

Review

Severe and fatal central nervous system disease in humans caused by *Baylisascaris procyonis*, the common roundworm of raccoons: a review of current literature

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Available online 08 January 2005

Abstract

Baylisascaris procyonis, a parasitic infection of raccoons, causes severe neurologic disease in humans when infective eggs from raccoon feces are ingested. Definitive diagnosis is challenging, but can be made by isolation of larvae in brain biopsy or exclusion of other potential causes of eosinophilic meningoencephalitis. Prevention efforts are critical due to the lack of effective treatment.

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Keywords: *Baylisascaris procyonis*; Zoonoses; Encephalitis; Raccoons; Parasite

1. Introduction

In the last 20 years, *Baylisascaris procyonis*, the common intestinal roundworm of raccoons, has increasingly been recognized as a source of severe human neurologic disease [1]. As human populations continue to expand and come into contact with animal populations, the impact of zoonoses like baylisascariasis will likewise increase. The confluence of ample food sources, lack of raccoon control programs, and expansion of human populations into previously rural areas is making urban and suburban locations the most risky areas for exposure to *B. procyonis* [2]. While human baylisascariasis appears to be rare, the devastating neurologic disease that is caused by this infection and the lack of effective treatment make it a disease of public health importance.

2. Epizootiology and life cycle of *B. procyonis*

Adult raccoons infected with *B. procyonis* can shed millions of unembryonated eggs in feces each day. Within as

little as 2 weeks they can mature to infective larvae [3–5]. Once infective, eggs can remain viable in the environment for years, even during harsh winters or in dry conditions, and are resistant to most typical decontamination methods [4]. In fact, *B. procyonis* eggs are so resistant that they will embryonate and become infective in a weak formalin solution [4]. Eggs tend to be brown and ellipsoid, ranging in size from 63–88 µm by 50–70 µm (Fig. 1) [5]. Identification of eggs in the environment is a strong indicator of infection risk for humans and other animals.

Raccoons can acquire *B. procyonis* through two distinct routes. In one mode of transmission, juvenile raccoons ingest infective eggs directly through contact with fur of the mother or through contaminated dens or latrines (preferential communal defecation sites) [5]. The larvae from these eggs are released and invade the wall of the intestine, where they develop for several weeks. They enter the small intestine and mature into male and female worms (Fig. 2) [5]. Gravid females begin releasing eggs into the fecal stream at 50–76 days post-infection, where they are carried out of the intestine and into the environment, undergo embryonation, and become infective [5]. Juvenile raccoons tend to shed greater numbers of eggs than adult raccoons [3,4].

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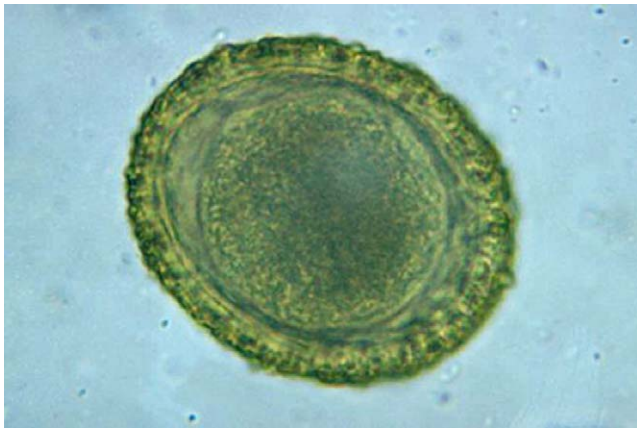


Fig. 1. Unembryonated egg of *B. procyonis* (originally photographed at 40× magnification).

In the second mode of transmission, paratenic hosts such as squirrels or other rodents ingest infectious *B. procyonis* eggs from the environment, often during normal food gathering [6]. In these non-ideal hosts, eggs hatch in the intestine, larvae penetrate the intestinal wall and enter the blood stream, and are disseminated to various organs and tissues. While the sites of larval migration vary by host species, larvae often migrate to the central nervous system (CNS), causing behavioral changes in these animals, potentially making them easier prey for raccoons [7]. When adult raccoons consume these paratenic hosts, they ingest the third-stage larvae contained in the tissues of the rodent [4]. These larvae mature in the raccoon's gut, mate, and shed eggs within 32–38 days, which are released in feces and embryonate in the environment [5].

Surveys of the prevalence of *B. procyonis* infection in raccoon populations have been conducted in numerous locations throughout North America. For a comprehensive review of *B. procyonis* prevalence studies in raccoons, see *Parasitic Diseases of Wild Mammals* [5]. Thirty-three prevalence studies of intestinal *B. procyonis* infection in raccoons have been conducted in the Midwest region of the United States in which 3967 total raccoons were examined, and 58% were found to be infected. The range of prevalences found for these 33 studies was from 0% to 100%. Seven prevalence studies were conducted in the Northeast/Mid-Atlantic region of the US,



Fig. 2. Adult male and female worms of *B. procyonis*.

with a total of 476 raccoons. Overall, 64% were found to be positive, with an individual study range from 5% to 100%. Twenty-eight studies were conducted in the Southeast region in which 1868 raccoons were examined, with 4% positive for *B. procyonis*. The range for individual studies in the Southeast was from 0% to 71%. Finally, 10 studies were conducted in the West/Southwest region among 229 raccoons with 49% found to be positive. The range for the West/Southwest region was 0–100%. Other countries that have demonstrated the existence of *B. procyonis* in raccoon populations include Canada, Germany, Poland, Japan, and the Czech Republic [5]. It is important to note that the prevalence of *B. procyonis* may be substantially higher or lower in localized areas than these aggregate measures indicate. Prevalences can approach 100%, especially among juvenile raccoon populations.

A number of intermediate, paratenic, or dead-end hosts for *B. procyonis* have been identified both in nature and experimentally. Somatic tissue migration has been observed to occur in mice, dogs, rabbits, guinea pigs, chickens, pheasants, swine, subhuman primates, prairie dogs, and other mammalian, avian, and marsupial species [5,6,8–14]. These hosts can be considered paratenic if they serve to transmit *B. procyonis* to raccoons. Most paratenic hosts are small rodents or other animals, which can be easily caught and consumed by raccoons. The designation of a paratenic or dead-end host is primarily determined by feeding behaviors of raccoons. It is also important to note that differences exist between these paratenic and dead-end hosts with respect to the extent and location of larval tissue migration. While *B. procyonis* enters the CNS relatively frequently in primates and rodents, it rarely enters the CNS of swine [8]. This is relevant to human pathogenesis, because estimates of the proportion of larval migration to the brain of humans have been calculated using animal models [15].

While most animals that ingest eggs of *B. procyonis* will have a disease process similar to that of humans, in which larvae migrate throughout tissues in the body, it is possible for several species to act as ancillary definitive hosts and to develop adult intestinal *B. procyonis* infections similar to that of raccoons. Intestinal infections of non-raccoon species have been documented in kinkajous, olingos, dogs, and experimentally in opossums [5]. When adult forms of *B. procyonis* establish intestinal infection in these hosts, the parasite is able to mature and mate in the gut and shed eggs in the feces. This phenomenon is particularly problematic with regard to dogs. If *B. procyonis* infections were to become prevalent among domestic dogs, both the proximity of dogs and humans and the indiscriminant defecation practices of dogs (as opposed to the localized latrine defecation of raccoons) could greatly increase the risk of human infection [5,16].

3. Epidemiology and case reports

Human infection with *B. procyonis* occurs by the ingestion of infective eggs from environmental sources. Severe neu-

rologic disease was caused experimentally in squirrel monkeys by oral inoculation of 5000 infective eggs [17]. Based on the infectious dose for other ascarids, however, substantially fewer ingested eggs could potentially cause severe disease in humans. Numerous environmental sources have been implicated in the transmission of *B. procyonis* to humans. Transmission occurs when items contaminated with raccoon feces are placed in the mouth or eaten. The most commonly reported suspected vehicles include soil, wood, leaves and other vegetation, bark, sand, and stones, along with the direct ingestion of raccoon feces (Table 1). Woodpiles, the base of trees, roofs, decks, and sandboxes have all been reported to serve as raccoon latrine sites [5,18–20]. Raccoon feces on rooftops may either wash over the edges during rain, contaminating soil around homes, or may fall down chimneys, contaminating indoor areas, especially in and around fireplaces [21,22]. Keeping pet raccoons may also serve to contaminate areas inside and outside the home, unless the raccoons are regularly dewormed by administering antihelminthics [4]. Finally, cases of human infection have been linked to increased residential raccoon activity, and this activity may be

exacerbated by accidental or deliberate feeding of peridomestic raccoon populations [2].

Fourteen probable or confirmed cases of severe or fatal human *B. procyonis* infection have been documented in the literature, with cases being reported approximately once every 1–2 years (Table 1) [1,18,19,21,23–30]. Ninety-two percent of subjects in whom the gender was known were male, and 64% of all subjects became infected under the age of 2 years. There has been a wide geographic distribution of cases, with human *B. procyonis* infection documented in California, Illinois, Minnesota, Oregon, Pennsylvania, New York, Massachusetts, and Missouri. In addition, epizootic infection of *B. procyonis* in rabbits was recently documented in Japan, and ocular disease in humans has been reported in Germany [13,31]. Forty-six percent of subjects with known developmental disability status had developmental disabilities prior to infection, and four of the five subjects over the age of 2 years had developmental disabilities prior to infection. Seventy-nine percent of all subjects were reported to exhibit pica or geophagia. Forty-six percent of cases with known outcome were fatal (Table 2).

Table 1
Probable and confirmed cases of human *B. procyonis* infection, 1973–2002

| Year/sex/age/location ^a | Developmental disability | Infection source | Symptoms | Outcome | References |
|---|--------------------------|---|---|---|--------------|
| 1973/Female/18 months/Missouri ^b | No | Soil | Moderate right side weakness, irritability | Weakness, spasticity | [18,24] |
| 1980/Male/10 months/Pennsylvania | No | Fireplace, wood, raccoon feces | Loss of movement, eosinophilic meningoencephalitis | Fatal | [1,18,21,25] |
| 1984/Male/18 months/Illinois | Down's Syndrome | Bark, wood chips | Lethargy, eosinophilia, encephalitis | Fatal | [1,18,25,26] |
| 1986/Male/21 years/Oregon ^b | Yes | Unknown, pica | Abnormal behavior, CNS disease | Unknown | [18,27] |
| 1990/Male/13 months/New York | No | Soil | Refusal to walk, eosinophilia | Developmental delay, blindness, hemiparesis | [1,18,25,27] |
| 1995/Male/10 years/Massachusetts | Yes, Mild | Unknown | Abdominal pain, eosinophilia, unresponsiveness | Fatal, due to cardiac pseudo-tumor | [1,28] |
| 1995/Male/13 months/Minnesota | No | Pet raccoon, feces on floor of home | Irritability, weakness, eosinophilia | Fatal | [25,29] |
| 1996/Male/6 years/Illinois | Yes | Soil, raccoon feces | Developmental delay, DUSN | Developmental delay, seizures | [1,25] |
| 1997/Male/13 months/California | No | Sandbox, base of trees, wood pile, soil | Lethargy, speech deterioration, stagger, eosinophilia | Seizures, blindness, neurologic deficits | [1,18,19,25] |
| 1997/Male/19 months/Minnesota ^c | Yes | Wood chips, soil | Ataxia, unresponsiveness, eosinophilia | Fatal | [25,29] |
| 1998/Male/11 months/California | No | Stones, pica | Irritability, behavioral regression, ocular symptoms | Seizures, encephalopathy, visual impairment | [1,18,25] |
| 2000/Male/17 years/California | Yes | Sandbox, feces, pica | Drowsy, eosinophilic meningoencephalitis | Fatal | [1,23] |
| 2000/Male/2.5 years/Illinois | No | Soil | Encephalopathy, fever, lethargy, eosinophilia | Developmental delay, spasticity, blindness | [1,23,25] |
| 2002/Unknown/11 months/California | Unknown | Soil | Unknown | Unknown | [30] |

^a Year, age, and location refer to the presumed time and place of initial infection.

^b Probable cases.

^c Some information on this case was obtained through personal communication with J. Watterson.

Table 2
Demographic characteristics of confirmed and probable cases of human *B. procyonis* infection

| Demographic characteristics | Number of cases | Percent of cases (%) |
|--|-----------------|----------------------|
| <i>Sex</i> | | |
| Male | 12 | 92 |
| Female | 1 | 8 |
| Unknown | 1 | — |
| <i>Age at diagnosis</i> | | |
| 2 years old or younger | 9 | 64 |
| Older than 2 years | 5 | 36 |
| Unknown | 0 | — |
| <i>Developmental disability prior to infection</i> | | |
| Yes | 6 | 46 |
| No | 7 | 54 |
| Unknown | 1 | — |
| <i>Outcome</i> | | |
| Fatal | 6 | 46 |
| Severe neurologic sequelae | 7 | 54 |
| Unknown | 1 | — |

It is important to note that there have been no seroepidemiologic studies published that attempt to quantify the extent of human infection with *B. procyonis* or to assess the clinical spectrum of disease caused by this agent. Consequently, while the current number of cases identified is small, it may be that a substantial amount of undiagnosed or misdiagnosed neurologic illness could be attributable to *B. procyonis*. Until studies are undertaken to evaluate the role of this agent in neurologic disease, it will be impossible to determine whether the cases identified thus far are representative of normal human infection with *B. procyonis*, or are merely a select group of extremely severe infections. There also have been no controlled epidemiologic studies conducted to better define risk factors for infection with *B. procyonis*, primarily due to the small number of documented cases.

4. Pathogenesis and clinical manifestations of human infection

The severe clinical manifestations of *B. procyonis* in humans and other paratenic or dead-end hosts are a result of several factors. First, the tissue migration of *B. procyonis* is far more aggressive than that of other parasitic infections that cause larva migrans, such that migration tracks through the CNS and other tissues may be substantial [5,32]. Second, the larvae of *B. procyonis* continue to grow as they migrate, exacerbating mechanical damage to tissues [5,32]. Third, mechanical damage is further aggravated by their large size compared to other tissue-migrating nematodes [5]. Fourth, both the release of excretory–secretory proteins by migrating larvae and the release of toxic eosinophil proteins by the host cause severe inflammatory reactions [5,29,33]. The combined effect of these factors creates debilitating and often fatal CNS illness in the host [5,32]. While the pathogenesis of severe neurologic disease due to *B. procyonis* is thought to be relatively

well understood, much is yet to be learned about the pathogenesis of less severe or unrecognized infections.

Initial symptoms of infection with *B. procyonis* may vary according to the primary site of larval migration in the body, as do clinical outcomes. Three categories of human *B. procyonis* infection can be defined. The first category is neural larva migrans (NLM), in which the brain is the primary site of larval migration. Studies using animal models have estimated that approximately 5–7% of ingested *B. procyonis* eggs migrate to the CNS [15]. Cases of baylisascariasis NLM initially may present with eosinophilic meningoencephalitis, irritability, weakness, lethargy, behavioral changes, or speech deterioration, and are often accompanied by ocular involvement. Forty-six percent of neurologic infections with known outcome have been fatal; non-fatal infections result in permanent neurologic sequelae including developmental disabilities, seizures, paralysis, and blindness [1,18,19,21,23–29].

The second type of *B. procyonis* infection is ocular larva migrans (OLM), in which the eyes are the primary site of larval migration. Although OLM occurs concurrently among cases of *B. procyonis* NLM, it has also been observed to occur independently. Symptoms typically include vision loss, transient visual obscuration, and diffuse unilateral subacute neuroretinitis (DUSN). Cases in which infection is limited only to the eyes are not fatal, but have resulted in permanent vision loss and optic nerve impairment [31,34].

The third category of infection is visceral larva migrans (VLM), in which organs in the abdominal and thoracic cavities and other body tissues are the primary sites of larval migration. Only one case of human *B. procyonis* VLM has been documented, in which a child had initial symptoms of abdominal pain, eosinophilia, and eventually unresponsiveness. This case was fatal, with death resulting from a cardiac pseudo-tumor [28]. It is probable that some level of VLM occurs with any heavy *B. procyonis* infection; however, symptoms due to VLM are likely to be overshadowed by the severe neurologic symptoms due to NLM. Larval migration in tissues other than the eye and CNS may cause non-specific symptoms as well.

5. Diagnosis

CNS infection with *B. procyonis* should be considered in the differential diagnosis of encephalitis with cerebrospinal fluid or peripheral eosinophilia [35]. Definitive diagnosis of *B. procyonis* as the etiologic agent is complicated by a number of factors, however. First, as humans are not definitive hosts, *B. procyonis* does not establish intestinal infection, and consequently eggs will never be observed in the feces, regardless of infection status. Second, infection with many parasitic agents such as *Toxocara canis*, *Toxocara cati*, *Angiostrongylus cantonensis*, *Gnathostoma spinigerum*, and free-living amoebae may cause both neurologic symptoms and eosinophilia. Specific serologic tests are available for some of these infections, such as *T. canis*, and it thus can be

excluded as a cause relatively easily. ELISA and IFA tests using *B. procyonis*-specific excretory/secretory antigens were developed at Purdue University. Although it is possible to find *Baylisascaris*-specific antigens that do not cross-react with other larval infections, the availability of such tests is limited, and the sensitivities and specificities have not been assessed. Consequently, while currently available serologic tests may be helpful in providing support for a clinical diagnosis of *B. procyonis* when the prior probability of infection is relatively high, it is not appropriate for seroprevalence or seroepidemiologic studies in the general population, where the prior probability (prevalence) of infection is low and the potential for false-positives is high. A third difficulty in diagnosing *B. procyonis* infection of the CNS is that white matter changes in the brain typically lag behind clinical symptoms, reducing the effectiveness of neuroimaging for early diagnosis of *B. procyonis* infection [35]. Currently, definitive diagnosis is based on identification of larvae in biopsy specimens. However, the use of needle aspiration brain biopsies is problematic. As very few larvae can cause disease, the probability of detection in such a small sample is exceedingly low. Moreover, to best diagnose the larvae of *B. procyonis* histologically, a cross-section of a larvae is most helpful. In addition, morphologic diagnosis of tissue parasites is challenging, because it requires considerable expertise and experience.

The morphology of *B. procyonis* is sufficiently unique to allow it to be differentiated from other tissue-migrating nematodes, given an adequate tissue section is obtained. The larvae of *B. procyonis* are large, with a length of 1500–1900 μm and a width of 60–80 μm [5]. Cross-sections of *B. procyonis* are identifiable by a number of characteristics. The body wall has a thin cuticle with prominent lateral alae. The hypodermis is expanded laterally to form large lateral chords which do not have sharp boundaries, but do have visible nuclei at the base of the chords. Dorsal and ventral chords are not well defined. There are typically four to eight coelomyarian muscle cells in each quadrant of the body. Irregularly shaped paired columns with a central canal make up the excretory system, with the canal visible only occasionally. The esophagus is cylindrical and muscular with a triradiate lumen. The intestine is typically round or oval and is lined with microvilli. Basophilic inclusions are conspicuous within the cytoplasm of the intestinal cells (Fig. 3). *B. procyonis* can be differentiated from *Toxocara* species based on their larger size, a patent gut, and predilection for invasion of the CNS [36].

Other indicators of human *B. procyonis* infection include history of exposure to or contact with raccoons and elevations in isohemagglutinins due to cross-reaction of larval excretory–secretory proteins and human blood group antigens [35]. Obtaining sections of brain from post-mortem biopsies may be the most likely method to obtain larval samples that are identifiable, but this option has the disadvantage of allowing for definitive diagnosis only after death and also may not be acceptable to family members. Several methods for recovering larvae from brain tissue are explained in detail elsewhere [5]. Diagnosis of *B. procyonis* as the etiologic agent of neu-

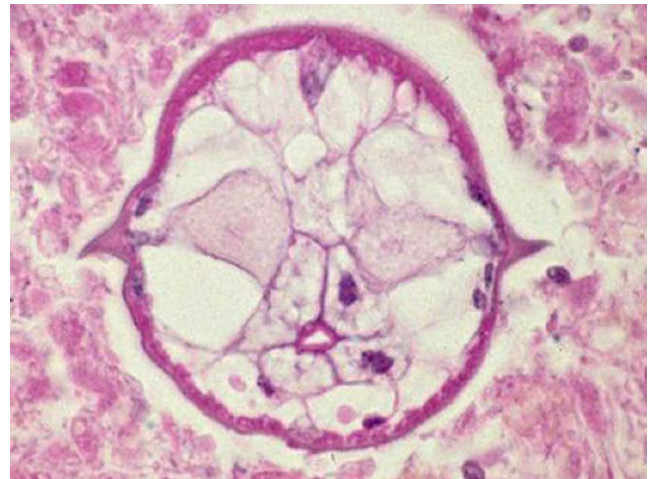


Fig. 3. Cross-section of *B. procyonis* larva in lymph node tissue (originally photographed at 180 X magnification).

rologic disease is typically the result of a constellation of imperfect indicators and the systematic exclusion of all other potential causal agents. Given the difficulties in diagnosis, the human impact of baylisascariasis is probably underrecognized. To definitively answer this question, better diagnostics for human *B. procyonis* infection need to be developed, including a widely available and validated diagnostic serologic test.

6. Treatment

To date no cases of human neurologic disease caused by *B. procyonis* have been successfully treated. While antihelminthic treatment has been attempted in some cases, it has been impossible to tell whether treatment may have slowed or halted the progression of disease, and neurologic disease has never been reversed or cured. The apparent ineffectiveness of antihelminthics may be due to the fact that once larvae have entered the CNS, significant tissue damage has already occurred. It is thought that administration of a course of antihelminthics within 1–7 days of the initial exposure may be able to abort infection [5,37]. Unfortunately this is a time when infection is still asymptomatic and likely to be unrecognized. Furthermore, the success of this regimen was only observed in experimentally infected mice [37]. The most promising treatment is albendazole, due to its anti-nematode activity and ability to cross the blood-brain barrier. It is possible that administration of this drug immediately after the onset of symptoms may limit the sequelae of infection. Simultaneous administration of corticosteroids has the dual benefit of reducing the inflammatory response caused by albendazole and increasing the plasma concentration of the drug [18,35]. There is a dire need to find other effective antihelminthic drugs and to determine optimal therapeutic regimens to treat infections caused by *B. procyonis*.

7. Limiting potential for human infection

Risk of human infection with *B. procyonis* is present in any area where humans, especially children, come into direct or indirect contact with raccoon populations infected with *B. procyonis*. As no effective treatment is available for infection, efforts to minimize the impact of *B. procyonis* on human populations must emphasize prevention [5]. One important means of limiting infection potential is to reduce raccoon populations near residential areas. This can be accomplished by reducing food sources through restricting intentional feeding of raccoons, ensuring that raccoons do not have access to trash or food for domestic pets, and by trapping and relocating raccoons [5]. In addition, it is advised not to keep raccoons as pets unless they are meticulously checked and treated for *B. procyonis* infection, although this is difficult to ensure.

Potential for human infection can also be mitigated by decontamination of areas known to be contaminated with the eggs of *B. procyonis*. Eggs are quite hardy, and can remain infective in the environment, given adequate moisture, for several years and are resistant to most forms of normal decontamination [4]. Vigorous decontamination efforts employing boiling lye water, boiling Lysol, or propane torches likely provide the highest chance of inactivating the eggs of *B. procyonis* [4]. Contaminated soil or feces can also be removed, although difficult, and decontaminated by burning [4]. Unfortunately, there have been no comprehensive studies published to determine practical and effective methods for decontamination or inactivation of *B. procyonis* eggs. Given current knowledge, the preferred method of decontamination is with a propane torch, because it is known to inactivate the agent and is acceptable to use in the environment [5].

Finally, prevention efforts may also be enhanced through steps taken in the home. Hand washing and good hygiene, especially after outdoor playing, are important in preventing many infections in children, including baylisascariasis. Oral exploration of the environment by children or by people with developmental disabilities should also be discouraged. Any known raccoon latrine sites in yards or other areas should be decontaminated immediately, and access to these areas should be restricted to individuals with appropriate personal protective equipment such as gloves and, in dry conditions, masks [4,32,38]. It is important to note that the use of masks when handling dry feces is recommended not because *B. procyonis* can be acquired through inhalation, but because of other infections that can be acquired through the inhalation of fecal material.

8. Conclusions

B. procyonis infection is an important, albeit rare, condition to be considered in the differential diagnosis of children presenting with neurologic symptoms and eosinophilia. Due to the current limitations in the diagnosis and identification of *B. procyonis* infections, there is still much to be learned

regarding the incidence of infection and the clinical spectrum of disease. Improvements in diagnostic procedures, better control of raccoon populations, and greater awareness of *B. procyonis* as a cause of human disease are all critical in limiting and controlling this potentially devastating infection.

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