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Baylisascariasis

Raccoon Roundworm

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Overview

- Organism
- History
- Epidemiology
- Transmission
- Disease in Humans
- Disease in Animals
- Prevention and Control
- Actions to Take

In today's presentation we will cover information regarding the organism that causes baylisascariasis and its epidemiology. We will also talk about the history of the disease, how it is transmitted, species that it affects (including humans), and clinical and necropsy signs observed. Finally, we will address prevention and control measures, as well as actions to take if baylisascariasis is suspected.

[This photo shows a raccoon, *Procyon lotor*. Source: U.S. Fish and Wildlife Service National Digital Library]

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THE ORGANISM

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The Organism

- Intestinal nematode
  - Family Ascarididae
  - Genus *Baylisascaris*
    - *B. procyonis* (raccoons)**
      - **Zoonotic**
    - *B. melis* (European badgers)
    - *B. columnaris* (skunks)
- Extensive tissue migration

Baylisascariasis is caused by intestinal nematodes (family Ascarididae) in the genus *Baylisascaris*. The three most pathogenic species are *Baylisascaris procyonis*, a parasite of raccoons (*Procyon lotor*), *B. melis*, which occurs in European badgers (*Meles meles*), and *B. columnaris*, which is found in skunks and was, at one time, thought to be the same species as *B. procyonis*. The larvae of these three species can cause extensive damage in their intermediate/paratenic hosts: they migrate extensively, continue to grow considerably within these hosts, and sometimes invade the CNS or the eye. As of 2009, *B. procyonis* is the only species reported to cause disease in both humans and animals; however, *B. melis* and *B. columnaris* may be of veterinary importance, particularly in zoo animals and exotic pets.

[This photo shows several adults *Baylisascaris procyonis* roundworms from a raccoon. Source: CDC DPDx Image Library]

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HISTORY

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History

- 1951: First identified in Europe
  - Raccoons
  - Classified as *Ascaris procyo*nis
- 1933: First identified in the U.S.
  - Raccoons, New York
- 1968: Reclassified
  - *Baylisascaris procyonis*
- 1984: Recognized as helminth

The organism was first identified in raccoons in Europe in 1951. Initially, the organism was classified as *Ascaris procyonis*. In 1933, the organism was isolated for the first time in the United States from raccoons in New York. In 1986, the organism was reclassified as *Baylisascaris procyonis;* however, it was not recognized as a helminth infection until 1984, when *Baylisascaris* was identified as the cause of fatal eosinophilic meningoencephalitis in an infant.

[Source: *Baylisascaris procyon*is The raccoon roundworm. Available at: http://www.stanford.edu/class/humbio103/ParaSites2002/baylisascariasis/paraSite%20finished%20draft.html#references]

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EPIDEMIOLOGY

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Species Affected

- Raccoons
  - Definitive hosts
- Worm burden
  - Infected raccoons carry 43 to 52 worms
  - One worm may produce 179,000 eggs per day
  - Highest in juvenile raccoons

Raccoons are normally the definitive hosts for *B. procyonis*. Mature worms are found 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 to 52 worms. Higher worm burdens are usually found in juvenile raccoons than adults. The development of *B. procyonis* eggs to the infective stage, containing second stage larvae, occurs in the environment. Under optimal conditions, this process can take as little as 11 to 14 days; however, under natural conditions, it is estimated to take 2 to 4 weeks or longer.

[Photo: A raccoon. Source: U.S. Fish and Wildlife Service National Digital Library]

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Populations at Risk

- Exposure to raccoon environments
- Young children or developmentally disabled
  - Especially those with pica
- Occupational exposure
  - Hunters, pest control workers, trappers, wildlife handlers

The major factor in human cases is exposure to raccoon environments. Severe *B. procyonis* infections tend 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. Some cases have also been seen in older, developmentally challenged individuals, particularly those who have a history of pica or eating dirt. People who hunt, trap, perform taxidermy and handle wildlife are expected to have an increased risk of exposure.

[Photo: A child playing in dirt. Source: CDC Public Health Image Library]

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Geographic Distribution

- Indigenous in raccoons
  - United States
    - Middle Atlantic
    - Midwest
    - Northeast
  - Canada
  - Europe

Raccoons 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 known to be indigenous 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 the U.S.

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TRANSMISSION

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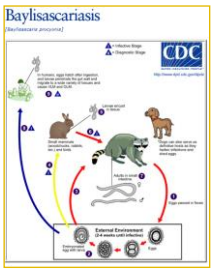
Life Cycle

Baylisascaris procyonis

- Raccoons
  - Definitive host
- Humans
  - Accidental host



Baylisascaris procyonis completes its life cycle in raccoons, with humans acquiring the infection as accidental hosts (dogs serve as alternate definitive hosts, as they can harbor patent infection and shed eggs). This graphic, from the CDC, shows how unembryonated eggs are shed in the environment and ingested by raccoons, birds, and other mammals including humans. In raccoons, the larvae develop into egg-laying adult worms in the small intestine. In humans, larvae migrate in the tissues causing extensive damage.

[Photo: The life cycle of a *Baylisascaris procyonis* roundworm. Source: CDC DPDx at http://www.dpd.cdc.gov/DPDx/HTML/Baylisascariasis.htm]

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Transmission in Humans

- Humans are accidental hosts
- Ingestion of eggs
  - Dirt
  - Animal fur
  - Fomites
- Persist in environment
- Resistant to disinfection

Humans become accidentally infected when they ingest infective eggs from the environment; typically this occurs in young children playing in the dirt. *B. procyonis* eggs also adhere readily to fur and various fomites. They persist in the environment and can be resistant to disinfection. Infected humans do not transmit *B. procyonis* to others.

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Transmission in Animals

- Ingestion of eggs
  - Young raccoons and dogs
- Ingestion of larvae in intermediate hosts
  - Most common route for adult raccoons
  - Common route for other animals

Raccoons and other definitive hosts, including dogs, can be infected either by ingesting eggs from the environment, or by eating larvae in the tissues of an intermediate host. Ingestion of the intermediate host is the most common route in adult raccoons. In contrast, young animals typically become infected from embryonated eggs on their mother's fur and in their environment. 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.

[Photo: Raccoon in a tree. Source: U.S. Fish and Wildlife Service National Digital Library]

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DISEASE IN HUMANS

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Disease in Humans

- Incubation period uncertain
- Symptoms variable
  - Location of larvae
  - Number of migrating larvae
- Visceral larva migrans
  - Nonspecific signs
  - Hepatomegaly
  - Pneumonitis

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. The symptoms vary with the location and number of the migrating larvae. 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. A macular rash, seen mainly on the face and trunk, has also been reported. Subclinical cases might also occur.

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Disease in Humans

- Neural larva migrans
  - Parasite migration through CNS
  - Initial signs mild
  - Seizures common
  - Ocular signs may also occur
- Some cases are fatal
- Serious neurological deficits may persist despite treatment

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 can rapidly become severe. A variety of symptoms including ataxia, paresis or paralysis, developmental regression, tremors, torticollis, nystagmus and coma have been reported. 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 as of 2009, almost all surviving patients have been left with serious neurological defects despite treatment. In one recent case, however, a child developed relatively mild symptoms (headache, right arm pain, vomiting, mild upper extremity tremors and dysmetria, progressing to ataxia) and appeared to recover completely.

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Disease in Humans

- Ocular larva migrans
  - More frequent than neural
  - Inflammatory and degenerative changes
    - Retina, optic disk
    - Usually only in one eye
  - Obscured vision, photophobia, loss of vision
  - Visual defects may be permanent

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.

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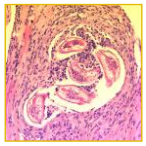
Diagnosis

- Ante-mortem diagnosis difficult
  - Serology
  - Ophthalmoscopic exam
- Definitive diagnosis
  - Brain/CNS biopsy
  - Larvae identification difficult
- PCR



The diagnosis of baylisascariasis is difficult in live patients; there is no widely available, non-invasive definitive test. Antemortem diagnosis usually depends on serology, with supportive evidence from other tests. In neural larva migrans, antibodies to *Baylisascaris* can be found in serum and cerebrospinal fluid (CSF); a rising titer is usually seen. In ocular larva migrans, an ophthalmoscopic examination may be diagnostic. Biopsies of the CNS are occasionally definitive, but larvae are often absent from the sample. A definitive diagnosis can also be made retrospectively from CNS samples taken at autopsy. *Baylisascaris* larvae are much larger (up to 80 µm in diameter and up to 1900 µm long) than *Toxocara* larvae, and can also be distinguished by their morphology. However, parasite larvae can be difficult to identify within tissues, and misidentification is common. Polymerase chain reaction (PCR) assays for *Baylisascaris* have been published. One assay was able to distinguish *B. procyonis* from parasites in other genera, as well as from *B. transfuga*, but not from *B. columnaris*.

[Photo: Cross-sections of larvae of *B. columnaris* in the muscle of a laboratory-infected mouse. The larval morphology and microscopic manifestations would be similar with *B. procyonis* in human tissue. CDC DPDx Image Library]

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Treatment

- Drug therapy
  - Albendazole
    - Prophylactic use in humans
  - Albendazole and corticosteroids
    - Clinical patients
- Early diagnosis and treatment key
  - Improvement may not occur despite treatment in advanced disease

Treatment with anthelmintic drugs, particularly albendazole, has been recommended in specific cases. This drug is protective in animal models if eggs have been ingested, but symptoms have not yet developed. In humans, albendazole has been used prophylactically after exposure to raccoon latrines (sites where raccoons return regularly to defecate) or other sources of eggs. Whether it is helpful in patients with clinical signs is uncertain, because the death of the parasite might worsen the inflammation. Most clinical cases have been treated concurrently with anthelmintics and corticosteroids; the corticosteroids are used 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.

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Morbidity and Mortality

- Baylisascariasis rare in humans
  - Neural larva migrans
    - Infants and young children
    - Exposure to raccoon feces
  - Ocular larva migrans
    - Healthy adults
    - No raccoon exposure
    - Hunting, trapping, taxidermy, wildlife handling are risk factors

Baylisascariasis appears to be rare in humans. The exact number of cases is uncertain; however, more than a dozen cases of probable or confirmed neural larva migrans have been published, and at least a 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, perhaps from an unrecognized immune defect. 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. 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.

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DISEASE IN ANIMALS

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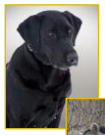
Disease in Animals

- Incubation period
  - 1 to 4 weeks
- Definitive hosts
  - Raccoons
    - Usually asymptomatic
    - Intestinal obstruction in severe cases
  - Dogs
    - Usually asymptomatic



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. 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.

[Photos: (Top) Dogs can be infected with *B. procyonis*. Source: www.public-domain-image.com; (Bottom) A raccoon. Source: USDA APHIS Wildlife Damage Management Photo Gallery]

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Disease in Animals

- Intermediate hosts
  - Nonspecific signs
  - Neurological disease
  - Visual defects
- Clinical signs
  - May develop acutely or progress slowly
  - May stabilize when larvae become encapsulated in tissues
  - May wax and wane

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. A wide variety of signs including circling or rolling, torticollis, opisthotonos, stargazing, ataxia, tremors, nystagmus, progressive weakness, paresis or paralysis, hypertonia, extensor rigidity, seizures and dysphagia have been reported. Birds may have difficulty perching. Visual defects including blindness and defective pupillary reflexes may also be seen. In some animals, the clinical signs develop acutely and progress quickly. Other cases may be insidious and progress more slowly. 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.

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Post Mortem Lesions

- Nematodes found in intestine
- Migrating larvae
  - Hemorrhagic or necrotic lesions
  - Granulomas
  - Focal softening in CNS
- Differentiate by:
  - Larvae size, morphology

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 (9 to 11 cm). Migrating larvae can cause hemorrhagic or necrotic lesions and tracks, as well as granulomas, in any tissue where they are found. In the CNS, there may be focal areas of palpable softening and discoloration, as well as small multifocal hemorrhages. Microscopic lesions can include multiple tracts with debris, gitter cells, neuronal degeneration, gliosis, vascular rupture with hemorrhage, malacia and eosinophilic and granulomatous inflammation, as well as perivascular cuffing. Larvae may be found both within the lesions and in areas of the brain that appear to be normal. In some cases, they may no longer be present in the CNS. They can be differentiated from some other nematodes such as *Toxocara* spp. by their large size and their morphology.

[Photo: Raccoon, intestine. This partially opened small intestine contains many adult *B. procyonis*. Source: Dr. A. Hamir, ARS, USDA/CFSPH]

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Diagnosis

- Identification of eggs or worms
  - Feces
  - Vomitus
- Larva migrans difficult to diagnose
  - Eosinophilia suggestive
  - Serology
  - Identification of parasite in tissues
  - PCR

Intestinal infections in raccoons and dogs can be diagnosed by identifying the eggs in feces, or worms in the feces or vomitus. 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, unless they are examined very carefully, these eggs can be readily confused. *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 CSF and blood are supportive in mammals; however, peripheral eosinophilia does not necessarily occur in birds with neural larval 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. Epidemiological evidence, such as a history of exposure to raccoons, but not to skunks or badgers, can be suggestive. A PCR assay has been published. [Photo: Raccoon, feces. *Baylisascaris procyonis* eggs are typical ascarid eggs with thick, finely pitted shells; they are slightly smaller than *Toxocara canis* eggs. Iowa State University, College of Veterinary Medicine/CFSPH]

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Treatment

- Anthelmintics
  - Piperazine, pyrantel, ivermectin, moxidectin, albendazole, fenbendazole, flubendazole
  - Monthly heartworm preventatives
- Corticosteroids
  - Useful for control of inflammation
- Supportive care

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 preventative 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.

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Morbidity and Mortality

- Raccoons
  - Widespread
  - Local prevalence varies widely
- Dogs
  - Reported cases infrequent
  - May increase human exposure
- Clinical cases often fatal
  - Illness/death rare in raccoons

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 overall prevalence is reported to be 58% in the Midwest, 64% in the Northeast/Mid-Atlantic states, 49% in the West/Southwest and 4% in the Southeast. However, the local prevalence varies widely. *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 unknown. 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 usually serious and often fatal. Significant illness or deaths have not been reported in infected raccoons.

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PREVENTION AND CONTROL

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Prevention and Control

- Avoid contact with raccoons
  - Don't keep raccoons as pets
    - Examine and deworm captive raccoons
  - Don't allow access to homes
- Good hygiene
  - Hand washing
  - Prevent pica
- Exposed persons
  - Albendazole

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. 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 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.

[Photo: A girl washing her hands. Source: CDC Public Health Image Library]

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Prevention and Control

- Eliminate raccoon latrines
  - Remove, burn, and/or bury feces
  - Wear gloves and protective clothing
  - Wash hands immediately afterward
  - Treat hard surfaces
    - Boiling water
    - Steam clean
    - Propane flame gun
- *Baylisascaris* eggs may remain

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 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). 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.

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Prevention and Control

- Additional disinfection measures
  - High heat (fomites)
  - Boiling lye water
  - Xylene-ethanol mixture

High heat (e.g., a propane torch, boiling water or incineration) is also used to decontaminate fomites. Boiling lye water has also been recommended. A xylene-ethanol mixture has been used after the solid waste was removed. 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.

[Photo: A raccoon. Source: U.S. Fish and Wildlife Service National Digital Library]

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Prevention and Control

- Dogs
  - Heartworm/nematode preventatives
  - Regular fecal examinations
- Captive animals
  - Prevent contact with raccoons
  - Clean cages regularly
  - Quarantine, test, deworm
- Treat exposed animals

Infections in pets allowed outdoors are difficult to prevent, as the infective eggs can survive for long periods in the environment. In dogs, monthly heartworm/ nematode preventatives appear to decrease the risk of intestinal infection with *B. procyonis*. In high-risk areas, dogs that are not on these preventatives should receive regular fecal examinations to decrease the risk that they will shed eggs. In zoos and other facilities, the 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, it can be difficult to remove completely. Intermediate hosts in exhibits are sometimes treated prophylactically with pyrantel tartrate or ivermectin. Similarly to humans, animals with recent exposure might also be treated with albendazole to prevent the development of clinical signs.

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Additional Resources

- Center for Food Security and Public Health
  - www.cfsph.iastate.edu
- CDC: *Baylisascaris* infection
  - http://www.cdc.gov/parasites/baylisascaris/index.html
- CDC: Raccoon latrine cleanup
  - http://www.cdc.gov/parasites/baylisascaris/resources/raccoonLatrines.pdf

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Acknowledgments

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