Veterinary Parasitology 193 (2013) 404–412

Contents lists available at SciVerse ScienceDirect

Veterinary Parasitology

journal homepage: www.elsevier.com/locate/vetpar

# Baylisascariosis—Infections of animals and humans with 'unusual' roundworms

Christian Bauer*

*Institute of Parasitology, Justus Liebig University Giessen, Rudolf-Buchheim-Strasse 2, 35392 Giessen, Germany*

ARTICLE INFO

*Keywords:*
*Baylisascaris procyonis*
*Baylisascaris schroederi*
*Baylisascaris transfuga*
Bear
Giant panda
Raccoon
Larva migrans

ABSTRACT

The nematode genus *Baylisascaris* (order Ascaridida, superfamily Ascaridoidea) contains nine relatively host-specific, parasite species of carnivores, omnivores, herbivores, carnivorous marsupials or rodents. They have a facultative heteroxenous life cycle, at least under experimental conditions. Eggs passed in faeces embryonate in the environment and the second-stage larva infective for both definitive and intermediate hosts develops. In intermediate hosts larvae migrate extensively through tissues, where they grow and moult to the third-stage, causing extensive damage. All *Baylisascaris* spp. are considered a potential cause of visceral, ocular and/or neural larval migrans in mammals including humans and in birds. This paper summarises our current knowledge on the prevalence, biology, pathogenicity and zoonotic significance of three *Baylisascaris* species: *B. transfuga*, *B. schroederi* and *B. procyonis* which have as definitive hosts bears, giant pandas and raccoons (occasionally dogs), respectively.



## 1. Introduction

The genus *Baylisascaris* (superfamily Ascaridoidea) contains nine species that can be differentiated using morphological criteria and in the case of four species by genotypic analysis of the mitochondrial genomes (Xie et al., 2011a,b). They are relatively host-specific parasites of the small intestine of carnivores, omnivores, herbivores, carnivorous marsupials or rodents (Table 1). Adult female worms can reach lengths of 14–28 cm and males 7–12 cm (Sprent, 1968). *Baylisascaris* spp. eggs are not embryonated when passed in fresh faeces. The eggs embryonate in the environment and become infective within 2–4 weeks (Sakla et al., 1989; Papini and Casarosa, 1994). Unlike other ascarid species such as *Toxocara canis* (Bruňaská et al., 1995) and *Ascaris suum* (Geenen et al., 1999) in which the third-stage larva is the infective stage within the egg, second stage larvae in *Baylisascaris* spp. eggs are considered to be the infective stage, with the second moult thought to occur in the infected host animal. All *Baylisascaris* spp. have a facultative heteroxenous life cycle, at least under experimental conditions (Sprent, 1953). When ingested by animals such as mice, the embryonated eggs hatch and second-stage larvae start to migrate through organs and tissues. During the migration phase the larvae grow considerably (Bowman, 1987; Goldberg et al., 1993) and moult to the third-stage (*Baylisascaris procyonis* third stage larvae are 1300–1900 μm in length) meaning these animals serve as intermediate hosts. This is in contrast to *T. canis* whose larvae (approximately 400 μm long; Goldberg et al., 1993) do not further develop in mice (paratenic hosts; Schnieder et al., 2011). The migrating *Baylisascaris* spp. larvae can cause extensive tissue damage, and all members of this genus are considered potential causes of visceral, ocular and/or neural larval migrans in mammals including humans and in birds.

This review focuses on three important *Baylisascaris* spp.: *B. transfuga* which occurs worldwide in bears, *B. schroederi*, a species which is pathogenic in its definitive host, the giant panda, and *B. procyonis*, of raccoons, which is

* Tel.: +49 641 9938463; fax: +49 641 9938469.
*E-mail address:* christian.bauer@vetmed.uni-giessen.de

0304-4017 
http://dx.doi.org/10.1016/j.vetpar.2012.12.036

**Table 1**
List of *Baylisascaris* spp., their natural definitive hosts and geographical distribution.

| Species | Definitive hosts | Geographical distribution |
|---|---|---|
| *B. ailuri* (Wu, He and Hu, 1987) | Red panda (*Ailurus fulgens*) | Asia (China) |
| *B. columnaris* (Leidy, 1856) | Skunks (*Mephitis* spp.) | North America |
| *B. devosi* (Sprent, 1952) | Mustelids (*Martes* spp.) | North America, eastern Europe, Asia (Russia) |
| *B. laevis* (Leidy, 1856) | Rodents (*Marmota* spp., *Citellus* spp.) | North America |
| *B. melis* (Gedoelst, 1920) | Badger (*Meles meles*) | Europe |
| *B. procyonis* (Stefanski and Zarnowski, 1951) | Raccoon (*Procyon lotor*), other procyonids (*Potos flavus*, *Bassaricyon gabbii*), domestic dog | North and possibly Central America, central Europe, Asia (Japan) |
| *B. schroederi* (McIntosh, 1939) | Giant panda (*Ailuropoda melanoleuca*) | Asia (China) |
| *B. tasmaniensis* (Sprent, 1970) | Carnivorous marsupials (*Sarcophilus harrisi*, and others) | Australia (Tasmania) |
| *B. transfuga* (Rudolphi, 1819) | Bears (*Ursus* spp.) | Arctic, North America, Europe, Asia |

the most pathogenic species for intermediate hosts, including man.

## 2. Baylisascaris transfuga

### 2.1. Geographic distribution, prevalence and intensity of infection

*B. transfuga* occurs worldwide in both free-ranging and captive bears such as the American black bear (Foster et al., 2004), European brown bear (De Ambrogi et al., 2011), and polar bear (Testini et al., 2011). In a recent survey 13% of 96 faecal samples from free-ranging European brown bears in Croatia contained ascarid eggs (De Ambrogi et al., 2011). In Florida Foster et al. (2004) reported black bear cubs harboured 1–39 roundworms in their intestines. Infected, captive bears may shed as many as 10,000–20,000 eggs per gram of faeces (Papini et al., 1994) and therefore heavily contaminate their domestic area.

### 2.2. Life cycle

The life cycle of *B. transfuga* is unclear but it is assumed that infections occur following ingestion of embryonated eggs from the environment. It is not known whether prey animal intermediate hosts are a source of *B. transfuga* infection for omnivorous or carnivorous bears under natural conditions. Because adult worms are first detected in bear cubs from five months of age (Foster et al., 2004) intrauterine or lactogenic transmission of larvae is unlikely to occur. The endogenous development of *B. transfuga* in definitive hosts and the prepatent period remain to be elucidated.

### 2.3. Pathogenic importance

In bears, the pathogenicity of intestinal infection with *B. transfuga* appears low; one case of granulomatous peritonitis caused by roundworms was described in a cub (Szczepaniak et al., 2012). In contrast, it has been shown that in rodents, e.g. white mice and Mongolian gerbils, which are susceptible to experimental infection with *B. transfuga*, following infection the larvae migrate through different tissues growing and developing to the third-stage, causing various degrees of visceral, neural or ocular larva migrans (Sprent, 1953; Papini et al., 1996; Sato et al., 2004; Cho et al., 2007). Whereas experimentally infected chickens did not show clinical signs although larvae were present in tissues (Papini et al., 1993). There is anecdotal evidence from epidemiological observations that *B. transfuga* was the possible cause of an outbreak of fatal larva migrans in Japanese macaques (Sato et al., 2005). To date however, there is no unequivocal evidence of naturally occurring *B. transfuga* infection in non-ursid animals or humans.

## 3. Baylisascaris schroederi

### 3.1. Geographic distribution, prevalence and intensity of infection

Giant pandas are the definitive host of *B. schroederi*, and its geographical distribution is therefore restricted to China. In recent faecal surveys *B. schroederi* infections were detected in 54% of 126 and 48% of 31 free-ranging pandas examined by classical coprological and molecular biology methods, respectively (Zhang et al., 2011, 2012). Surprisingly, the faecal egg output was rather low (Zhang et al., 2011). Intestinal roundworm burdens varied from 1 to 619 worms in 11 animals at post mortem (Xue, 1987, cited by Zhang et al., 2012).

### 3.2. Life cycle

Because giant pandas are herbivorous animals, infection is assumed to occur by ingestion of embryonated eggs in soil or from faecally contaminated plant material (monoxenous cycle) (Wu et al., 1985, cited by Zhang et al., 2011). There is no evidence of intrauterine or lactogenic transmission of *B. schroederi* larvae from dam to cub. In experimentally infected mice, larvae did not migrate into the placenta (Li, 1990a). From studies in experimentally infected mice (Li, 1990a) and from post-mortem findings in pandas, it is postulated that *B. schroederi* larvae migrate through liver and lungs of pandas and may also

disseminate into other tissues. In mice, larvae grow and moult from second-stage to third-stage (450–1150 μm long, 21–60 μm wide) during the migration phase (Li, 1990a). No data about the prepatent period are available.

### 3.3. Pathogenic importance

*B. schroederi* is a pathogenic species in giant pandas. The parasite was reported to be the main cause of death of this endangered animal species in China between 2001 and 2005 (Zhang et al., 2008). Pandas can die of either visceral larva migrans (larvae found in liver, lungs, heart, or brain), or intestinal obstruction caused by large worm burdens (Loeffler et al., 2006; Zhang et al., 2008). *B. schroederi* was moderately pathogenic for mice after experimental infection (Li, 1990b); however, there is no evidence that it affects other animal species or humans under natural conditions.

## 4. Baylisascaris procyonis

Raccoons and related procyonids (Table 1), e.g. kinkajous (Kazacos et al., 2011), are the natural definitive hosts of *B. procyonis*. Domestic dogs, but not cats, are also susceptible to this roundworm and can shed eggs (Miyashita, 1993; Kazacos, 2001; Bowman et al., 2005). Unlike other *Baylisascaris* spp., *B. procyonis* is well documented as an important and frequent cause of visceral, ocular or neural larva migrans in mammals including humans and in birds. Dogs, but not raccoons, can also develop neural larva migrans following *B. procyonis* infection (Rudmann et al., 1996).

### 4.1. Geographic distribution, prevalence and intensity of infection

*B. procyonis* is an indigenous parasite of North American raccoons, especially in the eastern and Pacific regions of Canada and throughout the Midwest (including Texas), Northeast, Mid-Atlantic and Pacific States of the USA. In southeastern USA, *B. procyonis* was historically reported from the mountainous regions of Virginia, West-Virginia, Kentucky and Tennessee (Kazacos, 2001; Kresta et al., 2010; Chavez et al., 2012); however, recent studies have shown its geographic expansion into the southeast states (Blizzard et al., 2010a,b). The prevalence of infection in raccoon populations varies by region but may be 60% or more in some areas (Kazacos, 2001).

*B. procyonis* infection has also been identified in central Europe and Japan. Following the introduction of raccoons from North America into Germany, Russia and Poland many decades ago, this invasive mammal is now part of the endemic wildlife fauna in these countries (Beltrán-Beck et al., 2012)—in Germany, the hunting bag for the 2010/2011 season included more than 67,000 raccoons (Anonymous, 2012). *B. procyonis* is currently restricted to central Germany, where the parasite was detected in 71% of 147 free-ranging animals examined (Gey, 1998; Bauer, 2011). In western Poland, roundworm infections were found in free-ranging raccoons (Popiolek et al., 2011). In Japan, raccoons originally introduced as fur and pet animals have now become naturalised in almost all regions (Ikeda et al., 2004) and although *B. procyonis* infection has been frequently detected in captive animals there (Miyashita, 1993), none of 1688 free-ranging raccoons screened in Japan were found to be positive (Matoba et al., 2006).

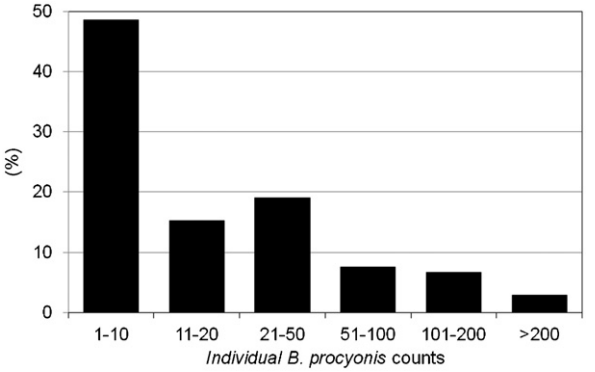


**Fig. 1.** Intensity of *Baylisascaris procyonis* infection in wild raccoons from central Germany.
(Data from: Gey, 1998).

Raccoons are often heavily infected. In Germany, for example, every sixth infected animal harboured at least 50 roundworms (Fig. 1). In many but not all studies, cubs were found to be more frequently infected than adult raccoons and had a higher intensity of infection (Snyder and Fitzgerald, 1987; Yeitz et al., 2009; Blizzard et al., 2010a). Only a few *B. procyonis* specimens (1–13 worms) were isolated from the intestines of domestic dogs in the USA (Kazacos, 2001).

### 4.2. Life cycle

The life cycle of *B. procyonis* is that of a facultative heteroxenous parasite (Kazacos, 2001; Fig. 2); definitive hosts becoming infected either by ingesting eggs, or by consuming an intermediate host. It is generally assumed that cubs acquire infection by ingesting embryonated eggs from the contaminated environment. After hatching the 300 μm long second-stage larvae (Sakla et al., 1989) penetrate the intestinal wall where they develop during a histotropic phase of several weeks; finally the pre-adult stages return to the intestinal lumen to mature. Egg shedding starts 50–76 days after infection. Adult raccoons may also become infected by ingesting third-stage larvae from prey intermediate hosts after which further development to mature worms occurs in the intestinal lumen resulting in a shorter prepatent period of 32–38 days. Extensive larval migration does not seem to occur in definitive hosts (Kazacos, 2001), although detailed confirmatory studies are lacking. Ascarid larvae have, however, occasionally been found in extraintestinal tissues of raccoons (Cranfield et al., 1984). Failure to isolate ascarid larvae from organs of several newborn raccoons using pepsin–HCl digestion (C. Bauer, unpublished, 1992) suggests that intrauterine transmission of *B. procyonis* larvae does not occur.

When an intermediate host ingests embryonated eggs, the second-stage larvae hatch, penetrate the intestinal wall and migrate through the liver to the lungs and via blood to other tissues. A few larvae (5% or so) may enter the

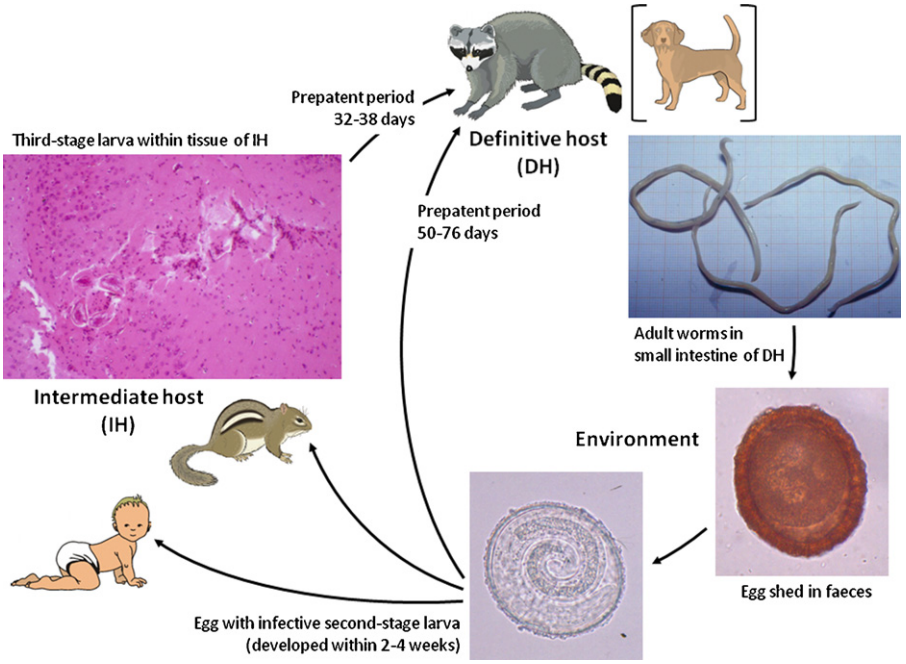


**Fig. 2.** Life cycle of *Baylisascaris procyonis*.

central nervous system including the eyes (Tiner, 1953; Sheppard and Kazacos, 1997). In the tissues of intermediate hosts, larvae grow and moult to the third-stage, reaching 1300–1900 μm in length and 50–80 μm in width 2–4 weeks after infection (Bowman, 1987; Donnelly et al., 1989; Goldberg et al., 1993).

More than 100 animal species including humans are known to act as intermediate hosts or 'dead-end' hosts of *B. procyonis*; they commonly develop clinical signs of larva migrans. The list includes mammals (rodents, lagomorphs, a few carnivore species, primates, and marsupials) and birds (Galliformes, Columbiformes, Passeriformes, Psittaciformes, and Struthioniformes). A few domestic animal species such as pigs, small ruminants and cats are less susceptible to *B. procyonis*. Infections have not been reported in reptiles, amphibians and fish (Kazacos, 2001).

### 4.3. Epidemiological aspects

Kazacos (2001) stated "wherever raccoons occur or are introduced, the potential exists for disease caused by *B. procyonis*". Female *B. procyonis* worms can produce huge numbers of eggs. Snyder and Fitzgerald (1987) estimated average egg outputs of 115,000–179,000 eggs per day, suggesting heavily infected raccoons (Fig. 1) may shed >1,000,000 eggs per day. This can result in heavy environmental contamination even considering that shedding of eggs is intermittent (Reed et al., 2012). *B. procyonis* eggs remain infective for years (Lindquist, 1978) and can withstand freezing temperatures of −15 °C (Shafir et al., 2011). The role of invertebrates as possible mechanical vectors for disseminating eggs in the environment is unknown.

Free-ranging raccoons habitually defecate at preferred sites ('latrines') where *B. procyonis* eggs can accumulate. These latrines are commonly near the raccoon resting and sleeping places (tree holes, crotches, wood pile, straw; in settlements: barn lofts, attics, chimneys, garages, sheds, drains, etc.). In *B. procyonis* endemic regions, soil around latrines (Evans, 2002; Roussere et al., 2003; Page et al., 2009) as well as wood and wood chips from trees (Van Andel et al., 1995) and straw and hay previously used by raccoons (Richardson et al., 1980) has been found to be highly contaminated with infective eggs. Because raccoons are omnivores their faeces usually contain undigested seeds and grain making raccoon latrines attractive for foraging granivorous mammals and birds (Page et al., 2001). This, of course, increases the risk of these animals becoming fatally infected with *B. procyonis*. Results of studies focussing on this topic strongly suggest that *B. procyonis* has contributed to the extirpation of the Allegheny woodrat (*Neotoma magister*) from several regions in eastern North America (LoGiudice, 2003; Page et al., 2012). Approximately 5% of deaths of white-footed mouse (*Peromyscus leucopus*) in Illinois have been attributed to the raccoon roundworm (Tiner, 1954). *B. procyonis* larvae can survive in carcasses of intermediate hosts for several days (Sprent, 1953) increasing the chances of the definitive hosts becoming infected as raccoons are scavengers and can prey on debilitated or dead intermediate hosts, consuming ascarid larvae in the tissues.

Raccoons live in a wide range of habitats and have colonised cultivated farmland and more urban areas in close proximity to humans. In North America and Germany their population densities in suburban and urban areas which offer rich food sources, are often much higher than

in adjacent rural or forest areas (Ghert, 2003; Bauer, 2011). In the suburbs of Chicago raccoon latrines occurred in 51% of backyards examined, and *B. procyonis* eggs were found in 23% of those latrines (Page et al., 2009). This close association with people results in an increased risk of transmission to humans and other animals kept in urban environments (e.g. in zoological gardens). Several human cases of neural larva migrans caused by *B. procyonis* have been reported from suburban areas (Wise et al., 2005). Enclosures, cages and aviaries previously used to house raccoons are often contaminated with *B. procyonis* eggs and remain a source of infection for other animals thereafter (Kazacos, 2001). The coats of infected raccoons may also contain embryonated *B. procyonis* eggs as observed for dog and fox hair contaminated with *Toxocara* spp. eggs (Overgaauw et al., 2009; Keegan and Holland, 2010; Nagy et al., 2011); if so, direct contact with the animals could be another possible source of infection. However it should be noted that direct contact with fur of dogs, cats or foxes is rather unlikely to be a significant route of *Toxocara* spp. transmission, since eggs need several weeks to embryonate and become infective and only a low percentage of eggs harvested from fur appear viable; furthermore the adhesive properties of *Toxocara* spp. eggs mean that significant quantities of hair probably need to be consumed to pose a realistic infection risk (Overgaauw et al., 2009; Keegan and Holland, 2010, in press; Deplazes et al., 2011; Nagy et al., 2011). The same caveats probably apply to *B. procyonis* infection from raccoon fur.

### 4.4. Pathology and clinical signs

In definitive hosts infections are generally asymptomatic, however high worm burdens may occasionally cause intestinal obstruction and death (Carlson and Nielsen, 1984). In contrast, in intermediate hosts *B. procyonis* larva migrans usually results in progressive, neurological and/or ocular disease within a few days or weeks of infection. The severity of clinical signs depends on the number, extent and location of the migrating larvae; in small animals, e.g. mice and small birds, a single *B. procyonis* larva in the brain is usually fatal (Tiner, 1953; Armstrong et al., 1989).

Following infection, the larvae quickly migrate to liver, lungs and then to other tissues causing haemorrhagic or necrotic lesions and eosinophilic inflammation (hepatitis, pneumonia, myocarditis, and myositis). They become encapsulated in eosinophilic granulomas in extraneural tissues in mammals (Tiner, 1953; Sheppard and Kazacos, 1997). Extraneural granulomas are less common in birds, the larvae being mostly confined to the brain (Richardson et al., 1980; Armstrong et al., 1989). When larvae enter the brain encapsulation is delayed and the prolonged migration results in extensive damage (Fig. 3), although only a few *B. procyonis* larvae usually invade the central nervous system. Histopathological alterations include haemorrhagic migration tracks, focal or diffuse eosinophilic meningoencephalitis, malacia, necrosis, spongiosis, and occasionally myelitis (Kazacos, 2001; Gavin et al., 2005). The pathogenicity of *B. procyonis* larva migrans is thought to be the result of several factors. The larvae grow rapidly (Donnelly et al., 1989; Goldberg et al., 1993) and do not usually become encapsulated in the brain, prolonging their migration (Tiner, 1953; Sheppard and Kazacos, 1997), which may result in mechanical damage and tissue necrosis. Additionally, the larvae produce excretory–secretory antigens which trigger a massive eosinophil-associated host immune reaction, releasing a neurotoxin that probably contributes to the pathology and clinical signs (Moertel et al., 2001).

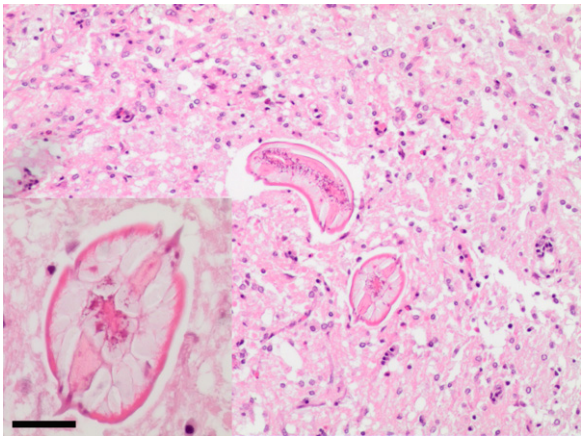

**Fig. 3.** Neural larva migrans caused by *Baylisascaris procyonis* in a naturally infected rabbit (C. Bauer, 2003, unpublished). Section of the brain (H&E stain) containing tangential and transversal sections of a larva; in inset, note the paired lateral alae, excretory columns and laterally compressed intestine (scale bar, 25 μm).

Neurological disease has been reported in most but not all cases of *B. procyonis* infection in animals and humans. A wide variety of clinical signs can be observed including sudden lethargy, circling, ataxia, paralysis, tremor, seizures, torticollis, opisthotonus, nystagmus, dysphagia, and stupor progressing to coma, and in birds difficulty in perching (Kazacos, 2001; Gavin et al., 2005). Death usually occurs a few weeks after onset of signs. When a larva invades an eye typically unilateral signs are granulomatous chorioretinitis, optic nerve atrophy, retinal depigmentation and visual loss (Kazacos, 2001; Gavin et al., 2005).

### 4.5. Human baylisascariosis

Four different manifestations of *B. procyonis* infection have been described in humans: neural larva migrans, ocular larva migrans, visceral larva migrans, and subclinical baylisascariosis. The *intra vitam* diagnosis of *B. procyonis* larva migrans is difficult and is based on some or all of the following; clinical signs, history of exposure to raccoons, neuroimaging, laboratory findings, detection of specific serum antibodies and detection of larvae in needle aspiration brain biopsies (Gavin et al., 2005). An ELISA (Boyce et al., 1988) and immunoblotting (Conraths et al., 1996; Dangoudoubiyam and Kazacos, 2009), both based on excretory–secretory antigens from second-stage *B. procyonis* larvae have been used for serological testing. Recently, an ELISA with recombinant RAG1 antigen from *B. procyonis* third-stage larvae has been developed (Dangoudoubiyam et al., 2010). In cases of ocular larva migrans a single large,

**Table 2**
Efficacy of anthelmintic compounds tested against *Baylisascaris procyonis* in raccoons or dogs.

| Anthelmintic compound | Treatment days × dosage (mg/kg) | Application route | Cure rate | Reference |
|---|---|---|---|---|
| Albendazole | 3 × 50 | p.o. (food) | 7/7[a] | Bauer and Gey (1995) |
| Fenbendazole | 3 × 50 | p.o. (food) | 7/7[a] | Bauer and Gey (1995) |
| Flubendazole | 3 × 22 | p.o. (food) | 7/7[a] | Bauer and Gey (1995) |
| Ivermectin | 1 × 1 | p.o. (food) | 7/7[a] | Bauer and Gey (1995) |
| | 1 × 2 | i.m. | 9/10[a] | Hill et al. (1991) |
| Milbemycin oxime | 1 × 0.5–1.0 | p.o. (tablet) | 6/8[b] | Bowman et al. (2005) |
| Moxidectin | 1 × 1 | p.o. (food) | 7/7[a] | Bauer and Gey (1995) |
| Piperazine citrate | 1 × 120–140 | p.o. | 'Successfully'[a] | Kazacos (2001) |
| Pyrantel | 1 × 20 | p.o. (food) | 7/7[a] | Bauer and Gey (1995) |

[a] Raccoon.
[b] Dog.

motile larva may occasionally be detected in the retina by ophthalmoscopic examination (Fig. 4; Küchle et al., 1993; Saffra et al., 2010). Post mortem diagnosis can be made by histopathological findings and detection of *B. procyonis* larvae (Fig. 3; Goldberg et al., 1993) or *B. procyonis*-specific DNA using PCR in tissue samples (Dangoudoubiyam et al., 2009).

Clinical neural larva migrans has been diagnosed in at least 23 humans from North America to date (Wise et al., 2005; Bauer, 2011; Mehta et al., 2010; Haider et al., 2012). Patients were mainly toddlers or young children (13 of 23 aged <2 years) or individuals with mental disability or developmental impairment (7 of 23), almost all (20 of 23) were male. Pica or geophagia was reported in most patients, and this behaviour was considered the most likely route of infection. Neural larva migrans is generally assumed to be the result of ingesting large numbers of eggs, for example, the brain of one 18-month-old child who died contained numerous *B. procyonis* larvae (185 larvae in 60 g tissue; Fox et al., 1985). A significant number of the human cases of neural larva migrans caused by *B. procyonis* were fatal (6 of 23) or resulted in more or less severe neurological sequelae. There is only one report in the literature of full recovery of a 4-year-old boy, this followed very early intervention with targeted treatment using albendazole and a high-dose corticosteroid daily for weeks (Pai et al., 2007).

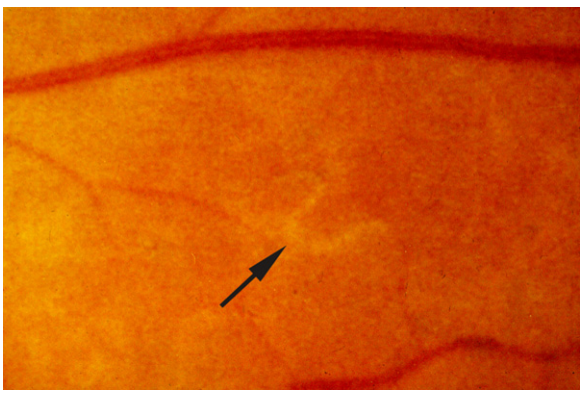

**Fig. 4.** Ocular larva migrans caused by *Baylisascaris procyonis* in a human patient with diffuse unilateral subacute neuroretinitis syndrome; the patient had kept a raccoon as pet indoors (Küchle et al., 1993). Portion of fundus photograph with an intraretinal, S-shaped, approximately 1500 μm long larva (arrow).

Ocular larva migrans can occur with or without other clinical manifestations. Cases without other signs are assumed to be the result of infection with few *B. procyonis* larvae. The raccoon roundworm is considered the most common cause of the 'diffuse unilateral subacute neuroretinitis syndrome' (Gavin et al., 2005). Progressive inflammatory and degenerative alterations in the retina and optic disc usually affect only one eye. The disease has been observed in both children and adults. Most cases have been reported from North America but one case has been reported in Germany; the patient had kept a raccoon as pet indoors (Fig. 4; Küchle et al., 1993). Laser photocoagulation, sometimes combined with a systemic corticosteroid, has been successfully used to destroy the intraretinal larva and diminish the inflammation (Küchle et al., 1993; Saffra et al., 2010).

A few cases of visceral larva migrans have been described in young children associated with nonspecific clinical signs (fever, lethargy, nausea, macular rash, pneumonia, and hepatomegaly). Clinical signs and internal pathology are caused by extensive extraneural migration of *B. procyonis* larvae (Gavin et al., 2005). In a German case-control study two patients with previous contact to infected raccoons had antibodies to *B. procyonis* detected using immunoblotting and elevated serum levels of immunoglobulin E and specific liver enzymes, consistent with visceral larva migrans (Conraths et al., 1996).

Baylisascariosis can also occur without any symptoms. In the *B. procyonis*-endemic region of Chicago, 8% of 389 children screened by ELISA had antibodies to *B. procyonis* but no history of disease (Brinkman et al., 2003). In a case-control study performed in Germany, a few individuals with previous contact to roundworm-infected raccoons or their faeces, were positive for anti-*B. procyonis* antibodies using immunoblotting, but no disease was reported (Conraths et al., 1996). Several *B. procyonis*-like larvae surrounded by mild chronic reaction were found in the brain of an elderly patient who suffered from Alzheimer dementia but had been in good health until dying of cardiac arrest (Hung et al., 2012).

### 4.6. Prevention and control

The most effective means of preventing infection in people and animals is to avoid contact with raccoons and their faeces. Practical control measurements should include

identification and rapid elimination of raccoon latrines from backyards (including the soil below latrines) and buildings (attics, barns, etc.) by burning using a propane torch or by steam cleaning with boiling water (Vantassel, 2012). Heat is the best method of killing *B. procyonis* eggs, which become unviable at temperatures above 62 °C (Shafir et al., 2011). Children should not be allowed to play in the areas likely to be contaminated with raccoon faeces. Raccoon populations should be reduced by hunting and other measures. The results of a recent pilot project suggest that *B. procyonis* infection might be reduced in raccoon populations by a combination of heat disinfection of raccoon latrines and the laying of food baits containing an anthelmintic compound (Page et al., 2011).

If raccoons are kept as pet animals or for public display in zoological gardens and other enterprises, preventive measurements are indispensable to reduce the risk of transmission of *B. procyonis* to other animals and humans. Recently captured raccoons should be kept in quarantine and dewormed. For this, several anthelmintic compounds with proven high efficacy after oral application are available (Table 2).

## Conflict of interest statement

None.

## References


Anonymous, 2012. Jahresstrecken Neozoen. www.jagdnetz.de/datenundfakten/jahresstrecken

Armstrong, D.L., Montali, R.J., Doster, A.R., Kazacos, K.R., 1989. Cerebrospinal nematodiasis in macaws due to *Baylisascaris procyonis*. J. Zoo Wildl. Med. 20, 354–359.

Bauer, C., 2011. Baylisascariosis (*Baylisascaris procyonis*)—a rare parasitic zoonosis in Europe. Berl. Münch. Tierärztl. Wochenschr. 124, 465–472 (in German).

Bauer, C., Gey, A., 1995. Efficacy of six anthelmintics against luminal stages of *Baylisascaris procyonis* in naturally infected raccoons (*Procyon lotor*). Vet. Parasitol. 60, 155–159.

Beltrán-Beck, B., García, F.J., Gortázar, C., 2012. Raccoons in Europe: disease hazards due to the establishment of an invasive species. Eur. J. Wildl. Res. 58, 5–15.

Blizzard, E.L., Davis, C.D., Henke, S., Long, D.B., Hall, C.A., Yabsley, M.J., 2010a. Distribution, prevalence, and genetic characterization of *Baylisascaris procyonis* in selected areas of Georgia. J. Parasitol. 96, 1128–1133.

Blizzard, E.L., Yabsley, M.J., Beck, M.F., Harsch, S., 2010b. Geographic expansion of *Baylisascaris procyonis* roundworms, Florida, USA. Emerg. Infect. Dis. 16, 1803–1804.

Bowman, D.D., 1987. Diagnostic morphology of four larval ascaridoid nematodes that may cause visceral larva migrans: *Toxascaris leonina*, *Baylisascaris procyonis*, *Lagochilascaris sprenti*, and *Hexametra leidyi*. J. Parasitol. 73, 1198–1215.

Bowman, D.D., Ulrich, M.A., Gregory, D.E., Neumann, N.R., Legg, W., Stansfield, D., 2005. Treatment of *Baylisascaris procyonis* infections in dogs with milbemycin oxime. Vet. Parasitol. 129, 285–290.

Boyce, W.M., Branstetter, B.A., Kazacos, K.R., 1988. Comparative analysis of larval excretory–secretory antigens of *Baylisascaris procyonis*, *Toxocara canis* and *Ascaris suum* by western blotting and enzyme immunoassay. Int. J. Parasitol. 18, 109–113.

Brinkman, W.B., Kazacos, K.R., Gavin, P.J., Binns, H.J., Robichaud, J.D., O'Gorman, M., Shulman, S.T., 2003. Seroprevalence of *Baylisascaris procyonis* (raccoon roundworm) in Chicago area children. In: Proc. 2003 Ann. Meet. Pediatr. Acad. Soc., Seattle (Abstr. no. 1872).

Bruňaská, M., Dubinský, P., Reiterová, K., 1995. *Toxocara canis*: ultrastructural aspects of larval moulting in the maturing eggs. Int. J. Parasitol. 25, 683–690.

Carlson, B.L., Nielsen, S.W., 1984. Jejunal obstruction due to *Baylisascaris procyonis* in a raccoon. J. Am. Vet. Med. Assoc. 185, 1396–1397.

Chavez, D.J., LeVan, I.K., Miller, M.W., Ballweber, L.R., 2012. *Baylisascaris procyonis* in raccoons (*Procyon lotor*) from eastern Colorado, an aera of undefined prevalence. Vet. Parasitol. 185, 330–334.

Cho, S., Egami, M., Ohnuki, H., Saito, Y., Chinone, S., Shichinohe, K., Suganuma, M., Akao, N., 2007. Migration behavior and pathogenesis of five ascarid nematode species in the Mongolian gerbil *Meriones unguiculatus*. J. Helminthol. 81, 43–47.

Conraths, F.J., Bauer, C., Cseke, J., Laube, H., 1996. Arbeitsplatzbedingte Infektionen des Menschen mit dem Waschbärspulwurm *Baylisascaris procyonis*. Arbeitsmed. Sozialmed. Umweltmed. 31, 13–17.

Cranfield, M.R., Barker, I.K., Mehren, K.G., Rapley, W.A., 1984. Canine distemper in wild raccoons (*Procyon lotor*) at the Metropolitan Toronto Zoo. Can. Vet. J. 25, 63–66.

Dangoudoubiyam, S., Kazacos, K.R., 2009. Differentiation of larva migrans caused by *Baylisascaris procyonis* and *Toxocara* species by Western blotting. Clin. Vaccine Immunol. 16, 1563–1568.

Dangoudoubiyam, S., Vemulapalli, R., Hancock, K., Kazacos, K.R., 2010. Molecular cloning of an immunogenic protein of *Baylisascaris procyonis* and expression in *Escherichia coli* for use in developing improved serodiagnostic assays. Clin. Vaccine Immunol. 17, 1933–1939.

Dangoudoubiyam, S., Vemulapalli, R., Kazacos, K.R., 2009. PCR assays for detection of *Baylisascaris procyonis* eggs and larvae. J. Parasitol. 95, 571–577.

De Ambrogi, M., Aghazadeh, M., Hermosilla, C., Huber, D., Majnaric, D., Reljic, S., Elson-Riggins, J., 2011. Occurrence of *Baylisascaris transfuga* in wild populations of European brown bears (*Ursus arctos*) as identified by a new PCR method. Vet. Parasitol. 179, 272–276.

Deplazes, P., Van Knapen, F., Schweiger, A., Overgaauw, P.A.M., 2011. Role of pet dogs and cats in the transmission of helminthic zoonoses in Europe, with a focus on echinococcosis and toxocarosis. Vet. Parasitol. 182, 41–53.

Donnelly, J.J., Sakla, A.A., Khatami, M., Rockey, J.H., 1989. *Baylisascaris procyonis* (Stiefanski and Zarnowski, 1951) Ascarididae: Nematoda. II. Third stage larvae, morphogenesis and migratory behaviour. Assiut Vet. Med. J. 21, 77–91.

Evans, R.H., 2002. *Baylisascaris procyonis* (Nematoda: Ascaridoidea) eggs in raccoon (*Procyon lotor*) latrine scats in Orange County, California. J. Parasitol. 88, 189–190.

Foster, G.W., Cunningham, M.W., Kinsella, J.M., Forrester, D.J., 2004. Parasitic helminths in black bear cubs (*Ursus americanus*) from Florida. J. Parasitol. 90, 173–175.

Fox, A.S., Kazacos, K.R., Gould, N.S., Heydemann, P.T., Thomas, C., Boyer, K.M., 1985. Fatal eosinophilic meningoencephalitis and visceral larva migrans caused by the raccoon ascarid *Baylisascaris procyonis*. N. Engl. J. Med. 312, 1619–1623.

Gavin, P.J., Kazacos, K.R., Shulman, S.T., 2005. Baylisascaris. J. Clin. Microbiol. 18, 703–718.

Geenen, P.L., Bresciani, J., Boes, J., Pedersen, A., Eriksen, L., Fagerholm, H.-P., Nansen, P., 1999. The morphogenesis of *Ascaris suum* to the infective third-stage larvae within the egg. J. Parasitol. 85, 616–622.

Gey, A.B., 1998. Synopsis der Parasitenfauna des Waschbären (*Procyon lotor*) unter Berücksichtigung von Befunden aus Hessen. Dr. med. vet. Thesis. Justus Liebig University, Giessen. 202 pp.

Ghert, S.D., 2003. Raccoon (*Procyon lotor*) and allies. In: Feldhamer, G.A., Thompson, B.C., Chapman, J.A. (Eds.), Wild Mammals of North America. , 2nd ed. John Hopkins University Press, Baltimore, pp. 611–634.

Goldberg, M.A., Kazacos, K.R., Boyce, W.M., Ai, E., Katz, B., 1993. Diffuse unilateral subacute neuroretinitis. Morphometric, serologic, and epidemiologic support for *Baylisascaris* as a causative agent. Ophthalmology 100, 1695–1701.

Haider, S., Khairnar, K., Martin, D.S., Yang, J., Ralenvski, F., Kazacos, K.R., Pillai, D.R., 2012. Possible pet-associated baylisascariasis in child, Canada. Emerg. Infect. Dis. 18, 347–349.

Hill, R.E., Zimmerman, J.J., Greve, J.H., Beran, G.W., 1991. Use of ivermectin against several nematodes in naturally infected raccoons (*Procyon lotor*). J. Zoo Wildl. Med. 22, 417–420.

Hung, T., Neafie, R.C., Mackenzie, I.R.A., 2012. *Baylisascaris procyonis* infection in elderly person, British Columbia, Canada. Emerg. Infect. Dis. 18, 341–342.

Ikeda, T., Asano, M., Matoba, Y., Abe, G., 2004. Present status of invasive alien raccoon and its impact in Japan. Global Environ. Res. 8, 125–131.

Kazacos, K.R., 2001. *Baylisascaris procyonis* and related species. In: Samuel, W.M., Pybus, M.J., Kocan, A.A. (Eds.), Parasitic Diseases of Wild Animals. , 2nd ed. Manson, London, pp. 301–341.

Kazacos, K.R., Kilbane, T.P., Zimmerman, K.D., Chavez-Lindell, T., Parman, B., Lane, T., Carpenter, L.R., Green, A.L., Mann, P.M., Murphy, T.W., Bertucci, B., Gray, A.C., Goldsmith, T.L., Cunningham, M., Stanek, D.R., Blackmore, C., Yabsley, M.J., Montgomery, S.P., Bosserman, E., 2011.

Raccoon roundworms in pet kinkajous—three States, 1999 and 2010. Morb. Mortal. Wkly. Rep. 60, 302–305.
Keegan, J.D., Holland, C.V., 2010. Contamination of the hair of owned dogs with the eggs of *Toxocara* spp. Vet. Parasitol. 173, 161–164.
Keegan, J.D., Holland, C.V. A comparison of *Toxocara canis* embryonation under controlled conditions in soil and hair. J. Helminthol., http://dx.doi.org/10.1017/S0022149X12000065, in press.
Kresta, A.E., Henke, S.E., Pence, D.B., 2010. *Baylisascaris procyonis* in raccoons in Texas and its relationship to habitat characteristics. J. Wildl. Dis. 46, 843–853.
Küchle, M., Knorr, H.L.J., Medenblik-Frysch, S., Weber, A., Bauer, C., Naumann, G.O.H., 1993. Diffuse unilateral subacute neuroretinitis syndrome in a German most likely caused by the raccoon roundworm, *Baylisascaris procyonis*. Graefes Arch. Clin. Exp. Ophthalmol. 231, 48–51.
Li, J.H., 1990a. Migration, distribution and development of larvae of panda ascarid, *Baylisascaris schroederi* in mice (in Chinese). Acta Zool. Sin. 36, 236–243.
Li, J.H., 1990b. Observations on the pathogenicity of *Baylisascaris schroederi* in experimental mice. Chin. J. Zoonoses 6 (4), 32–34 (in Chinese).
Lindquist, W.D., 1978. *Baylisascaris procyonis* for testing anthelmintics against migratory ascarids. Am. J. Vet. Res. 39, 1868–1869.
Loeffler, I.K., Montali, R.J., Rideout, B.A., 2006. Diseases and pathology of giant pandas. In: Wildt, D.E., Zhang, A.J., Zhang, H.M., Janssen, D.L., Ellis, S. (Eds.), Giant Pandas: Biology, Veterinary Medicine and Management. Cambridge University Press, New York, pp. 388–390.
LoGiudice, K., 2003. Trophically transmitted parasites and the conservation of small populations: raccoon roundworm and the imperiled Allegheny woodrat. Conserv. Biol. 17, 258–266.
Matoba, Y., Yamada, D., Asano, M., Oku, Y., Kitaura, K., Yagi, K., Tenora, F., Asakawa, M., 2006. Parasitic helminths from feral raccoons (*Procyon lotor*) in Japan. Helminthologia 43, 139–146.
Mehta, P., Boyd, Z., Cully, B., 2010. Raccoon roundworm encephalitis. Pediatr. Radiol. 40, 1834–1836.
Miyashita, M., 1993. Prevalence of *Baylisascaris procyonis* in raccoons in Japan and experimental infections of the worm to laboratory animals. J. Urban Living Health Assoc. 37, 137–151 (in Japanese).
Moertel, C.L., Kazacos, K.R., Butterfield, J.H., Kita, H., Watterson, J., Gleich, G.J., 2001. Eosinophil-associated inflammation and elaboration of eosinophil-derived proteins in 2 children with raccoon roundworm (*Baylisascaris procyonis*) encephalitis. Pediatrics 108, e93.
Nagy, A., Ziadinov, I., Schweiger, A., Schnyder, M., Deplazes, P., 2011. Fellkontamination mit Eiern von zoonotischen Helminthen bei Hof- und Haushunden sowie bei Füchsen. Berl. Münch. Tierärztl. Wochenschr. 124, 503–511.
Overgaauw, P.A.M., Van Zutphen, L., Hoek, D., F.O., Roelfsema, J., Pinelli, E., Van Knapen, F., Kortbeek, L.M., 2009. Zoonotic parasites in fecal samples and fur from dogs and cats in The Netherlands. Vet. Parasitol. 163, 115–122.
Page, L.K., Swihart, R.K., Kazacos, K.R., 2001. Seed preferences and foraging by granivores at raccoon latrines in the transmission dynamics of the raccoon roundworm (*Baylisascaris procyonis*). Can. J. Zool., 616–622.
Page, L.K., Anchor, C., Luy, E., Kron, S., Larson, G., Madsen, L., Kellner, K., Smyser, T.J., 2009. Backyard raccoon latrines and risk for *Baylisascaris procyonis* transmission to humans. Emerg. Infect. Dis. 15, 1530–1531.
Page, K., Beasley, J.C., Olson, Z.H., Smyser, T.J., Downey, M., Kellner, K.F., McCord, S.E., Egan, T.S., Rhodes, O.E., 2011. Reducing *Baylisascaris procyonis* roundworm larvae in raccoon latrines. Emerg. Infect. Dis. 17, 90–93.
Page, L.K., Johnson, S.A., Swihart, R.K., Kazacos, K.R., 2012. Prevalence of *Baylisascaris procyonis* in habitat associated with Allegheny woodrat (*Neotoma magister*) in populations in Indiana. J. Wildl. Dis. 48, 503–507.
Pai, P.J., Blackburn, B.R., Kazacos, K.R., Warrier, R.P., Bégué, R.E., 2007. Full recovery from *Baylisascaris procyonis* eosinophilic meningitis. Emerg. Infect. Dis. 13, 928–930.
Papini, R., Casarosa, L., 1994. Observations on the infectivity of *Baylisascaris transfuga* eggs for mice. Vet. Parasitol. 51, 283–288.
Papini, R., Cavicchio, P., Casarosa, L., 1993. Experimental infection in chickens with larvae of *Baylisascaris transfuga* (Nematoda: Ascaridoidea). Folia Parasitol. 40, 141–143.
Papini, R., Cavicchio, P., Casarosa, L., 1994. Effet ovicide in vivo mébendazole sur les œufs de *Baylisascaris transfuga*. Rev. Méd. Vét. 145, 55–57.
Papini, R., Renzoni, G., Lo Piccolo, S., Casarosa, L., 1996. Ocular larva migrans and histopathological lesions in mice experimentally infected with *Baylisascaris transfuga* embryonated eggs. Vet. Parasitol. 61, 315–320.
Popiolek, M., Szczesna-Staśkiewicz, J., Bartoszewicz, M., Okarma, H., Smalec, B., Zalewski, A., 2011. Helminth parasites of an introduced invasive carnivore species, the raccoon (*Procyon lotor* L.), from the Warta Mouth National Park (Poland). J. Parasitol. 97, 357–360.
Reed, C., Henke, S.E., Kresta, A.E., 2012. Frequency of deposition and location of *Baylisascaris procyonis* eggs in raccoon feces. J. Wildl. Dis. 48, 190–194.
Richardson, J.A., Kazacos, K.R., Thacker, H.L., Dhillon, A.S., Winterfield, R.W., 1980. Verminous encephalitis in commercial chickens. Avian Dis. 24, 498–503.
Roussere, G.P., Murray, W.J., Raudenbush, C.B., Kutilek, M.J., Levee, D.J., Kazacos, K.R., 2003. Raccoon roundworm eggs near homes and risk for larva migrans disease, California communities. Emerg. Infect. Dis. 9, 1516–1522.
Rudmann, D.G., Kazacos, K.R., Storandt, S.T., Harris, D.L., Janovitz, E.B., 1996. *Baylisascaris procyonis* larva migrans in a puppy: a case report and update for the veterinarian. J. Am. Anim. Hosp. Assoc. 32, 73–76.
Saffra, N.A., Perlman, J.E., Desai, R.U., Kazacos, K.R., Coyle, C.M., Machado, F.S., Kedhar, S.R., Engelbert, M., Tannowitz, H.B., 2010. *Baylisascaris procyonis* induced diffuse unilateral subacute neuroretinitis in New Youk City. J. Neuroparasitol. 1, N100401.
Sakla, A.A., Donnelly, J.J., Khatami, M., Rockey, J.H., 1989. *Baylisascaris procyonis* (Stefanski and Zarnowski, 1951) Ascarididae: Nematoda. I. Embryonic development and morphogenesis of second stage larvae. Assiut Vet. Med. J. 21, 68–75.
Sato, H., Matsuo, K., Osanai, A., Kamiya, H., Akao, N., Owaki, S., Furuoka, H., 2004. Larva migrans by *Baylisascaris transfuga*: fatal neurological diseases in Mongolian jirds, but not in mice. J. Parasitol. 90, 774–781.
Sato, H., Une, Y., Kawakami, S., Saito, E., Kamiya, H., Akao, N., Furuoka, H., 2005. Fatal *Baylisascaris* larva migrans in a colony of Japanese macaques kept by a safari-style zoo in Japan. J. Parasitol. 91, 716–719.
Schnieder, T., Laabs, E.-M., Welz, C., 2011. Larval development of *Toxocara canis* in dogs. Vet. Parasitol. 175, 193–206.
Shafir, S.C., Sorvillo, F.J., Sorvillo, T., Eberhard, M.L., 2011. Viability of *Baylisascaris procyonis* eggs. Emerg. Infect. Dis. 17, 1293–1295.
Sheppard, C., Kazacos, K.R., 1997. Susceptibility of *Peromyscus leucopus* and *Mus musculus* to infection with *Baylisascaris procyonis*. J. Parasitol. 83, 1104–1111.
Snyder, D.E., Fitzgerald, P.R., 1987. Contaminative potential, egg prevalence, and intensity of *Baylisascaris procyonis*-infected raccoon (*Procyon lotor*) from Illinois, with a comparison to worm intensity. Proc. Helminthol. Soc. Wash. 54, 141–145.
Sprent, J.F.A., 1953. On the migratory behavior of larvae of various ascarid species in white mice. II. Longevity of encapsulated larvae and their resistance to freezing and putrefaction. J. Infect. Dis. 92, 114–117.
Sprent, J.F.A., 1968. Notes on *Ascaris* and *Toxascaris*, with a definition of *Baylisascaris* gen. nov. Parasitology 58, 185–198.
Szczepaniak, K., Listos, P., Lopuszynski, W., Skrzypek, T., Kazimierczak, W., 2012. Granulomatous peritonitis in a European brown bear caused by *Baylisascaris transfuga*. J. Wildl. Dis. 48, 517–519.
Testini, G., Papini, R., Lia, R.P., Parisi, A., Dantas-Torres, F., Traversa, D., Otranto, D., 2011. New insights into the morphology, molecular characterization and identification of *Baylisascaris transfuga* (Ascaridida, Ascarididae). Vet. Parasitol. 175, 97–102.
Tiner, J.D., 1953. Fatalities in rodents cased by larval *Ascaris* in the central nervous system. J. Mammal. 34, 153–167.
Tiner, J.D., 1954. The fraction of *Peromyscus leucopus* fatalities caused by raccoon ascarid larvae. J. Mammal. 35, 589–592.
Van Andel, R.A., Franklin, C.L., Besch-Williford, C., Riley, L.K., Hook, R.R., Kazacos, K.R., 1995. Cerebrospinal larva migrans due to *Baylisascaris procyonis* in a guinea pig colony. Lab. Anim. Sci. 45, 27–30.
Vantassel, S.M., 2012. A draft proposal for an industry protocol for the cleanup of raccoon latrines. In: Proc. 14th Wildl. Damage Manage. Conf., Nebraska City, 18–21 April 2011, pp. 33–39.
Wise, M.E., Sorvillo, F.J., Shafir, S.C., Ash, L.R., Berlin, O.G., 2005. Severe and fatal central nervous system disease in humans caused by *Baylisascaris procyonis*, the common roundworm of raccoons: a review of current literature. Microbes Infect. 7, 317–323.
Xie, Y., Zhang, Z., Niu, L., Wang, Q., Wang, C., Lan, J., Deng, J., Fu, Y., Nie, H., Yan, N., Yang, D., Hao, G., Gu, X., Wang, S., Peng, X., Yang, G., 2011a. The mitochondrial genome of *Baylisascaris procyonis*. PLoS ONE 6, e27066.
Xie, Y., Zhang, Z., Wang, C., Lan, J., Li, Y., Chen, Z., Fu, Y., Nie, H., Yan, N., Gu, X., Wang, S., Peng, X., Yang, G., 2011b. Complete mitochondrial genomes of *Baylisascaris schroederi*, *Baylisascaris ailuri* and

*Baylisascaris transfuga* from giant panda, red panda and polar bear. Gene 482, 59–67.
Yeitz, J.L., Gillin, C.M., Bildfell, R.J., DeBess, E.E., 2009. Prevalence of *Baylisascaris procyonis* in raccoons (*Procyon lotor*) in Portland, Oregon, USA. J. Wildl. Dis. 45, 14–18.
Zhang, J.-S., Daszak, P., Huang, H.-L., Yang, G.-Y., Kilpatrick, A.M., Zhang, S., 2008. Parasite threat to panda conservation. EcoHealth 5, 6–9.
Zhang, L., Yang, X., Wu, H., Gu, X., Hu, Y., Wie, F., 2011. The parasites of giant pandas: individual-based measurement in wild animals. J. Wildl. Dis. 47, 164–171.
Zhang, W., Yie, S., Yue, B., Zhou, J., An, R., Yang, J., Chen, W., Wang, C., Zhang, L., Shen, F., Yang, G., Hou, R., Zhang, Z., 2012. Determination of *Baylisascaris schroederi* infection in wild giant pandas by an accurate and sensitive PCR/CESSCP method. PLoS ONE 7, e41995.