Wildlife Management
Publication Series

WARNELL
School of Forestry and Natural Resources
The University of Georgia

WMS – 10 --22 January 2010

Intestinal roundworm (*Baylisascaris procyonis)* of raccoons (*Procyon lotor*): Information for public health and wildlife professionals

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INTRODUCTION

The raccoon (*Procyon lotor*) occurs in a variety of habitats throughout much of their native range in North and Central America and in introduced regions of Europe and Asia (Figure 1). Typically rural raccoon population densities range from 1-27 raccoons/km^2. However, in suburban and urban areas raccoon population densities can range from 67-333/km^2 (Ghert 2003). Raccoons often thrive in habitats closely associated with humans where ample food sources are readily obtained through scavenging garbage, eating pet food, intentional feedings by people, or raiding birdfeeders (Riley et al. 1998, Gehrt 2003). This close association with people and domestic animals can be problematic because raccoons can be destructive and can harbor several pathogens that may pose a risk to humans and/or domestic animals (De Almeida 1987, DeStefano and DeGraaf 2003, Kazacos 2000). The raccoon roundworm, *Baylisascaris procyonis*, is a large parasitic intestinal nematode that has the potential to cause disease in numerous avian and mammalian hosts including humans.

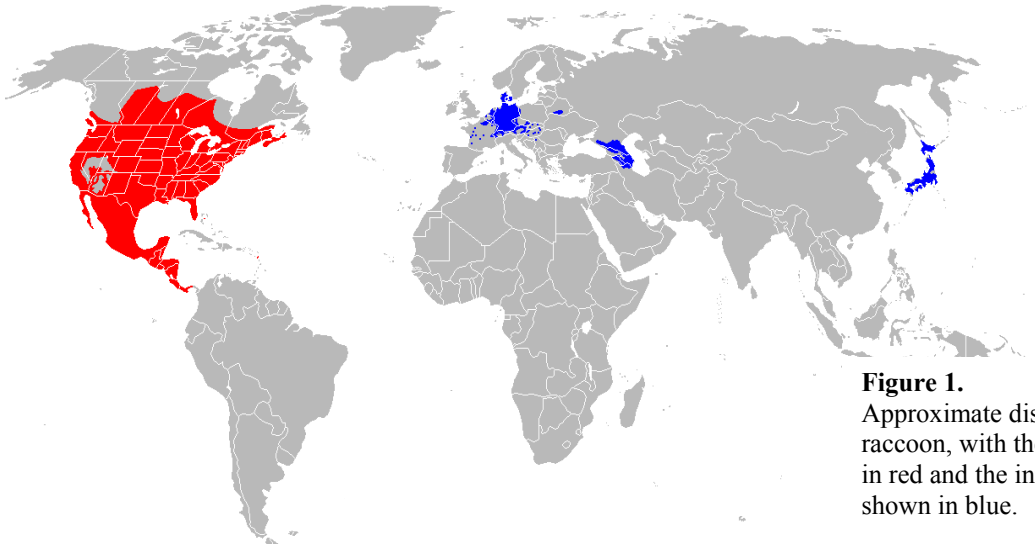

Figure 1.
Approximate distribution of the raccoon, with the native range shown in red and the introduced populations shown in blue.

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Where does this parasite occur?

Baylisascaris procyonis can be found throughout the northeastern, midwestern, and Mid-Atlantic States and along coastal areas of California, Washington, and Oregon (Figure 2). Prevalence rates in raccoon populations vary considerably by state and region with highest rates in the upper Midwest and coastal western states (Figure 2). In these areas, 90% of juveniles and 37-55% adults can be infected (Kazacos 2001). On average, juvenile raccoons harbor an average of 50 worms while adults typically harbor around 18 worms (Kazacos 2001).

Historically, *B. procyonis* has been absent from the southeastern United States. The first report, outside of the Appalachian regions of Kentucky and Virginia, occurred in 2001 from suburban Dekalb County, Georgia (Eberhard et al. 2003). In that study, 11 of 50 (22%) raccoons were infected. During the same year, a wildlife rehabilitator in Clarke County, Georgia reported finding *B. procyonis* in a single raccoon. However, the history of this raccoon was not available so the county of origin is not known (Eberhard et al. 2003.) The parasite was later detected in two raccoons from Clarke County, Georgia that were submitted to the Southeastern Cooperative Wildlife Disease Study (SCWDS) at the University of Georgia in 2006. An ongoing study has detected *B. procyonis* in approximately 9% of raccoons from Clarke County, GA (Blizzard et al., unpublished data).

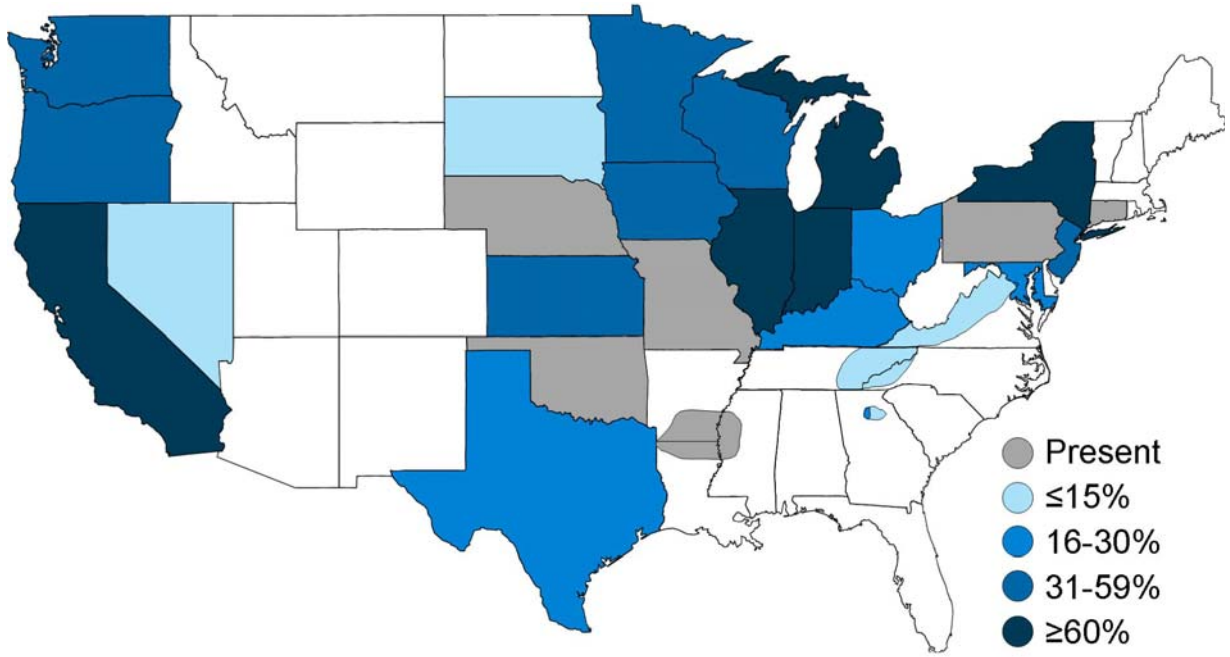

Figure 2: General distribution and percentage of raccoons infected with *B. procyonis*.

How is *Baylisascaris* spread among raccoons?

Raccoons can become infected by two different routes of transmission. Juveniles (i. e., kits) can become infected by ingesting *B. procyonis* eggs. Nursing kits might ingest eggs stuck to the mother's fur. Older individuals can ingest eggs while grooming themselves and others, and/or eating feces scattered on the floor of the denning area. Adult raccoons typically acquire infections through ingestion of paratenic hosts. Paratenic hosts are birds or mammals that ingest infective eggs and serve as a host to migrating *B. proyconis* larvae. These migrating larvae cause tissue damage that may weaken the host and expose the animal to higher predation risk. Alternatively, the paratenic host might be killed by the migrating larvae which could then be scavenged by a raccoon (Kazacos 1983, Kazacos and Boyce 1989, DeVault et al. 2004). If a paratenic host is ingested by a raccoon, the larvae migrate to the small intestine of the raccoon where they develop to adults.

What does *Baylisascaris procyonis* look like?

Baylisascaris procyonis is a large parasitic intestinal nematode (i. e., round worm) residing in the small intestine of raccoons. They can be white, beige, or tan in color (Figure 3). Female worms may reach 20-22 cm in length and males 9-11 cm. Raccoons serve as a primary or definitive host for this parasite. A definitive host is a host in which an organism, in this case *B. procyonis*, matures and sexually reproduces. Adult worms mate in the intestine and pass microscopic non-infective eggs into the environment in the feces of an infected raccoon. These non-infective eggs must embroyonate (develop) (Figure 4) in the environment for 10-14 days before they can infect a host.

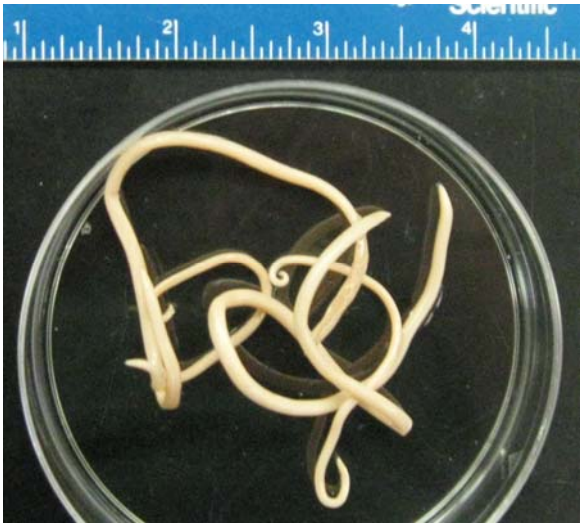

Figure 4. Adult *B. procyonis* from the small intestine of a raccoon. Ruler shown in inches.

Do infected raccoons exhibit any signs of disease once infected with *B. procyonis*?

Typically raccoons do not develop any clinical signs. Mortality has only been reported twice. Both cases were juvenile raccoons with intestinal obstructions due to extremely high numbers of worms.

Figure 4. Embryonated egg of *B. procyonis* that has been mechanically ruptured to allow larvae to exit.

What other animals can become infected?

Over 90 species of birds and mammals are susceptible to *B. procyonis* infections. Rodents, rabbits, primates, and birds are particularly susceptible to *B. procyonis*. These species are more susceptible to severe larval migrans that occurs when worm larvae travel throughout a host's body. While other species such as opossums, cats, birds of prey, and domestic/wild hoofstock (e.g., sheep, goats, and swine) appear to be resistant (Kazacos 2001). In resistant hosts, either the larvae fail to hatch and migrate through tissues or the host mounts an immune response that kills the larvae before they cause substantial tissue damage (Kazacos 2001). Clinical disease has most often been reported in captive and/or free-ranging rodents, rabbits, and birds. In addition, significant outbreaks have been reported animals from rehabilitation centers and zoological parks including Patagonia Mara (*Dolichotis patagonum)*, Capybara (*Hydrochaeris hydrochaeris)*, Ruffed Lemur (*Varecia variegata variegata)*, Coquerel's Mouse Lemur (*Mirza coquereli)*, and the Australian Brush Turkey (*Alectua lathami)* (Kazacos 2001).

How do paratenic hosts become infected with *B. procyonis*?

Paratenic hosts, a host in which the parasite does not sexually reproduce but is used for development, often become infected by foraging among raccoon latrine sites (Giles 1939, Stains 1956, Yeager and Rennels 1943). Raccoons use communal latrines, where seeds voided in feces accumulate and serve as an easy food source for some granivorous birds and mammals (Figure 5) (Tiner 1952, 1953; Wirtz 1982; Kazacos and Boyce 1989; Sheppard and Kazacos 1997; Page 1998; Page et al. 1999). One study documented 31 species of mammals and birds foraging among raccoon latrine sites (Page et al. 1998, Page et al 2001). In addition, *B. procyonis* eggs are sticky allowing them to adhere to the fur where they can be ingested while grooming (Kazacos and Boyce 1989, Yeitz et al. 2009). Once an egg is ingested by a paratenic host, it hatches in the small intestine and the larvae burrows out of the intestine and migrates through the host's tissues. Captive animals can become infected when they are housed in cages that previously housed infected raccoons, fed food contaminated with infected raccoon feces, or eat other paratenic hosts (Kidder et al. 1989, Stringfield and Sedgwick 1997, Kazacos 2001).

Figure 5. Latrine site in a tree (blue arrow). Inset: close-up of latrine site showing buildup of feces and a plant that has started to grow from old feces.

Do paratenic hosts exhibit clinical symptoms of the disease?

The degree of clinical signs, if any, depends on the number of larvae ingested and the tissues that are damaged. If large numbers of larvae are migrating through tissues, tissue damage to the lungs, heart, and muscles can be fatal. Unfortunately, *B. procyonis* larvae seem to prefer the central nervous system (spinal cord and brain or CNS), and even small numbers of migrating larvae can cause significant CNS damage. Animals with CNS damage develop abnormal behaviors such as circling, rolling, falling over, paralysis, laying on their side or back and paddling the air with their feet. These clinical signs also occur in animals with rabies, as well as other diseases; therefore, care should be taken if animals are acting abnormally.

Are there any other pathogens that can cause symptoms similar to *B. procyonis*?

Yes, several other pathogens can cause similar clinical symptoms. In the United States, there are three other *Baylisascaris* species (*B. columnaris* in skunks, *B. transfuga* in bears, and *B. melis* in badgers), that cause disease in paratenic hosts. A common parasite of puppies and young dogs, *Toxocara canis*, can cause both ocular and visceral larval migrans, worm larvae that travel throughout a host's body, in people. However, unlike *B. procyonis*, *T. canis* isn't associated with neural larval migrans. Cats and kittens can be infected with a similar parasite, *T. cati*. Regular testing and antihelminthic treatment of domestic dogs and cats should be encouraged. In addition, numerous other pathogens (e.g., rabies, canine distemper, West Nile virus, *Toxoplasma gondii*, *Sarcocystis neurona*, etc.) can cause neurologic disease in a wide range of mammalian hosts. Care should be taken when handling or collecting a neurologic mammal.

How can I insure that my dog does not become infected and put my family at risk?

Domestic dogs can serve as hosts for *B. procyonis*. This is particularly worrisome because dogs frequently reside and defecate in close proximity to human dwellings and yards (Kazacos 2001). In addition to patent infections, an infection in a host in which a parasite can sexually produce and/or cause disease, several dogs have developed fatal larval migrans (Thomas 1988; Rudmann et al. 1996; Kazacos 2001). Because of the

severity of *B. procyonis* larval migrans in humans, regular testing and antihelminthic treatment of dogs is necessary. It is likely that a larger proportion of dogs harbor *B. procyonis* than previous studies have documented because of the difficulty in distinguishing the eggs of *Baylisascaris* and *T. canis*.

How many human cases have there been?

Since first recognized in the 1980's, there have been nearly 30 documented human cases. Because humans usually ingest only a few eggs, the low numbers of migrating larvae cause only minimal damage in most infections. Many cases (some might only have mild CNS disease) likely never are diagnosed. Because no serologic test is available, no large-scale serologic studies have been conducted to determine the prevalence of infection among people.

How is an infection with *B. procyonis* diagnosed?

Definitive hosts (raccoons and domestic dogs): Recovery of eggs in fecal samples of raccoons or dogs can be accomplished by fecal floatation using standard parasite recovery sugar or salt solutions. These eggs must be differentiated from related parasites such as *Toxocara* spp. and *Toxascaris leonina*. Additionally, adult worms can be recovered from the small intestine of infected hosts at necropsy.

Paratenic hosts: Larval migrans in paratenic hosts often result in severe neurologic disease and/or death. If these animals are necropsied, gross lesions are rarely observed, but in severe infections, inflammatory reactions (nodules) may be observed in various organs. Microscopically, larval migrations will be characterized by areas with high numbers of eosinophils and necrosis. Finding a cross section of the larvae is diagnostic for a nematode but the species of worm cannot be determined by histology. *Baylisascaris* larvae are approximately 60µm in diameter and have prominent lateral projections. If *Baylisascaris* larval migrans are suspected, large numbers of histologic tissue sections (especially of neural tissue) should be examined. Additionally, rabies and other pathogens, that can cause neurologic signs, must be ruled out as a possible cause.

Humans: Diagnosis of Baylisascariasis in humans is difficult. It should be suspected when a person develops acute neurologic signs and/or vision impairment and has had potential exposure to infective eggs. Parents of young children may not recall instances of eating clay or dirt (geophagia) or eating inedible objects (pica). Additionally, many physicians rarely include Baylisascariasis as a potential differential diagnosis. Although serologic tests have been developed the availability of the antigen is limited, thus serologic testing is only prescribed for highly suspect cases. Most often, infections are diagnosed by post mortem examination of tissues. Ocular larval migrans are easier to diagnose as migrating larvae can be observed.

What treatment options exist?

Currently, an effective treatment does not exist for larval migrans associated with *B. procyonis* in humans (Murray and Kazacos 2004). Treatment of neural larval migrans is complicated because the majority of antihelminths do not cross the blood-brain barrier. However, albendazole has been shown to penetrate the blood-brain barrier and has been used to successfully treat a single case (Pai et al. 2007). Treatment with antihelminthic drugs, even if they do not penetrate the blood-brain barrier, may be useful because it would result in a reduction in the number of larvae migrating through other organ systems. However, any damage that has occurred from larvae migrating into and through the brain and other organs before treatment may lead to permanent damage. Treatment with albendazole should be paired with corticosteroids to reduce inflammation caused by albendazole and the host's reaction to dead parasites (Wise et al. 2005). Treatment of ocular larval migrans involves using a laser to kill the larval migrans in the eye followed by anti-inflammatory drugs and frequently steroids to aid in the possible recovery of any remaining visual acuity.

How long can *B. procyonis* eggs remain viable in the environment and how can I decontaminate areas I suspect may be contaminated with them?

B. procyonis eggs can remain viable for years. Because raccoons can pass approximately 100 grams of feces per defecation and an adult female worm can release over 26,000 eggs each day, the environment can rapidly become contaminated with very high numbers of parasite eggs. Currently only a few methods are effective at killing infective *B. procyonis* eggs. Flooding of the contaminated area with highly concentrated caustic chemicals such as a 50/50 mixture of xylene and absolute alcohol, boiling lye, or boiling Lysol can be effective (Kazacos 1983). However, application of these chemicals to the environment is not ideal and impractical. An alternative method to using chemicals is to burn or scorch the contaminated areas. Flaming is useful in decontaminating some items such as metal cages or localized latrine sites. However, most areas cannot be burned (e.g., roofs, decks, etc.).

Acknowledgements

Emily L. Blizzard and this publication were partially supported by a grant from the Southeastern Center for Emerging Biologic Threats.

Editor

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