B. columnaris* of skunks, can also cause neural and ocular larva migrans in animals, and are potential human pathogens.

Etiology

Baylisascariasis is caused by intestinal nematodes (family Ascarididae) in the genus *Baylisascaris*. The three most pathogenic species are *Baylisascaris procyonis*, *B. melis* and *B. columnaris*. The larvae of these three species can cause extensive damage in intermediate/paratenic hosts: they migrate extensively, continue to grow considerably within these hosts, and sometimes invade the CNS or the eye. Their larvae are very similar in appearance, which can make it very difficult to identify the causative agent in some clinical cases. Other species of *Baylisascaris* including *B. transfuga*, *B. devos*, *B. schroeder* and *B. tasmaniensis* may also cause larva migrans. In general, the latter organisms are smaller and tend to invade the muscles, intestines and mesentery; however, *B. transfuga* has been shown to cause ocular and neural larva migrans in some animals.

Species Affected

Raccoons (*Procyon lotor*) are usually the definitive hosts for *B. procyonis*. Other species known to serve as definitive hosts include dogs (which can be both definitive and intermediate hosts) and kinkajous. Coatimundis and ringtails, which are closely related to kinkajous, might also be able to harbor *B. procyonis* adults.

More than 130 species of mammals and marsupials are known to act as intermediate, paratenic or dead-end hosts for *B. procyonis*. While most infections have been documented in rodents (mice, guinea pigs, prairie dogs and other species), lagomorphs and primates, symptomatic cases have also been reported in lemurs, Australian marsupials, opossums, porcupines, woodchucks and fruit bats, as well as in carnivores such as dogs, foxes, sea otters and American badgers (*Taxidea taxus*). *Baylisascaris* larva migrans has not been reported in livestock, and experimental infections with *B. procyonis* in sheep, pigs or goats resulted in little migration of the larvae. However, one case of larva migrans occurred in a newborn lamb that had been infected before birth. Birds are highly susceptible; *B. procyonis* can affect at least 40 avian species. Clinical cases have been reported in chickens, bobwhite quail, commercial pheasants, cockatiels, cockatoos, macaws, parrots and other species. *B. procyonis* infections have not been reported in poikilotherms.

Other species of *Baylisascaris* may also cause neural larva migrans in intermediate hosts. The definitive hosts for these organisms are European badgers (*Meles meles*) for *B. melis* and skunks for *B. columnaris*, as well as bears (*B. transfuga*), fishers and martins (*B. devosi*), pandas (*B. schroederi*), Tasmanian devils (*B. tasmaniensis*) and other species. *B. schroederi* and *B. transfuga* can cause disease in their definitive hosts, as well as in intermediate hosts.

Zoonotic Potential

Although *B. procyonis* is the only species that has been reported in humans at present, other *Baylisascaris* species might also infect people.

Geographic Distribution

Raccoons, which are the definitive hosts for *B. procyonis*, are native to the Americas, where they can be found from Canada to Panama. They were introduced into Europe, the former U.S.S.R. and Asia for the commercial fur trade, and into Japan as pets, and have become naturalized in some of these areas. *B. procyonis* is maintained in raccoons in the United States, Canada and Europe. It has also been found in many raccoons kept as pets or zoo exhibits in Japan. Although surveys of

feral raccoons in Japan have not detected this organism, it is possible that some pets released into the wild were infected. Human infections with *B. procyonis* have been documented most often in North America, but they can occur wherever infected raccoons are found.

Other *Baylisascaris* species have been less well studied, but probably occur in most areas where their definitive hosts are found.

Transmission and Life Cycle

In the definitive hosts, mature *B. procyonis* reside in the intestines, and release unembryonated eggs into the feces. This organism produces very large numbers of eggs; each worm is estimated to lay up to 179,000 eggs per day, and raccoons carry an average of 43-52 worms. The worm burden seems to be greater in juvenile raccoons than adults. The development of *B. procyonis* eggs to the infective stage, containing second stage larvae, occurs in the environment. This can take as little as 11 to 14 days under optimal conditions; however, it is estimated to take 2 to 4 weeks or longer in most cases. *B. procyonis* eggs are very resistant to environmental conditions, especially in moist soil. Although they can be killed eventually by extreme heat and dryness, the eggs survive harsh winters, and under some conditions, they can remain viable for years.

Intermediate hosts become infected by ingesting embryonated eggs from the environment. Small birds and mammals are often infected when they forage for undigested seeds, grain and other foods at sites where raccoons defecate. Children may ingest the eggs when they eat dirt or place contaminated objects in their mouths. *B. procyonis* eggs also adhere readily to fur and various fomites. In an intermediate host, the eggs hatch in the small intestine, penetrate the intestinal wall and are carried in the bloodstream through the liver to the lungs, where they are eventually distributed via the blood to various organs. *B. procyonis* larvae migrate extensively in tissues, while continuing to grow. In mice, approximately 5-7% of *B. procyonis* larvae are estimated to enter the central nervous system (CNS). These larvae can cause considerable damage, both from mechanical damage during migration and from the inflammatory reaction they stimulate. Larvae in the eye can damage the retina and other structures. Eventually the larvae encyst, mainly in the connective tissues and muscles. Intermediate hosts can transmit the infection to definitive hosts only if their tissues are eaten. Larvae have been reported to survive in tissues for several days.

Definitive hosts can be infected either by ingesting eggs from the environment, or by eating intermediate hosts. The latter route seems to be more common in adult raccoons. Young raccoons are thought to become infected mainly from embryonated eggs in their environment. When a definitive host ingests embryonated eggs, the second stage larvae hatch in the intestines, then develop for a time in the

intestinal wall before completing their development in the lumen. Third stage larvae from intermediate hosts mature into adult worms in the intestine without undergoing further migration. As a result, the prepatent period is shorter when larvae are ingested (32-38 days in raccoons), compared to eggs (50-76 days in raccoons). Extraintestinal migration has been seen in dogs, but it does not seem to occur frequently in raccoons (although larvae have occasionally been found in tissues). Transplacental and transmammary transmission have not been studied extensively in definitive hosts. However, there was no evidence of intrauterine transmission to newborn raccoons in one study.

Other species of *Baylisascaris* have similar life cycles, but use different species as their definitive hosts.

[Note on terminology: The “intermediate” hosts described above may be called either intermediate or paratenic hosts by different sources. Like paratenic (transport) hosts, they are not essential for the completion of the parasite’s life cycle, as raccoons can also be infected from eggs. However, further development of a parasite does not take place in a paratenic host, and *Baylisascaris* larvae mature from the L2 to the L3 stage in rodents and some other hosts.]

Disinfection

B. procyonis eggs are highly resistant to disinfectants. These eggs can become embryonated even in a weak formalin solution. They are also very resistant to inactivation in the environment. Desiccation and heat, such as strong sunlight on an exposed surface or heat in a dry attic, will eventually destroy the eggs, but it is not known how long this will take. One group found that, under experimental conditions, *B. procyonis* eggs were no longer viable if they were heated to 62°C (144 °F) or desiccated for 7 months. However, the eggs remained viable after freezing at -15°C (5°F) for 6 months, and also survived desiccation for 6 months.

High heat (e.g., a propane torch, boiling water or incineration) is usually used to decontaminate fomites. Boiling lye water has also been recommended. A xylene-ethanol mixture has been used after the solid waste was removed. Removal of the top few inches of the soil is sometimes necessary. Eggs can be washed off surfaces with a 1% sodium hypochlorite solution, which stops them from sticking; however, the eggs are not killed by this treatment.

Infections in Animals

Incubation Period

The incubation period is thought to be at least a week, and probably 2 to 4 weeks or longer, in intermediate hosts. Experimentally infected mice developed clinical signs in 7 to 20 days.

Clinical Signs

B. procyonis in raccoons and dogs (definitive hosts)

Raccoons infected with *B. procyonis* are usually asymptomatic, but massive infections in young animals can cause intestinal obstruction. Intestinal infections in dogs have generally been found during routine fecal examination, and are unlikely to cause significant clinical signs.

B. procyonis in intermediate hosts

Nonspecific signs such as lethargy, depression, and a rough hair coat or ruffled feathers may be seen in some animals. Neurological disease has been reported in most diagnosed cases, with diverse presentations such as circling, rolling, torticollis, ataxia, tremors, progressive weakness, paralysis, hypertonia, extensor rigidity, seizures or dysphagia. Birds may have difficulty perching or become unable to fly. Visual defects including blindness may also be seen. In some animals, the clinical signs develop acutely and progress quickly. One puppy, for example, became recumbent 48 hours after the onset of ataxia. Other cases may be insidious and slowly become worse. Some birds have had clinical signs that progressed over weeks. If the larvae become encapsulated, the clinical signs sometimes stabilize. A waxing and waning course has also been seen, and might be caused by larval encystment followed by reinfection from other eggs in the environment.

Other *Baylisascaris* spp.

B. melis of European badgers and *B. columnaris* of skunks can cause extensive damage in their intermediate hosts, and sometimes invade the CNS or the eye. *B. melis* has been reported to cause CNS signs in mice, ground squirrels and rabbits, and ocular larva migrans in mice. Skunks were associated with one outbreak of baylisascariasis at a zoo, suggesting that *B. columnaris* may have been responsible. Other species of *Baylisascaris* are typically smaller and tend to invade the muscles, intestines and mesentery; however, neurological signs or ocular disease are possible. *B. transfuga* and *B. schroederi* have been reported to cause visceral, neural and/or ocular larva migrans in some experimentally infected intermediate hosts, such as rodents. These two organisms have also been linked to illness in their definitive hosts. *B. schroederi* can cause visceral larva migrans, as well as intestinal obstruction due to large worm burdens, in giant pandas (*Ailuropoda melanoleuca*). Both forms of disease can be fatal. Heavy infestations of *B. transfuga* are thought to result in illness or death in some bears, although few studies have been published. In one report, *B. transfuga* was associated with granulomatous peritonitis in a bear cub.

Post Mortem Lesions [Click to view images](#)

Nematodes may be found in the intestines of the definitive host. Mature *B. procyonis* are large, tan,

roundworms. The female is 20 to 22 cm long, approximately twice the size of the male.

Migrating larvae can cause hemorrhagic or necrotic lesions and tracks, as well as granulomas, in any tissue where they are found. Extraneural granulomas are found less often in birds than mammals; the larval burden in avian species can be very low, and the lesions may be limited to the brain. In the CNS, there may be focal areas of palpable softening and discoloration, as well as small multifocal hemorrhages. However, fatal cases with only microscopic lesions, and no gross lesions, have been seen in some birds. Microscopic lesions can include multiple tracts with debris, glitter cells, neuronal degeneration, gliosis, vascular rupture with hemorrhage, malacia and eosinophilic and granulomatous inflammation, as well as perivascular cuffing. Microscopic CNS lesions are not always associated with significant inflammation in birds; in some cases, the lesions were mainly degenerative and/or necrotizing, with only mild inflammation. Larvae may be found both within lesions and in areas of the brain that appear to be normal. In some cases, they may no longer be present in the CNS.

Diagnostic Tests

Intestinal infections in raccoons and dogs can be diagnosed by identifying the eggs in feces, or worms in the feces or vomitus. Fecal samples should be collected for at least 3 days before concluding that a raccoon is not infected. Eggs are more readily identified in fresh feces than from environmental samples. *B. procyonis* eggs are similar to *Toxocara* spp. eggs, but they are darker and somewhat smaller. They also have a finely granular surface, compared to the coarsely pitted surface of *Toxocara* eggs. However, these species can be readily confused unless they are examined very carefully.

Baylisascaris larva migrans is difficult to diagnose in live animals. A presumptive diagnosis can be made based on a history of exposure to raccoons or other definitive hosts, combined with the clinical signs. Eosinophilia in the cerebrospinal fluid (CSF) and blood are supportive in mammals, but peripheral eosinophilia does not necessarily occur in birds with neural larva migrans. ELISAs might be helpful in mammals, and imaging studies may be suggestive in conjunction with other tests.

A definitive diagnosis depends on the identification of the parasite within tissues by biopsy, or more often, in CNS samples taken at necropsy. However, parasite larvae can be difficult to identify within tissues, and misidentification is common. *Baylisascaris* larvae can be differentiated from some other nematodes such as *Toxocara* spp. by their large size (up to 80 µm in diameter and up to 1900 µm in length) and their morphology. The third stage larvae of *B. procyonis* cannot be differentiated from *B. columnaris* or *B. melis*. Epidemiological evidence, such as a history of exposure to raccoons, but not to skunks or badgers, can be suggestive.

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Polymerase chain reaction (PCR) assays for *Baylisascaris* have been published, but are not currently used in clinical laboratories.

Treatment

Definitive hosts can be treated with anthelmintic drugs; most common anthelmintics used in dogs and cats are effective against *B. procyonis* in raccoons. Piperazine, pyrantel, ivermectin, moxidectin, albendazole, fenbendazole and flubendazole have been used in various studies. One study reported that monthly heartworm/intestinal worm preventive tablets containing milbemycin oxime were able to treat patent infections in dogs, although one treatment was not always sufficient to clear all of the worms.

Neural larva migrans might be treated with anthelmintic drugs such as albendazole, mebendazole or other drugs that penetrate well into the CNS, but the prognosis is guarded. Corticosteroids have been used concurrently to control inflammation, which contributes to the pathology and can be exacerbated by the death of the larvae. Supportive treatment is given as appropriate. In one zoo, lemurs that were treated long-term with albendazole gradually improved.

Prevention

In intermediate hosts, the risk of infection can be decreased by avoiding contact with raccoons, other definitive hosts and their feces. Raccoons should be discouraged from visiting homes and farms. Anthelmintic baiting has been explored in wild raccoons, and appears to be promising. Infections are difficult to prevent completely in pets allowed outdoors, as eggs can remain viable for long periods in the environment. In dogs, monthly heartworm/nematode preventives appear to decrease the risk of intestinal infection with *B. procyonis*. In high-risk areas, dogs that are not on these preventives should receive regular fecal examinations.

In zoos and other facilities, housing for intermediate hosts should be designed to minimize exposure to raccoons, skunks and other definitive hosts. Captive raccoons and skunks should be kept in dedicated cages that can be cleaned, if necessary, with the harsh methods required to destroy *Baylisascaris* eggs. They should be tested regularly and dewormed when necessary, and they should not be fed wild animals that might carry larvae. Newly acquired definitive hosts should be quarantined and dewormed. Once contamination has occurred, the eggs can be difficult to remove completely. Intermediate hosts in exhibits are sometimes treated prophylactically with anthelmintics. Animals with recent exposure might be treated with drugs active against larvae in the tissues (e.g., albendazole) to prevent clinical signs.

Morbidity and Mortality

B. procyonis is widespread in raccoons, particularly young animals, in North America. In the U.S., infected

raccoons seem to be particularly common in the Mid-Atlantic, Northeast and Midwest and along the Pacific coast, but they can be found throughout the country. The prevalence of infection varies widely, but can be greater than 60% in some areas of North America. In the 1990s, *B. procyonis* was found in 8% of pet raccoons and 40% of zoo raccoons in Japan. It is also reported to be common among wild raccoons in Germany. Significant illnesses or deaths have not been reported in infected raccoons.

B. procyonis is occasionally reported in dogs, although cases seem to be infrequent. There are concerns that infected dogs might increase the risk of human exposure, both because they are in close contact with people and because dogs defecate indiscriminately rather than using localized sites as raccoons do.

The morbidity and mortality rates in intermediate hosts are still poorly understood. Birds that forage on the ground are at an increased risk of infection, as is any animal exposed to raccoons. Clinical cases, particularly those with CNS signs, are often serious and can be fatal. One outbreak in an aviary of 35 cockatiels (*Nymphicus hollandicus*) eventually affected 34 of the birds, and all but one affected bird died. *B. procyonis* seems to cause significant morbidity and mortality in some wild populations, such as the Allegheny woodrat (*Neotoma magister*) and the white-footed mouse (*Peromyscus leucopus*), in parts of North America.

Infections in Humans

Incubation Period

The incubation period in humans is uncertain, but neural larval migrans may occur as soon as 2 to 4 weeks after ingestion of the eggs.

Clinical Signs

Relatively few cases of baylisascariasis have been described in humans, and this disease is still incompletely understood. The symptoms are expected to vary with the location and number of the migrating larvae. Most reported cases have involved the CNS and/or eye.

Neural larva migrans occurs when the parasites migrate through the CNS. The initial signs may be mild, with subtle behavioral changes, lethargy, somnolence or irritability, weakness, speech defects and/or mild changes in vision, but they may rapidly become severe. A variety of neurological signs, including coma, can be seen. Seizures are common and can be severe. Ocular signs, including blindness, also occur in many cases. Some cases of neural larva migrans are fatal, and many surviving patients have been left with serious neurological defects despite treatment. However, a few cases with better outcomes, including apparent full recovery in one child with relatively mild symptoms, have been reported.

Ocular larva migrans has been reported more frequently than neural larva migrans, and can occur without

neurological signs. Inflammatory and degenerative changes are mainly seen in the retina and optic disk, usually only in one eye. The clinical signs may include transient obscuration of the vision, photophobia, other signs of diffuse unilateral subacute neuroretinitis (DUSN) and loss of vision. Some visual defects can be permanent.

Visceral larva migrans has not been well described for *B. procyonis*, but nonspecific signs such as low-grade fever, nausea and lethargy can be seen with most parasites. Invasion of the liver can result in hepatomegaly, and migration through the lung may cause symptoms of pneumonitis. In one fatal case of *B. procyonis* larva migrans, an eosinophilic mass resembling a tumor was found in the heart. A macular rash, seen mainly on the face and trunk, has been described. Serological evidence suggests that subclinical cases might also occur.

Diagnostic Tests

The diagnosis of baylisascariasis is difficult in live patients; there is no widely available, non-invasive definitive test. In ocular larva migrans, an ophthalmoscopic examination may occasionally reveal large, motile larvae in the retina, as well as chorioretinitis and other signs of DUSN. Unless a brain biopsy is done and a larva is found, antemortem diagnosis of neural larva migrans usually depends on serology, with supportive evidence from other tests. In this form of baylisascariasis, antibodies to *Baylisascaris* can be found in serum and CSF; a rising titer is usually seen. An ELISA, indirect immunofluorescence and immunoblotting (Western blotting) have been developed to detect anti-*Baylisascaris* antibodies. These serological assays are not commercially available, but they may be provided by university research laboratories or public health agencies. Imaging techniques and encephalography provide supportive evidence and help rule out other causes. The complete blood count (CBC) and CSF examination suggest a parasitic infection. The presence of *Baylisascaris* larvae in the eye is also suggestive in cases with neurological signs. Epidemiological evidence, such as a history of exposure to raccoons but not skunks or badgers, can support the diagnosis.

A definitive diagnosis can also be made retrospectively from CNS samples taken at autopsy, if larvae are identified.

Treatment

The optimal treatment of baylisascariasis is still uncertain. Albendazole is currently thought to be the drug of choice. This drug is protective in animal models if eggs have been ingested, but symptoms have not yet developed. It is recommended that albendazole be started as early as possible in suspected clinical cases. Corticosteroids are given concurrently to suppress inflammation caused by the death of the larvae, as well as to dampen the existing inflammatory response. Other supportive therapy may also be given. Laser photocoagulation, systemic corticosteroids and other therapies have been used in ocular larva migrans.

In many cases, significant damage has already occurred by the time treatment is begun, and improvement is not seen. The best chance of recovery is expected with a very early diagnosis and treatment.

Prevention

The risk of infection with *B. procyonis* can be decreased by avoiding contact with raccoons and their feces. Raccoons should not be kept as pets, especially in homes with young children. All captive raccoons should be examined regularly for *B. procyonis* eggs, and dewormed if necessary. Wild animals should not be fed or otherwise encouraged to visit areas around homes and playgrounds. Access to attics or basements should be prevented, and any accessible food or garbage should be kept in raccoon-proof containers. Raccoons can also be attracted to ponds, bird feeders and vegetable gardens. Sand boxes should be covered when not in use, to prevent raccoons from defecating in them. Cleaning up brush may discourage them from making a den on the property.

Raccoons tend to use “latrines” where they regularly defecate. Latrines are often found at the base of trees, in the forks of trees, or on raised horizontal surfaces such as fallen logs, stumps, large rocks, woodpiles, decks and rooftops. Raccoons will also defecate in attics, garages and haylofts if they have access. Their feces is typically dark and tubular, with a particularly pungent odor. It often contains undigested seeds, fragments of corn or other foods, bones and/or shells.

Raccoon feces and contaminated material at sites where people would be expected to contact them (e.g., decks and patios) should be removed and burned, buried or sent to a landfill, preferably before the eggs can become embryonated. Care should be taken to avoid getting the eggs and feces on hands and clothes; gloves and protective clothing should be worn, and the hands should be washed immediately afterward with soap and water. A face mask may be helpful in dry conditions to prevent the inhalation of other organisms that are present in feces, and to prevent accidental contamination of the mouth. Decks, patios and other surfaces may be treated with boiling water, steam cleaned, or flamed with a propane flame-gun (with proper precautions on appropriate surfaces). Incineration of contaminated objects is also effective. Ordinary disinfectants are ineffective against these eggs, but a xylene-ethanol mixture has been used in some circumstances after solid waste has been removed. The removal of the top few inches of soil may sometimes be necessary to decrease contamination. In many cases, *Baylisascaris* eggs may remain despite rigorous cleanup measures.

Exposure to *Baylisascaris* spp. is difficult to prevent completely, as the infective eggs can survive for long periods in contaminated soil. Good hygiene, especially hand washing after outdoor play or contact with animals, including dogs, should be encouraged. Children should be

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stopped from eating dirt, and taught not to put objects into their mouths. Developmentally disabled individuals should be supervised when they are in areas that might contain raccoon latrines. Prophylactic albendazole has been used in people exposed to raccoon latrines or other sources of eggs.

Morbidity and Mortality

Clinical baylisascariasis appears to be rare in humans. The exact number of cases is uncertain, but more than a dozen cases of probable or confirmed neural larva migrans have been published, and at least two dozen additional unpublished infections are known. This disease might be underdiagnosed, if the symptoms are usually attributed to other causes. It is also possible that severe neurological disease occurs only after exposure to large numbers of eggs, or in hosts who are unusually susceptible.

Neural larva migrans tends to be seen in infants and young children with a history of exposure to raccoons or their feces. Young children are more likely to eat dirt or put contaminated fingers, soil or other fomites into their mouths. The small size of their brain may also contribute to the severity of the damage. Some cases of neural larva migrans have also been seen in older, developmentally challenged individuals or adults with neurological defects, particularly those who have a history of pica or eating dirt. Ocular larva migrans without neurological signs is usually reported in otherwise healthy adults. In this form of baylisascariasis, there may be no history of exposure to raccoons or the exposure may be incidental, and it is possible that it can be caused by small numbers of eggs. People who hunt, trap, perform taxidermy and handle wildlife are expected to have an increased risk of exposure. There is some evidence that subclinical visceral larva migrans may also occur. Seropositive but asymptomatic individuals have been reported in North America and Germany. One study reported that 30 of 389 children (8%) in the Chicago area had antibodies to *Baylisascaris* spp., but no history of disease.

Neural larva migrans is usually a severe disease. Several cases have been fatal, and most survivors have had permanent neurological damage. In one case, however, a small number of larvae were found in the brain of an elderly woman who died of unrelated causes, and had no recent clinical signs attributed to this organism (mild signs might have been masked by pre-existing Alzheimer's disease). Treatment was apparently ineffective in many clinical cases, possibly because irreversible damage was already present. One apparent full recovery was reported in child who had relatively mild neurological signs and was treated with albendazole, corticosteroids and mannitol. Whether the outcome was due to the treatment, host factors or the relatively mild case is unknown. In another recent case, a toddler had a relatively good outcome, with mild to moderate residual neurological deficits, after an early clinical diagnosis and aggressive empirical treatment with albendazole and steroids.

Internet Resources

- Centers for Disease Control and Prevention (CDC)
<http://www.cdc.gov/ncidod/dpd/parasites/baylisacaris/default.htm>
- Centers for Disease Control and Prevention (CDC) Parasitic Diseases Branch
http://www.cdc.gov/parasites/health_professionals.html
- Companion and Exotic Animal Parasitology
http://www.ivis.org/advances/Parasit_Bowman/toc.asp
- Department of Comparative Pathobiology at Purdue University
<http://www.vet.purdue.edu/cpb/>
- International Veterinary Information Service (IVIS)
<http://www.ivis.org>
- The Merck Manual
<http://www.merckmanuals.com/professional/index.html>
- The Merck Veterinary Manual
<http://www.merckmanuals.com/vet/index.html>
- National Reference Centre for Parasitology (NRCP Canada)
<http://www.nrcp.ca/>

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