



Invited Review

Beyond the raccoon roundworm: The natural history of non-raccoon *Baylisascaris* species in the New World



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ABSTRACT

A total of 10 species of *Baylisascaris*, a genus of ascaridoid nematodes, occur worldwide and 6 of them occur in the New World. Most of the *Baylisascaris* species have a similar life cycle with carnivorous mammals or marsupials serving as definitive hosts and a smaller prey host serving as paratenic (or intermediate) hosts. However, one species in rodents is unique in that it only has one host. Considerable research has been conducted on *B. procyonis*, the raccoon roundworm, as it is a well-known cause of severe to fatal neurologic disease in humans and many wildlife species. However, other *Baylisascaris* species could cause larva migrans but research on them is limited in comparison. In addition to concerns related to the potential impacts of larva migrans on potential paratenic hosts, there are many questions about the geographic ranges, definitive and paratenic host diversity, and general ecology of these non-raccoon *Baylisascaris* species. Here, we provide a comprehensive review of the current knowledge of New World *Baylisascaris* species, including *B. columnaris* of skunks, *B. transfuga* and *B. venezuelensis* of bears, *B. laevis* of sciurids, *B. devosi* of gulonids, *B. melis* of badgers, and *B. potosis* of kinkajou. Discussed are what is known regarding the morphology, host range, geographic distribution, ecoepidemiology, infection dynamics in definitive and paratenic hosts, treatment, and control of these under-studied species. Also, we discuss the currently used molecular tools used to investigate this group of parasites. Because of morphologic similarities among larval stages of sympatric *Baylisascaris* species, these molecular tools should provide critical insight into these poorly-understood areas, especially paratenic and definitive host diversity and the possible risk these parasites pose to the health to the former group. This, paired with traditional experimental infections, morphological analysis, and field surveys will lead to a greater understanding of this interesting and important nematode genus.

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1. Genus *Baylisascaris*

1.1. History and relationships within Ascarididae

Baylisascaris is a genus within the family Ascarididae, comprising mostly heteroxenous nematodes with carnivorous definitive hosts. *Baylisascaris procyonis*, the raccoon roundworm, is by far the most well-known and extensively studied member of the genus, primarily because of its association with severe neurologic disease in humans and numerous species of animals. As a consequence, many other *Baylisascaris* species are relatively poorly studied compared to *B. procyonis*. Here, we review the life history and current knowledge of non-raccoon *Baylisascaris* spp. in the Americas.

The genus *Baylisascaris* was officially described in 1968 and was named in honor of parasitologist H. A. Baylis of the British Museum of Natural History (Sprent, 1968). This genus united some previous members of *Ascaris* and *Toxascaris* and was mainly differentiated from other ascarid genera based on the presence of pericloacal rough patches and subventral postcloacal papillae (versus the absence of subdorsal postcloacal papillae as in *Toxascaris*) (Sprent, 1968). Former members of *Ascaris* reassigned into *Baylisascaris* include *B. devosi*, *B. columnaris*, *B. procyonis*, and *B. laevis*, while *B. transfuga* and *B. melis* were formerly within the genus *Toxascaris*. While *Baylisascaris* and *Toxocara* share biological similarities and are often discussed together in the context of zoonotic ascarids, they are in different subfamilies and are well-separated within Ascarididae (Nadler and Hudspeth, 2000). Molecular phylogenetic analyses of several genetic targets also support the separation of *Baylisascaris* from other ascarid genera (Zhu et al., 1998; Nadler and Hudspeth, 2000; Franssen et al., 2013; Tokiwa et al., 2014).

1.2. Life cycle characteristics

With the exception of *B. laevis*, all members of *Baylisascaris* utilize a carnivore definitive host. Most of these carnivore-infecting species also utilize a wide range of natural paratenic hosts (although there are some data to suggest these hosts are intermediate hosts, see below). Adult nematodes develop in the small intestinal lumen of the definitive host where they feed on host digesta. Females are remarkably fecund and can release >100,000

eggs/worm/day (primarily based on data from *B. procyonis*), which are shed in the feces (Snyder and Fitzgerald, 1987). Over a variable period of time (10–14 days under ideal conditions), the zygote within the egg develops into an infective-stage larva that may infect either definitive hosts in a direct cycle, or paratenic hosts in an indirect cycle (Fig. 1). However, it is likely that definitive hosts acquire some immunity to infection via the direct route with age; experimental infections show that egg inoculation can generally only establish infection in young definitive hosts (Kazacos, 1983; Berry, 1985). Like other ascarids, *Baylisascaris* spp. eggs are covered in an adhesive proteinaceous coat that confers a high degree of resilience to desiccation, freezing, heat (up to 62 C), and disinfectants, and may remain viable in the environment for years (Shafir et al., 2007; Kazacos, 2016).

When ingested by paratenic hosts, larvae hatch from eggs in the small intestine, penetrate the intestinal wall, and undergo tissue migration after entering circulation. The pattern of migration and the resulting larva migrans syndromes vary among *Baylisascaris* spp. and host species. In *B. procyonis*, three larva migrans syndromes are well-described: visceral larva migrans (VLM), ocular larva migrans (OLM), and neural larva migrans (NLM), the latter of which can cause severe neurologic disease with permanent sequelae and death (Kazacos, 2016). Migrating larvae often become encapsulated in paratenic host tissues and are infective to definitive hosts upon predation. Once ingested by the definitive host, it is presumed L3 larvae mature in the mucosa of the small intestine, returning to the lumen at the L4 stage and molting into adults, as has been shown with *Toxascaris*, although it is not clear whether further somatic and/or tracheal migration takes place within all definitive hosts (Sprent, 1954).

It is possible that the route of infection determines whether further migration occurs within the definitive host. Assuming that the stage within the egg is L2, after egg inoculation, migration out of the gastrointestinal tract may be required for advancement to L3. However, if infection occurs via the ingestion of L3 larvae in host tissues, somatic migration may not be necessary; maturation may occur solely in the intestinal wall and lumen without migration, as has been shown with *Toxascaris leonina* (Sprent, 1954). Phylogenetically, *Baylisascaris* forms a sister clade with other Ascarididae (e.g., *Toxascaris*, *Ascaris*, *Parascaris*) although within this group,

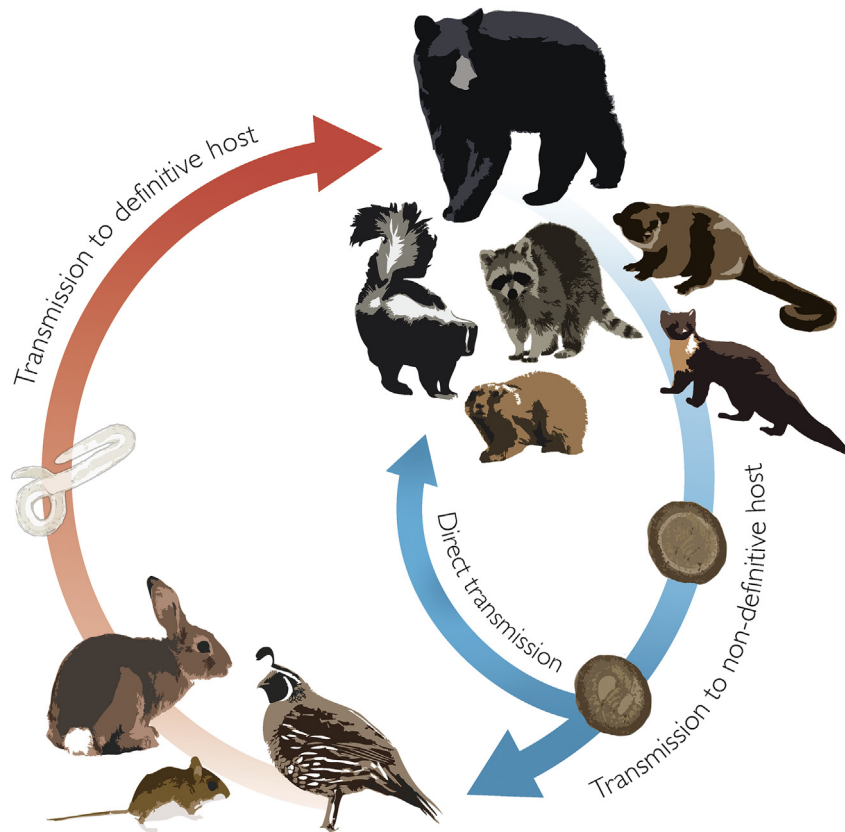


Fig. 1. Generalized life cycle scheme for *Baylisascaris* spp.

Toxascaris is the only member not known to undergo larva migration during development (Li et al., 2012). Experimental data on differential infection dynamics based on exposure route for *Baylisascaris* are lacking (Sprent, 1954; Nadler and Hudspeth, 2000). However, a skunk orally inoculated with *B. columnaris* eggs had an L3 recovered from skeletal muscle whereas this was not observed in other skunks in the same trial that had been inoculated with infected mouse carcasses (Berry, 1985). Migrating *Baylisascaris* sp. larvae have been recovered from skeletal muscle of naturally-infected wild definitive hosts but route of infection is unknown (Hoberg et al., 1990).

Debate exists as to whether hosts in which larvae migrate should be referred to as paratenic or intermediate hosts, which would be dictated by the presence of either a L2 or L3 larvae within the egg. In developmental observations of *Baylisascaris tasma-niensis*, changes in the oral region and a duplication of the cuticular sheath were observed at six days post infection in laboratory mice, which is evidence that a second molt occurs during larva migrans (i.e. L2 to L3) (Sprent et al., 1973). Additionally, only one molt was described within the egg after differentiation of the embryo of *B. tasmaniensis*, so it is likely that larvae within fully developed eggs of this species represent the L2 stage and not L3 (Sprent et al., 1973). Similar observations were also reported in *Baylisascaris laevis* (Babero, 1960a,b). However, when hatched from eggs for *in vitro* culture, *B. procyonis* larvae were partially encased in a thin cuticle and did not molt further; it is not clear whether this cuticle represents a product of the first molt or a second molt within the egg (Boyce et al., 1988). Descriptions of *Toxocara canis* larval development suggest this thin cuticle is an artifact of the first stage cuticle, and could be misinterpreted as a second *in ovo* molt (Schacher,

1957). It is also possible that certain *Baylisascaris* species molt twice within the egg while others molt only once.

Whether or not the second molt occurs within the host or in the egg of *Baylisascaris* spp. and related ascaridoids, migrating *Baylisascaris* spp. larvae grow extensively while migrating through host tissues (depending on species, from ~200 μm to ~1800 μm over the course of weeks). It is possible this growth confers some advantage to larvae as infection efficiency in mature definitive hosts is remarkably higher when inoculated with larvae (i.e. infected carcasses) versus eggs and prepatent periods are shorter (Kazacos, 1983; Miyashita, 1993; Berry, 1985). This suggests that some larval development during somatic migration in non-definitive hosts.

1.3. Diagnostic features

Grossly, *Baylisascaris* spp. adults appear very typical of large ascarids (long cylindrical body, off-white to brown in color, three prominent lips, tail tapered to a point), with the females larger than males. Sprent (1968) described the main diagnostic features of the genus *Baylisascaris* as being:

- Males have a roughened area anterior and posterior of the cloaca, unlike *Ascaris* or *Toxascaris*; post-cloacal papillae arranged differently from *Toxascaris*
- Includes all characteristics within Ascarididae.
- Cervical alae present, either salient or reduced.
- Dorsal and sub-ventral labial papillae present in doublets.
- Excretory cell U-shaped, nucleus within or behind commissural region.

Table 1
Overview of *Baylisascaris* species endemic to the New World, including *B. procyonis* for comparison.

	<i>B. procyonis</i>	<i>B. columnaris</i>	<i>B. devosi</i>	<i>B. laevis</i>	<i>B. melis</i>	<i>B. potosis</i>	<i>B. transfuga</i>	<i>B. venezuelensis</i>
Authority	Stefanski and Zarnowski, 1951	Leidy, 1851	Sprent, 1952a,b	Leidy, 1851	Gedoelst, 1920	Tokiwa et al., 2014	Rudolphi, 1819	Pérez, García & Gauta, 2015
Historical synonyms	<i>Ascaris procyonis</i> , <i>Ascaris columnaris</i> "raccoon ascarid"	<i>Ascaris alienata</i> , <i>Ascaris columnaris</i> "skunk ascarid"	<i>Ascaris gulonis</i> , <i>Ascaris mustelarum</i> , <i>Ascaris devosi</i>	<i>Ascaris laevis</i> , <i>Ascaris tarbagan</i>	<i>Belascaris melis</i> , <i>Toxascaris melis</i> , <i>Ascaris columnaris</i> "badger ascarid"	N/A	<i>Ascaris transfuga</i> , <i>Toxascaris transfuga</i> , <i>Baylisascaris multipapillata</i>	N/A
Primary DH (genus or species)	Raccoons (<i>Procyon lotor</i>); Domestic dogs (<i>Canis familiaris</i>)	Skunks (<i>Mephitis mephitis</i> , <i>Spilogale</i>)	Fishers, Martens, Wolverines (<i>Martes</i> , <i>Gulo</i>)	Marmots, ground squirrels (<i>Marmota</i> , <i>Spermophilus</i>)	Badgers (<i>Meles meles</i> , <i>Taxidea</i>)	Kinkajou (<i>Potos flavus</i>)	Bears (<i>Ursus</i> , <i>Melursus</i>)	Spectacled bear (<i>Tremarctos ornatus</i>)
Adult length ^a (♂; ♀)	46–119; 55–337	43–110; 72–266	57–123; 105–285	37–108; 32–212	120–127; 22–260	117–123; 214–223	63–120; 102–240	102; 250 ^d
Adult midbody width (♂; ♀)	1.0–2.0; 1.5–2.5	1.3–2.5	1.3–2.5	1.0–2.3; 1.7–3.2	up to 3; up to 5	1.6–1.75; 2.51–2.9	1.2–1.9; 1.6–4.5	3.0; 4.1 ^d
Esophageal length (♂; ♀)	2.19–6.46; 2.41–7.53	2.69–5.21; 3.76–7.81	4.15–4.50; 4.00–4.83	2.73–5.64; 1.84–6.59	5.9; 7.25 ^d	4.10–4.56	3.7–4.7; 4.0–5.1	ND
Spicules	0.49–0.71	0.33–0.76	0.39–0.54	0.24–0.81	0.80–0.90	0.61–0.77	0.80–0.93	0.9
Vulvar position ^b	25%	25%	33%	34%	37%	28.2%	37%	40% ^d
Number of pre-anal papillae	43–67	36–53	30–40	41–61	up to 63	44–52	46–70	44 ^d
Cervical alae ^c	Inconspicuous	Inconspicuous	Inconspicuous	Inconspicuous	Prominent	Inconspicuous	Prominent	Prominent
Average egg size	80 × 60 μm	73 × 63 μm	77 × 61 μm	75 × 62 μm	87 × 75 μm	83 × 73 μm	90 × 75 μm	ND
Sources	Sprent 1968, Berry 1985, Kazacos 2016	Sprent 1968, Berry 1985	Sprent 1952a, Sprent 1953b, Sprent 1968	Babero 1960a, Sprent 1968, Berry 1985	Gedoelst 1920, Hartwich 1958, Sprent 1968	Tokiwa et al., 2014	Rudolphi 1819, Sprent, 1968, Testini et al., 2011, Moudgil et al., 2014	Pérez Mata et al., 2016

ND = not determined.

^a Measurements in mm unless otherwise specified.

^b Percentage of body length from anterior end.

^c Inconspicuous = only visible in transverse section.

^d Measurement represents examination of single specimen.

- Relatively short, stout spicules less than 1.0 mm in most members
- Male tail papillae are segregated into pre- and post-cloacal groups with varying numbers depending on species. The post-cloacal group comprises a pair in doublets, a single pair of closely associated singlet papillae, and one phasmid.
- Eggs ovoid to round and finely pitted.

Among the majority of *Baylisascaris* spp., considerable overlap occurs in the morphology of eggs, larva, and many adult characteristics (Table 1). However, there are some morphologic characteristics that can be used to distinguish the various species. For example, *B. transfuga* and *B. melis* are generally thicker and more stout in overall appearance than other *Baylisascaris* spp., have larger spicules, and have cervical alae that are grossly visible (Table 1) (Sprent, 1968). Distinguishing among *B. procyonis*, *B. columnaris*, *B. devosi*, *B. potosis*, and *B. laevis* on morphology alone is difficult or impossible, although *B. laevis* is generally smaller than the other species and although there is overlap, there are some differences in spicule length, number of pre-anal papillae or vulval position (Table 1). Most species are identified based on definitive host alone. However, because some *Baylisascaris* spp. can cross-infect various definitive host species, species identification should not be based on definitive host species alone (Berry, 1985; Sprent, 1952a; Kazacos, 2016). Molecular tools for species identification are readily available and should be used when possible to confirm identifications if needed (see Section 3).

2. New World species of *Baylisascaris*

2.1. *Baylisascaris columnaris*

Originally identified as *Ascaris alienata* by Leidy (1851), the species was renamed *Ascaris columnaris* in 1856, and then re-assigned to *Baylisascaris* by Sprent in 1968 (Leidy, 1851; Sprent, 1968). Morphologically, *B. columnaris* is highly similar to *B. procyonis*, with only subtle distinguishing features, including the structure of cervical support in the cuticle (a wide arch in *B. procyonis* compared with a narrow A-shape in *B. columnaris*), shape of denticles (equilateral triangles versus elongated triangles), male tail terminal shape (spike versus knob), and the number of preanal papillae (average of 40 versus 43, although there is considerable overlap in range) (Franssen et al., 2013; Sprent, 1968). However, it is likely that enough natural variability occurs between the two species such that identification based only on morphological characteristics is inadequate and molecular identification is ideal (Berry, 1985).

Skunks are the definitive host for *B. columnaris*. Infection has primarily been detected in the striped skunk (*Mephitis mephitis*), the most broadly-distributed and studied skunk species in North America, but infections have also been detected in Eastern spotted skunks (*Spilogale putorius*) (Table 2). Sympatric Western spotted skunks (*Spilogale gracilis*) and hog-nosed skunks (*Conepatus* sp.) and may be potential hosts as well but testing has been limited. Ascarids identified as *B. columnaris* have been reported in American badgers (*Taxidea taxus*); however, it is likely that these are actually *B. melis* or *B. devosi* (Table 2). Further surveys that utilize molecular

Table 2
Host and locality records for *Baylisascaris columnaris* in North America.

Host	Location	No. infected/no. examined (%)	Source
Striped skunk (<i>Mephitis mephitis</i>)	New York, USA (central)	NG/74 (>90)	Stegeman, 1939
	Massachusetts, USA (western)	15/19 (84)	Rankin, 1946
	Texas, USA (central)	0/NG (0)	Tiner, 1946
	Oregon, USA (northwestern)	1/1	Stegner and Neiland, 1955
	California, USA (central)	0/45 (0)	Mead, 1963
	North Dakota, USA	8/42 (19)	Dyer, 1970
	Ontario, Canada (southern)	(62)	Berry, 1985
	Illinois, USA (northeastern)	18/73 (25)	Gehrt, 2005
	Texas, USA (west-central)	0/23 (0)	Neiswenter et al., 2006
	Saskatchewan, Canada	65/173 (38)	Wirsing et al., 2007
Eastern spotted skunk (<i>Spilogale putorius</i>)	Minnesota, USA	4/23 (17)	Erickson, 1946
	California, USA (central)	0/14 (0)	Mead, 1963
	Arkansas, USA (western)	1/29 (3)	Lesmeister et al., 2008
Western spotted skunk (<i>Spilogale gracilis</i>)	Texas, USA (central)	0/NG (0)	Tiner, 1946
	Texas, USA (west-central)	0/9 (0)	Neiswenter et al., 2006
American hog-nosed skunk (<i>Conepatus leuconotus</i>)	Texas, USA (central)	0/NG (0)	Tiner, 1946
	Texas, USA (west-central)	0/28 (0)	Neiswenter et al., 2006

NG = not given.

parasite species identification tools will be useful in elucidating the full definitive host range of *B. columnaris*.

2.1.1. Distribution and ecology

Contemporary surveys on *B. columnaris* are generally lacking; more surveillance is needed to accurately characterize the distribution and eco-epidemiology of this parasite because most recent reports are from captive pet skunks (d'Ovidio et al., 2016). From published reports, *B. columnaris* appears to be well-established in the northeast, upper Midwest, and prairie regions of the United States and Canada, apparently uncommon in arid regions of the west and southwest (Table 2). In southern Ontario, Canada, prevalence in *M. mephitis* was significantly lower in spring months compared to late summer or early fall (Berry, 1985), similar to the general seasonal patterns observed for *B. procyonis* in northern climates (Kidder et al., 1989). Similar seasonal variation in prevalence and intensity have been observed for other gastrointestinal helminths of skunks, such as *Physaloptera maxillaris* (Cawthorn and Anderson, 1976). It is likely that the resource-limiting nature of skunk torpor/overwinter fasting causes the loss or developmental arrest of helminths, potentially including *B. columnaris*, but more research is needed to investigate this phenomenon (Dragoo, 2009).

2.1.2. Natural infections in definitive hosts

Similarly to *B. procyonis* in raccoons, *B. columnaris* infection is generally not associated with morbidity or mortality in wild skunk definitive hosts. However, peritonitis or intestinal perforation associated with high worm burdens in captive skunks have been documented (Goodey and Cameron, 1923; Nettles et al., 1978). Goodey and Cameron (1923) noted that skunks from a United Kingdom fur farm exhibited poor body condition, failure to thrive, and inferior coat quality possibly associated with high-intensity *B. columnaris* infection, and possibly resulting in economic losses. Few recent studies have investigated the occurrence of *B. columnaris* among farmed skunks or its economic impacts, although infection control should be straightforward with appropriate enclosure cleaning and regular administration of anthelmintics, most of which are highly efficacious against intestinal stages of the related parasite *B. procyonis* (Bauer and Gey, 1995). Given these data, it is possible that wild skunks with high worm burdens may develop disease.

In Europe, 15 of 60 (25%) pet striped skunks primarily from Germany and Italy were positive and additional infections have been reported from the Netherlands and Poland; all were

genetically-confirmed as *B. columnaris* (Franssen et al., 2013; d'Ovidio et al., 2016; Jańczak et al., 2016). Worm burdens were not determined, but in one study, eggs per gram of feces (EPG) ranged from 150 to 14,500 (mean of 4713 EPG) (d'Ovidio et al., 2016). This is relatively low compared to natural *B. procyonis* infections, which may average 26,000 EPG (Kazacos, 2016). Importantly, many of these infected pet skunks were housed near or with other pet species (e.g. dogs, guinea pigs, parrots), and none of the skunks had ever received anthelmintic treatment (d'Ovidio et al., 2016). Although there are no published reports, *B. columnaris* infections have been diagnosed in pet skunks from the United States (Yabsley, unpublished data). Given the popularity of skunks as pets and the potential for larva migrans in various hosts, education of pet owners is needed to reduce the risk of transmission.

2.1.3. Experimental infections in definitive hosts

Experimental infections of *B. columnaris* in skunks have provided data on the fate of larvae within the definitive host and the development of patency. Berry (1985) experimentally infected striped skunks by feeding them mouse carcasses containing unknown numbers of L3 larvae. Two juvenile female skunks became patent ~48 days post-inoculation (DPI) and one mature male skunk became patent at 93 DPI. Another route of exposure investigated was inoculation with larvated eggs; inoculation of an unspecified number of embryonated eggs resulted in intestinal infections in three juvenile skunks. The youngest individual (38 days old) was sacrificed at 10 DPI and one L3 larva was recovered from skeletal muscle, suggesting that at least some larvae undergo early somatic migration in the definitive host following egg inoculation. This individual also had a small number of L3 and L4 larvae within the lumen of intestine. Numerous L3 and L4 larvae were observed in the lumen of the small intestine in another juvenile "young of the year" skunk sacrificed at 19 DPI; additional larvae were recovered via digestion of the walls of the anterior and posterior small intestine. The remaining juvenile animal was sacrificed at 139 DPI, and although immature adults were found in the intestine at necropsy, eggs were never detected in the feces. The limited number of experimental infection trials in skunks and the low sample sizes makes determining the average onset of patency difficult, and host age and route of infection may be important factors not fully investigated.

To investigate susceptibility of raccoons to *B. columnaris*, two juvenile raccoons were inoculated with unreported numbers of either L3 larvae (in mouse tissue) or embryonated eggs (Berry,

1985). The raccoon inoculated with larvae became infected as L4 larvae were present in the small intestine of the raccoon upon necropsy; however, infection was not allowed to proceed so it is unknown if the raccoon would have become patent. Thus it is unknown if raccoons can serve as alternative definitive host for *B. columnaris*. The single raccoon inoculated with embryonated *B. columnaris* eggs did not develop an intestinal infection (Berry, 1985).

2.1.4. Natural infections in non-definitive hosts

There are no reports of naturally-acquired *B. columnaris* larva migrans in wild or captive paratenic hosts. There have been suspected cases in captive animals that were linked to co-housing with infected skunks. For example, an outbreak involving a white-headed marmoset (*Callithrix geoffroyi*) and two species of tamarins (*Saguinus nigricollis*, *Saguinus midas*) in a zoological park in Texas was likely due to a skunk (of unknown infection status) housed in the enclosure. These primates developed signs of NLM, were treated unsuccessfully with fenbendazole, and were subsequently euthanized (Huntress and Spraker, 1985). Infection with *B. columnaris* in a captive emu (*Dromaius novaehollandiae*) in Indiana with fatal NLM was suspected based on the history of a skunk (also of unknown infection status) being previously held in the enclosure (Kazacos et al., 1982). However, species identification was not confirmed in the emu case. Raccoons are also reportedly common in the area where the emu was housed and *B. procyonis* is highly prevalent in Indiana (Kazacos et al., 1982).

The paratenic host range of *B. columnaris* is likely broad given its biological and phylogenetic similarity to *B. procyonis* and experimental host range. However, molecular techniques will be required in future case studies or surveys to investigate possible natural paratenic hosts. Additionally, *B. columnaris* could be a zoonotic parasite, given its similarities to *B. procyonis* and case reports in primates. It is possible that some presumed *B. procyonis* natural infections are actually *B. columnaris*, due to the extreme difficulty of species identification through adult/larval morphology, egg morphology (from environmental samples), or current serologic techniques which are cross-reactive among *Baylisascaris* spp. (Dangoudubiyam et al., 2010; Berry, 1985). No human cases have been reported, and even if zoonotic, it is unlikely to represent as significant a public health threat as *B. procyonis*, as skunks are generally in lower densities in urban areas compared to raccoons (Gehrt, 2004). Therefore, potential human contact with skunks feces is limited. Nonetheless, individuals with frequent contact with skunks and skunk feces (pet owners, wildlife rehabilitators, fur farmers, trappers, etc.) should take precautions against potential exposure to *B. columnaris*. Recently, antibodies to *Baylisascaris* (presumed to be mostly due to *B. procyonis* exposure, but could be due to other species) were detected in wildlife rehabilitators (Sapp et al., 2016a).

2.1.5. Experimental infections of non-definitive hosts

B. columnaris produces disease due to larva migrans in a variety of experimentally-infected paratenic host species, particularly rodents and lagomorphs. Compared to *B. procyonis*, *B. columnaris* generally causes less mortality in experimentally-infected rodents due to slower and more limited NLM. Independent experiments by Sprent (1952b) and Tiner (1953a) demonstrated that neurological signs were generally noted between 17 and 25 days in laboratory mice inoculated with an unspecified number of eggs, compared to 7–10 days for *B. procyonis*. However, dose is likely important in the rate of disease development as has been shown with *B. procyonis* (Tiner, 1953a; Sheppard and Kazacos, 1997; Sapp et al., 2016b). Domestic rabbits (*Oryctolagus cuniculus*) inoculated with 100,000 eggs, considered a very high dose, rapidly developed severe

neurologic disease involving seizures, epistaxis, ataxia, and dyspnea, with onset between 4 and 10 DPI (Church et al., 1975).

This generally delayed onset of neurological disease compared to *B. procyonis* is most likely due to the relatively slower growth of L3s within paratenic hosts. In a 20 day trial of experimental infection of laboratory mice, *B. columnaris* larvae grew to approximately 1000 μm in length by the end of the trial, compared to *B. procyonis* that achieved this size by day 10 and reached an average maximal length of 1200 μm by day 20 (Tiner, 1953b). Experimental trials in laboratory mice suggest that neurological disease does not become readily apparent until larvae within the brain have reached a length of ~1000 μm (Tiner, 1953b). In laboratory mice, *B. procyonis* reaches an average length of ~1000 μm in ~8–10 days post infection, after which survival of infected hosts fell dramatically, whereas *B. columnaris* took ~16 days to reach 1000 μm in length, after which time some mortality occurred (Tiner, 1953b). In some cases, mice inoculated with *B. columnaris* eggs were able to recover from clinical disease or survived despite the presence of larvae in the brain (Tiner, 1953b). Similarly, *B. procyonis* larvae have been detected in wild-caught, presumably normally-acting, *Peromyscus leucopus* further suggesting that some rodents can survive infections of the brain (Page et al., 2001; Sapp and Yabsley unpublished data). The observed differences larval growth between *B. columnaris* and *B. procyonis* may also reflect differences in paratenic host species adaptation. In experimentally-infected meadow voles (*Microtus pennsylvanicus*), *B. columnaris* larvae achieved a greater average length (1570 μm) than *B. procyonis* (1060 μm) by 10 DPI (Berry, 1985). The study was terminated at 10 DPI for larval morphological analysis, so other infection dynamics were not assessed. The impact of larval size on disease severity could also explain why cerebral infection with other smaller larval ascarids such as *Toxocara canis* (that achieves a maximal length of about 500 μm in rodent brains) produces overt disease much less frequently than *B. columnaris* or *B. procyonis*, although host factors, including brain size, likely play a role in the rate and severity of neurologic disease (Tiner, 1953a; Sprent, 1955).

A large proportion of larvae in inoculated paratenic host species become encapsulated within the intestinal wall and mesentery within 1–4 DPI (Berry, 1985). Encapsulated larvae were also abundant in the lungs, heart, kidneys, and liver shortly after inoculation in laboratory mice and in a groundhog (*Marmota monax*), presumably due to liver-lung migration (Sprent, 1952b; Berry, 1985). Numerous larvae migrated within skeletal muscle and became encapsulated after 10 DPI in inoculated *Microtus pennsylvanicus* (Berry, 1985). In inoculated experimentally-infected rabbits, extensive larval granulomas with eosinophilic infiltration were observed in the lungs, liver, brain, eyes, kidneys, heart, and gastrointestinal tissues (Church et al., 1975).

Similar to other *Baylisascaris* species, *B. columnaris* larvae within tissues are resistant to freezing. Encapsulated larvae in tissues remained viable and recovered substantial motility after a periods of freezing ranging from 8 to 18 weeks at $-20\text{ }^{\circ}\text{C}$, which was superior to freeze-susceptible *B. transfuga* and *T. canis* larvae in the same experiment (Sprent, 1953a). However, experimental trials to confirm infectiousness of previously-frozen *Baylisascaris* larvae have not been conducted.

2.2. *Baylisascaris* spp. of bears

2.2.1. *Baylisascaris transfuga*

Baylisascaris transfuga was originally described by Rudolphi (1819) as *Ascaris transfuga* and re-described as *Toxascaris* species in 1922 (Baylis and Daubney, 1922). Ultimately Sprent (1968) formally described the *Baylisascaris* genus and designated *Baylisascaris transfuga* as the type species for this genus. Morphological

characteristics and/or molecular techniques can be used to distinguish this species from other *Baylisascaris* spp. (Table 3). *Baylisascaris transfuga* adults can be morphologically distinguished from other species by their spicule length (estimated between 0.80 and 0.92 mm), having between 46 and 70 precloacal papillae, rounded posterior margin of the pericloacal area, salient alae, denticles in equilateral triangles, and a saddle shape of the median lobe of the lip (Testini et al., 2011; Sprent, 1968; Baylis and Daubney, 1922). Eggs of *B. transfuga* are morphologically similar to other *Baylisascaris* spp. eggs and are largely considered indistinguishable (Sprent, 1968; Kazacos and Turek, 1983; Testini et al., 2011) (Table 1).

Of interest is that morphometrics of parasites reported as *B. transfuga* from numerous hosts across several continents show variation (Table 3). Measurements have been made on specimens from black bears (*Ursus americanus*) in Canada, multiple subspecies of brown bear (*Ursus arctos*) from across sites Eurasian and North American sites, multiple captive polar bears (*Ursus maritimus*) in various sites, sloth bears in India (*Melursus ursinus*), and a captive sun bear (*Helarctos malayanus*) (Canavan, 1929; Baylis and Daubney, 1922; Sprent, 1968; Testini et al., 2011; Moudgil et al., 2014). Sprent (1968) reported a maximal length of 120 mm for males and 240 mm for females; other studies have found smaller size ranges for both males and females (Table 3). Egg dimensions are also variable and have been reported as low as ~57 µm wide up to ~94 µm long, although fertilization or embryonation status may influence this morphology (Table 3).

These morphologic differences in a limited number of parasites examined across a wide geographic and host range suggest that these “*B. transfuga*” may represent several distinct species. Currently there are two distinct *Baylisascaris* species apart from *B. transfuga* reported within Ursidae, *B. schroederi* of giant pandas (*Ailuropoda melanoleuca*), and the recently-described *B. venezuelensis* from spectacled bears (*Tremarctos ornatus*). Further support is provided by preliminary molecular data on *B. transfuga* samples from Alberta, Canada and West Virginia that suggest these parasites are genetically distinct across locations (L. Camp, pers. comm.). Careful morphologic analysis combined with molecular characterization of *B. transfuga* samples from a diverse geographic and host range is needed to address parasite diversity in the Ursidae.

2.2.1.1. Epidemiology. Worldwide, *B. transfuga* has been reported in all extant species of bear in the family Ursidae excluding the spectacled bear although unidentified ascarid eggs have been detected in spectacled bear feces (Figueroa, 2015). *Baylisascaris transfuga* infections have been reported from American black bears (*Ursus americanus*), sloth bears, polar bears, brown bears (*Ursus arctos*), Malayan sun bears (*Helarctos malayanus*), Asiatic black bears (*Ursus thibetanus*), and giant pandas (*Ailuropoda melanoleuca*) although these reports are most likely *B. schroederi* (Sprent, 1968; Rudolphi, 1819; Canavan, 1929; Baylis and Daubney, 1922). No non-bear definitive hosts are known.

The prevalence of *B. transfuga* in bears varies widely among studies (Table 4). In North America, the majority of studies have been conducted on black bears but natural infections have been reported in brown bears and in captive polar bears. Prevalence of *B. transfuga* in black bears appears to be highest in Alberta, Canada and the Great lakes regions of the USA. In brown bears, prevalence of infection with *B. transfuga* was higher in the Wyoming and Montana, USA compared to Alaska and Canada.

A few studies have attempted to investigate seasonal trends in prevalence with some conflicting results. One, based on fecal floatation, detected a higher prevalence in spring compared to the fall in black bears in Quebec, Canada (Frechette and Rau, 1978). Another, based on collection of nematodes at necropsy in black bears and grizzly bears in western Canada observed the opposite seasonal trend with peaks in the fall but this study was (Frechette and Rau, 1978; Catalano et al., 2015). However, the seasonal association with prevalence in black bears was weakly significant ($p = 0.04$) and sample sizes were relatively small ($n = 40$); sample size for grizzly bears was too small for statistical analysis. Finally, Rausch (1954) and Rogers (1975) found that bears were shedding eggs in the spring soon after torpor and found evidence of egg shedding just prior to denning, so it remains unclear whether or not infections are cleared during winter torpor. Additional studies are needed with greater sample sizes and age class representation to accurately assess the seasonal ecology of *B. transfuga* in bears.

In the bear host, *B. transfuga* infections do not typically cause clinical disease, but heavy infections have been reported causing clinical disease or death. Reports of disease in the natural bear host include peritonitis in a brown bear in Europe and suggestions of enteric impactions (Mozgovi, 1953; Szczepaniak et al., 2012). Subclinical effects including reduced host condition have also been reported in infected bears (Fu et al., 2011).

2.2.1.2. Non-definitive hosts. The risk of *B. transfuga* to cause clinical disease as a result of larva migrans in aberrant hosts is considered low compared to other *Baylisascaris* species. This is partly due to their slower growth rate and smaller size as well as the decreased penetration of the intestinal wall by *B. transfuga* larvae resulting in fewer larvae being detected in visceral organs compared to other species (Sprent, 1952b, 1953a; Sato et al., 2004; Schaul, 2006).

Despite a reduced migratory capacity relative to other *Baylisascaris* spp., experimental infections in laboratory mice, Mongolian jirds (*Meriones unguiculatus*), guinea pigs, rabbits, and chickens indicate that *B. transfuga* can occasionally cause VLM, OLM, and/or NLM; however, in most hosts, clinical disease was mild or not apparent (Sprent, 1952b, 1955; Papini et al., 1993; Papini et al., 1994, 1996a; Papini and Casarosa, 1994; Sato et al., 2004; Matoff and Komandarev, 1965). There is substantial variation among hosts in disease severity. Laboratory mice developed only mild clinical disease with granulomas in the brain. Mongolian jirds developed severe clinical signs with malacia and lack of host immune reaction (Sato et al., 2004). Rabbits displayed a loss of appetite, dyspnea, and depression but no neurological signs (Papini et al., 1996a). No

Table 3
Morphometrics of *Baylisascaris transfuga* adults and eggs from various hosts and geographic regions.

Host	Location	Male length (mm)	Female length (mm)	Egg width (µm)	Egg length (µm)	Source
Multiple captive and wild species	Multiple	Up to 120	Up to 240	Up to 75.0	Up to 90	Sprent 1968
Sloth bear (<i>Melursus ursinus</i>)	India	64–94	138–183	47.0–75.2	65.8–94.0	Moudgil et al., 2014
Polar bear (<i>Ursus maritimus</i>)	Italy	63–116	102–203	NG	NG	Testini et al., 2011
		NG	NG	57.6–64.0	60.8–73.6	Papini and Casarosa 1994
Multiple captive species	Pennsylvania (USA)	81–142	108–166	NG	NG	Canavan 1929
Multiple captive species	Louisiana (USA)	NG	NG	66.3–74.7	78.3–88.0	Clark et al., 1969

NG = not given.

Table 4
Reports of *Baylisascaris transfuga* in free-ranging and captive bears in North America.

Host	Location	Captive/Wild	No. infected/no. examined (%)	Method of detection	Source	
Brown bear (<i>Ursus arctos</i>)	Northwest Territories, Canada	Wild	3/56 (5)	Fecal flotation	Gau et al., 1999	
	Wyoming and Montana, USA	Wild	53/70 (76)	Necropsy	Worley et al., 1976	
	Canada	Wild	16/21 (76)	Necropsy	Choquette et al., 1969	
	Alaska, USA	Wild	0/28 (0)	Fecal flotation	Schaul, 2006	
	British Columbia and Alberta, Canada	Wild	7/13(54)	Necropsy	Catalano et al., 2015	
Black bear (<i>Ursus americanus</i>)	Florida, USA	Wild	5/22 (23)	Necropsy	Foster et al., 2004	
	New York, USA	Wild	17/55 (31)	Necropsy	King et al., 1960	
	Minnesota and Michigan, USA	Wild	5/9 (56)	Necropsy	Rogers, 1975	
	Wisconsin, USA	Wild	59/92 (64)	Fecal Flotation	Manville, 1978.	
				25/29 (86)	Necropsy	
	Wyoming and Montana, USA	Wild	24/30 (80)	Necropsy	Worley et al., 1976	
	Minnesota, USA	Wild	1/1 (100)(august)	Necropsy	Barnes and Rogers, 1980	
	Southeastern USA	Wild	28/53 (53)	Necropsy	Crum et al., 1978	
	New Brunswick, Canada	Wild	1/12 (8)	Necropsy	Duffy et al., 1994	
	Alberta, Canada	Wild	35/56 (62)	Necropsy	Dies, 1979	
	Quebec, Canada	Wild	17/80 (21)	Fecal flotation	Frechette and Rau 1978	
	Quebec, Canada	Wild	38/168(23)	Fecal flotation	Frechette and Rau 1978	
				2/21(10)	Necropsy	
	Ontario, Canada	Wild	20/83(24)		Addison et al., 1978	
	British Columbia and Alberta, Canada	Wild	24/40(60)		Catalano et al., 2015	
Polar bear (<i>U. maritimus</i>)	Massachusetts, USA	Captive	1/1(100)	Fecal flotation	Johnson et al., 2013	
	California, USA	Captive	2/3 (66)	Necropsy	McOrist et al., 2002	
	USA, various	Captive	125/260 (48)	Fecal flotation	Abdel-Rasoul and Fowler, 1979	
Various	USA, various	Captive	125/260 (48)	Fecal flotation	Schaul, 2006	

clinical signs were observed in experimentally-inoculated chickens (Papini et al., 1993). In guinea pigs, Matoff and Komandarev (1965) showed that *B. transfuga* larvae migrate into the intestinal wall and either encyst in the wall, penetrate the intestinal wall and enter the abdominal cavity, or travel to the lungs, heart, and/or skeletal muscles through the lymphatics and systemic circulation. However, larvae were not noted in brains. Sprent (1953a) reported that larvae were still alive in experimentally-infected mice one year after infection.

Despite experimental studies suggesting a wide range of hosts may develop larva migrans, only one presumed report of natural infection of a paratenic host has been reported. Japanese macaques (*Macaca fuscata*) housed near American black bears at a zoo in Japan developed fatal neurological disease; however, identification of the parasite was not definitive in this case and identification of the parasite was based only on histology (Sato et al., 2005). While there are no confirmed reports of larva migrans in humans following *B. transfuga* infection, experimental evidence with other species shows that given a sufficiently high infection, larva migrans in people may be possible.

2.2.1.3. Treatment and control. *Baylisascaris* eggs present in the environment or in captive animal facilities are difficult to eliminate or kill. Similar to other *Baylisascaris* species, eggs of *B. transfuga* become infective after ~2 weeks and can remain infective for at least 15 months under artificial conditions (Papini and Casarosa, 1994). Eggs have reported to persist in the environment for up to five years, and infected bears can pass between 100 and 19,800 eggs per gram of feces so environments can become contaminated with large number of eggs quickly (Abdel-Rasoul and Fowler, 1979; Vercruyse et al., 1976). In captivity, the prevalence of *B. transfuga* is higher in certain species (*U. maritimus*, *Melursus ursinus*), although this could be sampling bias, the substrate (e.g. sand or soil) used in enclosures, or the housing of bears in groups (Schaul, 2006). Strict, routine quarantine and treatment of bears in captive settings can be an effective way to reduce shedding and prevent

subsequent infections.

Treatment of bears infected with *B. transfuga* has only been done in captive situations. Numerous anthelmintics have been used to manage *B. transfuga* infections in captive bears; however, efficacy is variable and dose-dependent (Clark et al., 1969; Moudgil et al., 2014). Dichlorvos (19 mg/lb) rapidly (1–2 days post treatment) reduced fecal egg counts (FEC) to zero in many bear species; however, these animals became reinfected within months after treatment, emphasizing the need to clean the environment (Clark et al., 1969). Orally-administered fenbendazole (10 mg/kg) on three consecutive days was unable to reduce fecal egg counts to zero in a sloth bear, however, this infection was cleared with 15 mg/kg for three days followed by the original treatment (Moudgil et al., 2014). Mebendazole was used successfully to treat five polar bears infected with *B. transfuga* (Vercruyse et al., 1976). Macrolides, benzimidazoles, and tetrahydropyrimidines have all been used in North American zoos to treat bears but efficacy data were not provided (Schaul, 2009).

Due to concerns about larva migrans, it is important to determine if larvae could be killed prior to entering the CNS. Laboratory mice given one dose of 2 mg/kg ivermectin had fewer lesions and resulted in fewer *B. transfuga* larvae recovered in visceral organs compared to those without treatment (Papini et al., 1996a,b,c). In another study, single doses of levamisole or ivermectin were administered subcutaneously and intramuscularly, respectively, to groups of inoculated laboratory mice 3 DPI or 14 DPI (Fu et al., 2011). Upon necropsy, reduced larval burdens were observed in treatment groups compared to controls. Levamisole resulted in an 81% decrease in “migrating” larvae (at 3 DPI) but only a 49% overall decrease in “encapsulated” larvae (at 14 DPI). Although ivermectin had similar activity against larvae at 3 DPI (88% reduction), it had greater activity against larvae at 14 DPI (75% reduction). Levamisole-treated mice at 14 DPI had fewer larvae within the brain (43% versus 24% in ivermectin group) and appeared to ameliorate the severity of neurologic signs in two of five mice displaying clinical disease after 17 DPI (Fu et al., 2011). These data

suggest that larvicidal activity will vary depending on the age of infection, resulting in changes in susceptibility to the drugs during migration, and in encapsulation status. Pharmacokinetics of anthelmintics also influence treatment efficacy; for example, ivermectin does not cross the blood-brain barrier whereas levamisole appears to do so (Fox, 2006; Lin and Tsai, 2006). Given this differential susceptibility and drug efficacy, it seems the best option for treating *B. transfuga* larva migrans in paratenic hosts is to use multiple drug classes in order to maximize larvicidal activity in brain, viscera, and skeletal muscle.

2.2.2. *Baylisascaris venezuelensis*

A new species, *Baylisascaris venezuelensis*, originating from a South American spectacled (Andean) bear (*Tremarctos ornatus*) in western Venezuela was recently described (Pérez Mata et al., 2016). A female spectacled bear in poor body condition was found dead and at necropsy, a large number of *Baylisascaris* were present in the gastrointestinal tract. Other gross pathologic findings included congestion and hemorrhagic foci in the lungs. The authors suggest that the high worm burden was the cause of mortality.

An adult male and female nematode were examined morphologically. Fewer post-cloacal papillae ($n = 44$) were present compared to Nearctic and Palearctic *B. transfuga* worms, which have an average of 66 (Sprent, 1968). Other morphologic features were similar to *B. transfuga*, including overall length, a stout appearance, salient cervical alae, similar length spicules, and a rounded posterior margin of the pre-cloacal area (with *B. venezuelensis* having a “little process” on this margin) (Pérez Mata et al., 2016). Molecular analysis supported the separation of *B. venezuelensis* as a separate species. Combined ITS-1 and ITS-2 sequences were only 91.8% and 90.6% similar to *B. transfuga* and *B. schroederi*, respectively (Pérez Mata et al., 2016). There were also three nucleotide differences in the highly conserved region of 5.8S rDNA that differentiated *B. venezuelensis* from the two other ursid-associated species and other *Baylisascaris* spp. Phylogenetic analysis of both ITS regions included *B. venezuelensis* in a clade containing the other two ursid-associated species along with *B. ailuri* from red panda (Pérez Mata et al., 2016).

A few instances of previously detected ascarid eggs in fecal examinations of captive and free-ranging spectacled bears may represent *B. venezuelensis* infections (Schaul, 2006; Figueroa, 2015). Eggs designated as “roundworm” eggs were present in 9/25 (36%) of spectacled bear fecal samples from zoos across the United States, although it is impossible to determine if this is *B. venezuelensis* or native *B. transfuga* acquired from other bears in the captive environment (Schaul, 2006). Ascarid eggs resembling those of *Baylisascaris* or *Toxocara* were reported in 6/28 (21%) of *T. ornatus* scats from northern Peru, but measurements of the eggs were not provided (Figueroa, 2015). It is currently unknown if *B. venezuelensis* is usually pathogenic for spectacled bears. The type host is believed to have died from the nematode infection, but most previously reported positive spectacled bears were presumably asymptomatic. Although most *Baylisascaris* spp., including *B. transfuga*, rarely cause mortality in their definitive hosts, *B. schroederi* from pandas is a major cause of morbidity and mortality so additional research on the potential risk of *B. venezuelensis* to spectacled bears is needed (Zhang et al., 2008).

The finding of a new, seemingly valid *Baylisascaris* species in a relatively isolated population of ursids further supports the idea that “*B. transfuga*” represents an assemblage of species globally, and highlights the need for further molecular and morphologic work to characterize possibly cryptic species. Field surveys are also necessary to determine the prevalence as well as definitive and paratenic host range of this new tropical species.

2.3. *Baylisascaris laevis*

Baylisascaris laevis uses rodents instead of carnivores as definitive hosts that makes it unique among the other *Baylisascaris* spp. in the New World (Berry, 1985). The parasite was first described in 1856 by Leidy as *Ascaris laevis* from naturally infected groundhogs (*Marmota monax*). Later it was reassigned to the genus *Baylisascaris* (Sprent, 1968).

Among the New World *Baylisascaris* species, *B. laevis* is generally smaller and wider than other species of *Baylisascaris* (Table 1). This species also has the smallest spicules and largest dorsal lip compared to other *Baylisascaris* spp. (Babero, 1960a; Sprent, 1968). Other differences involve the posterior end of the male and female worms with the female *B. laevis* tail abruptly tapered to a sharp point and the male tail narrowed mid-tail and appears swollen at the end (Tiner, 1951; Berry, 1985). Other diagnostic features included indistinct knobby protrusions near the cloacal opening of males (Tiner, 1951). The external genitalia of female *B. laevis* more anterior compared to *B. columaris* and *B. procyonis* (Berry, 1985).

2.3.1. Host range

The most commonly reported definitive host of *B. laevis* is the groundhog, a member of the Scuridae family. Infections in other Scuridae hosts have been reported in the Alaska marmot (*M. broweri*), hoary marmot (*M. caligata*), yellow-bellied marmot (*M. flaviventris*), Olympic marmot (*M. olympus*, California ground squirrel (*Otospermophilus beecheyi*), Barrow ground squirrel (*Spermophilus parryi barrowensis*), Richardson's ground squirrel (*U. richardsonii*), and long-tailed ground squirrel (*Urocitellus undulatus*) (Berry, 1985).

2.3.2. Differences in life cycle compared to other *Baylisascaris* spp.

B. laevis is the only member of the genus that has a strictly monoxenous life cycle with the apparent loss of a paratenic host during evolution. Development from L2 to adult occurs entirely in the sciurid definitive host (Berry, 1985). Unlike other *Baylisascaris* spp., larvae which migrate throughout the body of paratenic hosts, *B. laevis* only migrate within the liver and lungs of their hosts (Babero, 1960b).

When larvated eggs are ingested, L2 hatch and migrate to the liver by 10–12 DPI and develop into L3. These larvae migrate to the lungs where they molt into L4 that are coughed up and swallowed. Once in the small intestine they continue to develop into adults within the wall of the small intestine (Babero, 1960b). Adults enter the intestinal lumen, mate, and produce eggs that are then shed in feces. Reinfection likely occurs when embryonated eggs adhering to fur are ingested during grooming (Berry, 1985). *B. laevis* can produce liver lesions in its sciurid host (Tiner, 1953b).

2.3.3. Ecology and epidemiology

Although the distribution of known *B. laevis* sciurid hosts extends throughout North America, the parasite has only been reported in New York, Pennsylvania, California, and Alaska. Outside the United States it has been reported in southern Ontario and Saskatchewan, Canada (Berry, 1985). Further surveillance is needed to characterize the distribution of *B. laevis* in North America.

A few studies have been conducted on the seasonality of *B. laevis* infectious. During a 2 year study in southern Ontario, Canada, *B. laevis* prevalence peaked in September, with intensity showing similar seasonal variation (Berry, 1985). Prevalence was lowest during winter months and increased during the spring, similar to the annual cycles observed in *B. columaris* and *B. procyonis* (Berry, 1985). This seasonality of *B. laevis* seems to be primarily driven by feeding habits; groundhogs and ground squirrels continually feed throughout the spring and summer, and then in the fall begin to

consume less in preparation for hibernation. In the winter, absence of (Young and Sims, 1979) and physiological changes accompanying hibernation, such as lowered temperature, heart and metabolic rate of hosts, likely prevents *B. laevis* from developing if acquired late in the year (Babero, 1960b). Despite lower rates of shedding in the winter, eggs can persist in the environment because they are resistant to sub-zero temperatures. Most (94%) non-embryonated eggs survived -10 C temperatures after exposure for 10 days, while $>70\%$ of embryonated L3 eggs survived -10 C temperatures for 16 days (Berry, 1985). Also, eggs deposited in the environment are often protected from extreme temperatures because they are covered by leaf litter and snow or in subterranean burrows (Berry, 1985).

2.3.4. Experimental infections

A variety of species have been assessed as experimental hosts for *B. laevis* using experimental infections. Babero (1959) conducted oral infection trials to assess susceptibility and pathology of *B. laevis* infection in eleven species including laboratory mice, laboratory rats (*Rattus norvegicus*), cotton rats (*Sigmodon hispidus*), hamsters, guinea pigs (*Cavia porcellus*), thirteen-lined ground squirrel (*Ictidomys tridecemlineatus*) and Franklin ground squirrels (*Poliocitellus franklinii*), opossums (*Didelphis virginiana*), groundhogs, domestic cats, and raccoons. Many of these experimental hosts were examined for infection via necropsy, although details on the duration of infection and stages recovered are not given so interpretation is difficult.

Some inoculated hosts developed disease, including a groundhog with signs of pneumonia due to larva migration. Two guinea pigs, who died ~ 40 days post infection, exhibited dyspnea, bloody stools, ataxia and emaciation. Granulomatous liver lesions were frequently observed, sometimes containing L2 larval sheaths, as well as foci of pulmonary hemorrhage (Babero, 1959). In separate experimental infections of laboratory mice, larvae apparently did not become completely encapsulated in the liver, but lesions due to migration of the larvae through the liver were present (Tiner, 1953a). Other experimental infections on multiple hosts (groundhogs, ground squirrels, cats, mice, guinea pigs, and hamsters) showed that larvae become encapsulated in the liver. When larvae migrated to the lungs, very few were retained, and even fewer were found in the small intestine after migration was completed (Babero, 1960b).

2.4. *Baylisascaris devosi*

Baylisascaris devosi infects North American mustelids belonging to the clade Guloninae (martens, fishers and wolverines), which are all carnivorous mammals that inhabit forests in the northern United States and Canada (Ruggiero et al., 1994; Li et al., 2014) (Table 5). *B. devosi* was formally described by Sprent (1952a) showing that parasites from fisher (*Martes pennant*) and marten (*Martes americana*) were distinct from *B. columnaris* from striped skunks. Prior to the description of *B. devosi*, a specimen from a Pacific marten (*Martes caurina*) in Idaho was recorded as *B. columnaris* but this parasite was likely *B. devosi* (Sprent, 1952a; Marshall, 1942). *Baylisascaris devosi* can be distinguished from *B. columnaris* by several morphologic characteristics including body length, length of spicules, the position of the vulva, and the width of denticles on the denticular ridges. Specimens recovered from fisher were longer than those parasites recovered from martens (Sprent, 1952a). Egg sizes range from $58\text{--}77 \times 51\text{--}61$ μm (Sprent, 1953a). However, given potential overlap in morphometric features, species identification should be confirmed using molecular analysis.

Eggs of *B. devosi* can become fully larvated in 12 days and remain

infective for at least one year (Sprent, 1953a,b). In experimentally-inoculated laboratory mice, larvae migrated out of the intestine and by three days were found in the heart, lungs, brain and kidney. Mice showed severe symptoms of pulmonary disease between 3 and 4 DPI and lungs were dark red (Sprent, 1952b). By 8–12 DPI, larvae were present in muscular and subcutaneous tissues of the neck, shoulders and thorax. These third stage larvae remained encapsulated in muscle tissue for at least 6 months. Larvae from a mouse infected for 25 days were infective to a domestic ferret (Sprent, 1953b). Larvae appear to be tolerant to freezing as larvae recovered from mice >3 weeks post infection were motile after being frozen at -20 C for up to 8 weeks, but it is unknown if they were infectious to a definitive host (Sprent, 1953b). If larvae remain infectious following freezing, this would facilitate transmission of *B. devosi* from frozen carcasses that may be scavenged by a definitive host. Experimental inoculation of several adult and juvenile domestic ferrets and a single skunk via infected mouse carcasses established patent infections. An attempt to inoculate a single adult marten by the same manner was unsuccessful, but perhaps this wild-caught animal had pre-existing immunity to *B. devosi* (Sprent, 1953b).

Most larvae recovered from laboratory mice hosts experimentally infected with *B. devosi* were concentrated in the cervical and thoracic musculature (Sprent, 1953b). This localization of infection may be adaptive because marten are known to attack the head and neck region of prey and occasionally discard the rest of the carcass (Powell et al., 2003; Sprent, 1953b). If this concentration of *B. devosi* larvae in the anterior region occurs in natural paratenic hosts, larvae may be more likely to be ingested by the definitive mustelid host.

In the future, molecular studies on parasites recovered from free-living hosts could help more accurately identify *B. devosi* from other *Baylisascaris* species, which will provide important data on prevalence and host specificity. Further, experimental trials on *B. devosi* in various suspected or known natural definitive hosts would provide data on host suitability.

2.5. *Baylisascaris melis*

The definitive hosts of *Baylisascaris melis* are North American badgers (*Taxidea taxus*) and European badgers (*Meles meles*). The parasite was first described from European badgers in Belgium (Geddoelst, 1920). Morphological characteristics of *B. melis* are similar to those of *B. transfuga*; both of these species have salient alae, as opposed to the vestigial alae of other *Baylisascaris* spp. (Sprent, 1968). There have been multiple reports of ascarids in wild North American badgers that are assumed to be *B. melis* although most were reported as *A. columnaris* (Table 6). Contemporary studies, including molecular characterization, on this parasite are needed to confirm the identity of parasites from North American badgers and their conspecificity with *B. melis* from European badgers.

Baylisascaris melis can cause larva migrans in experimentally infected rodents. Tiner (1953a) fed 2000–3000 eggs collected from a naturally-infected badger in Wyoming to four rodent species: ground squirrel (*Citellus armatus*), laboratory mice, deer mice (*Peromyscus maniculatus*) and guinea pigs. Only the ground squirrels (5 of 7 infected) developed neurologic disease; however, larvae were found in the brains of the deer mice and encapsulated in the skeletal muscle of all four species (Tiner, 1953a). In one ground squirrel, larvae were recovered from the lungs and were widely distributed in the skeletal muscle, with greatest abundance in the intercostal spaces under the parietal pleura of the diaphragm. In *P. maniculatus*, *B. melis* larvae were encapsulated in the mesentery of the small intestine, on the epicardial surfaces, and in the brain on

Table 5
Host and locality records of *Baylisascaris devosi* in North American mustelid species.

Host species	Location	No. infected/No. examined (%)	Source
American marten (<i>Martes americana</i>)	Manitoba, Canada	1/139 (0.7)	Poole et al., 1983
	Alaska, USA	1/141 (0.7)	Scranton 1986
	Washington, USA	4/78 (5)	Hoberg et al. 1990, ^a
Wolverine (<i>Gulo gulo</i>)	Northwest Territories, Canada	NG	Addison and Boles 1978
	Alaska, USA	17/80 (21)	Rausch 1959
Fisher (<i>Martes pennanti</i>)	New Brunswick, Canada	NG	Dick and Leonard 1979
			(cited pers. comm. with C. Burse)
Pacific marten (<i>Martes caurina</i>) <i>Martes americana</i> , <i>Martes pennanti</i>	Manitoba, Canada	52/162 (32)	Dick and Leonard 1979
	Idaho, USA	6/17 (35)	Marshall 1942, ^b
	Ontario, Canada	NG	Sprent 1952a

NG=Not given.

^a Unidentified larvae authors suggested could be *B. devosi* were found in digestions of hind limb musculature of 10 martens in addition to the 23 adult nematodes identified as *B. devosi* in study.^b Identified as *Ascaris columnaris*.**Table 6**
Locality records of presumed *Baylisascaris melis* from North American badgers (*Taxidea taxus*).

Location	No. infected/No. examined	Source
Minnesota ^a	1/8 ^b	Erickson 1946
Iowa ^a	29/NG	Wittrock and Ulmer 1974
North Dakota ^a	6/17	Leiby et al., 1971
Kansas ^a	10/30	Pence and Dowler 1979
Wisconsin ^a	2/4	Morgan 1943
South Dakota ^a	NG	Jense 1968
Colorado ^a	NG	Leiby 1961
Wyoming	1 roadkill badger, 1 captive badger	Tiner 1953a

NG=Not given.

^a Identified as *Ascaris columnaris* but are assumed to be *B. melis*.^b 2 of the 8 badgers had been in captivity for 2 years.

3 DPI (Tiner, 1953b).

Although *B. melis* can cause central nervous system disease in experimentally-infected rodent species, there have been no confirmed cases of natural *B. melis* infections in wild rodents (Boyce et al., 1988; Kazakos, 2001; Tiner, 1953a,b). However, neurologic cases due to *Baylisascaris* sp. diagnosed in ground squirrels and other rodents in regions where the badger ranges overlap with raccoons could be due to *B. melis* (Kazakos, 2001). In future cases, identification of larvae in these cases using molecular techniques is needed to better understand the role of non-*B. procyonis* species in cases of neurologic disease in wildlife. Also, serum from mice infected with *B. melis* cross-reacted with larval excretory-secretory antigens from *B. procyonis* (Boyce et al., 1988), so it is important to consider *B. melis* as a possible etiologic agent of hosts with antibodies to *Baylisascaris* spp. in areas where badgers and raccoons are sympatric.

2.6. *Baylisascaris potosis*

A novel *Baylisascaris* species, *B. potosis*, was recently described in kinkajous (*Potos flavus*). Type specimens were collected from captive kinkajous that originated in Cooperative Republic of Guyana (Tokiwa et al., 2014). This species is morphologically similar to *B. procyonis* but was described as a new species based on genetic analysis of several gene targets (i.e., internal transcribed spacer (ITS) 2 region, 28S rRNA gene, and COX1 gene) (Taira et al., 2013; Tokiwa et al., 2014). Kinkajous are common exotic pets in the United States and other countries.

Previously, kinkajous were reported as a host of *B. procyonis*, both in the wild in Columbia and in captivity in the United States and Japan (Overstreet, 1970; Kazakos et al., 2011; Taira et al., 2013;

Parzansky, 2015). Another possible host, the bushy-tailed olingo (*Bassaricyon gabbii*) passed a male *Baylisascaris* (reported as *B. procyonis*) after being fed eggs from a naturally-infected kinkajou from Columbia; however, it is not known if the olingo was infected prior to the experiment (Overstreet, 1970). However, since the description of *B. potosis*, these reports are questionable, and where possible should be confirmed with molecular data.

To evaluate possible paratenic hosts that can develop larva migrans, Tokiwa et al. (2015a), experimentally inoculated Mongolian gerbils. Exposure of gerbils to 100–4000 embryonated eggs resulted in VLM, but no larvae were found in the brain. A squirrel monkey (*Saimiri sciureus*) inoculated with 10,000 *B. potosis* eggs did not develop clinical signs or gross lesions, although a few migrating larvae were recovered from liver and kidney tissues (Tokiwa et al., 2015b). Another squirrel monkey inoculated with 100,000 eggs in the same trial developed gross lesions, including liver congestion, pulmonary edema, and abundant intestinal granulomas. Small granulomatous lesions containing larvae were found in the outer layers of the cerebral cortex, without deeper invasion as is typical of *B. procyonis*. This animal was found dead at 30 DPI, but the animal lacked clinical signs and the authors state that no cause of death was determined. However, pulmonary edema, liver congestion, and nodular lesions containing non-degenerate larvae along the intestine were found at necropsy (Tokiwa et al., 2015b). Based on these preliminary trials, it appears that *B. potosis* can cause larva migrans in rodent and primate hosts, although the pathogenicity and capacity for neural invasion appears less than that of *B. procyonis* or *B. columnaris*.

Because of the recent description of this parasite and the paucity of surveillance in possible hosts in South America apart from a single infected individual, little is known about the natural history of *B. potosis*. Interestingly, raccoons were recently confirmed to have *B. procyonis* infections, based on sequence analysis, in Costa Rica (Baldi et al., 2016). It appears that *B. procyonis* and *B. potosis* are sympatric in procyonids in Central America, highlighting the need for additional research to understand these closely related parasites that may share hosts.

3. Molecular and diagnostic approaches to the study of *Baylisascaris*

3.1. Molecular epidemiology of *Baylisascaris* spp.

Microscopy has been traditionally used to identify *Baylisascaris* spp. based on adult morphological characters although some species can be difficult to distinguish, especially if only immature worms are found. However, because of the similarity among the

sizes of eggs in feces or larvae in tissues, molecular markers have been developed to facilitate identification. For example, multiple single nucleotide polymorphisms (SNPs) in mitochondrial and nuclear gene sequences of *B. columnaris*, *B. procyonis* and *B. transfuga*, have been used to develop species-specific diagnostic molecular markers for rapid identification of different *Baylisascaris* species (Blizzard et al., 2010; Testini et al., 2011; Franssen et al., 2013). Studies have also characterized the genetic diversity, investigated the population structure and the phylogenetic relationships among *Baylisascaris* species, which is important for understanding the zoonotic potential and host specificity of these parasites. To discuss the phylogenetic relationships among the *Baylisascaris* spp. that occur in the New World in a broader context, this section includes data published on *Baylisascaris* spp. from Asia and Europe and *Baylisascaris procyonis* from raccoons.

3.1.1. Phylogenetic relationships

Based on analysis of numerous genetic targets (Table 7), *Baylisascaris* is most closely related to the genus *Ascaris*. The entire mitochondrial genome has been sequenced for four species—*B. transfuga*, *B. ailuri*, *B. schroederi* and *B. procyonis* (all samples from China) (Xie et al., 2011a, 2011b; Li et al., 2012). Phylogenetic analyses of these mitochondrial genomes and concatenated partial mitochondrial and nuclear genes (12S rDNA, 18S rDNA and 28S rDNA) provide the strongest support for the relatedness of the genus *Baylisascaris* with *Ascaris* and other members of the order Ascaridida (Xie et al., 2011a, 2011b; Li et al., 2012).

Within the *Baylisascaris* genus, phylogenetic relationships have been investigated using several molecular targets (i.e., numerous mitochondrial genes, nuclear 5.8S, and second internal transcribed spacer (ITS-2) rDNA sequences). The three ursid-specific *Baylisascaris* species (*B. transfuga*, *B. ailuri* and *B. schroederi*) are more closely related to each other than to *B. procyonis* and *B. ailuri* is more similar to *B. transfuga* than to *B. schroederi* (Xie et al., 2011a, 2011b; Li et al., 2012). Evidence from nuclear 5.8S and ITS-2 rDNA sequences also showed higher genetic similarity between *B. transfuga* and *B. schroederi* compared to *B. procyonis* (Zhao et al., 2012).

Although no molecular data are available for *B. laevis*, *B. melis* or *B. devosi*; mitochondrial and nuclear genes of *B. columnaris* and *B. potosis* have recently been characterized and their phylogenetic relationship with other *Baylisascaris* species has been examined. The first phylogenetic analysis of *B. columnaris* from pet skunks in Europe showed closer affinity to *B. procyonis* compared to *B. transfuga* (Franssen et al., 2013) based on mitochondrial cytochrome c oxidase 1 and 2 (CO1 and CO2), ribosomal ITS1-5.8S-ITS2 and ribosomal 28S genes. This result was expected because *B. columnaris* was previously shown to be very similar to *B. procyonis* based on partial mitochondrial CO2 gene sequences (Danguodobiyam et al., 2009). Phylogenetic analyses of the mitochondrial CO1 and ITS2 rDNA gene sequences showed that *B. potosis* had high genetic similarity to *B. procyonis* and *B. columnaris* (Tokiwa et al., 2014).

A comprehensive phylogenetic assessment of different *Baylisascaris* species at common gene targets would facilitate a better understanding of genetic similarities/differences between the different species. Given that many species of *Baylisascaris* span a very wide geographic scale, genetic differences likely exist among these populations suggesting the existence of cryptic species. For example, *B. melis* is endemic to both Eurasia and the Americas and *B. transfuga* similarly has a large geographic and host range. The morphologic variability in *B. transfuga* also should be examined using molecular tools to assess if these indicate the presence of multiple species. Genetic studies are critically needed to evaluate species validity and both fine-scale and broad-scale geographic

variability for all *Baylisascaris* species.

3.1.2. Population structure

Genetic markers are widely used to assess population structure and provide important insights into host-parasite transmission dynamics. For example, giant panda (*Ailuropoda melanoleuca*) populations were genetically distinct across the three mountain ranges in China but *B. schroederi* were not, suggesting little co-evolution between hosts and parasites and high levels of parasite gene flow (Zhou et al., 2013; Xie et al., 2015). High genetic variation within the parasite populations on each mountain range was observed, but the lack of population diversity across the mountain ranges suggested a homogenous parasite population, based on the complete mitochondrial *cytb*, *atp6* and *cox1* gene targets. In contrast, use of microsatellite markers revealed two genetic clusters in *B. procyonis* across the Grand River in Western Michigan, USA (Sarkissian et al., 2015). Lack of population structure in *B. schroederi* parasites across the mountain ranges in China indicates the fast evolving rate of parasites compared to their hosts and microsatellite markers may be able to further confirm if there is any recent genetic divergence between the parasite populations. Thus, choice of appropriate genetic marker is important while assessing parasite population structure and understanding host-parasite evolutionary dynamics.

Low genetic diversity was found within *B. columnaris* in the Netherlands. Multi-locus genetic analysis revealed four distinct genotypes, possibly owing to the differences in the host or geographic origin of these parasites (Franssen et al., 2013). However, a wider sampling of infected hosts from other geographical regions is warranted to reveal the true genetic diversity of these parasites.

3.2. Diagnostic considerations

As discussed throughout this review, accurately distinguishing species based only on morphologic characteristics can be a challenge. Not only does considerable overlap in morphometry exist (Table 1), but most species associations may not be as strict as often assumed as limited experimental data suggest that *B. columnaris* can infect raccoons and *B. procyonis* and *B. devosi* can infect skunks (Berry, 1985; Sprent, 1953a). Although the prevalence of these cross-infections in naturally-infected hosts is not understood, parasite species identity should not be assumed based on host species. When possible, molecular identification of species should be used for species confirmation of eggs, larvae, and possibly adult nematodes if only female or immature worms are present. Until this is widely implemented, our understanding of host specificity among *Baylisascaris* spp. will remain limited.

Additionally, current serologic methods cannot distinguish between species of *Baylisascaris* causing larva migrans in paratenic hosts. Antisera from animals infected with different *Baylisascaris* spp. all show reactivity to crude *B. procyonis* excretory-secretory (ES) antigen fractions as well as a recently developed recombinant antigen (BpRAG-1) (Boyce et al., 1988; Danguodobiyam et al., 2010). Further work is needed to determine if species-level differences in ES or other antigen targets exist, and if these differences would be sufficiently different to allow the development of species-specific serodiagnostics. This is a potentially difficult goal as serologic differentiation between two other related ascarids, *Toxocara canis* and *Toxocara cati*, has not been successful with current platforms, and these are both important zoonotic parasites (Poulsen et al., 2015). Therefore molecular identification of recovered larvae from paratenic hosts, if possible, is the ideal method of confirming species. Some serologic assays may be useful in determining exposure to parasites in the genus, given that the target antigen is not cross-reactive with other ascarids.

Table 7
Summary of different PCR primers available for identification of *Baylisascaris* species.

Target gene	Length of the target gene	Species	Primer	Reference
ATPase subunit 6 (<i>atp6</i>)	600bp	<i>B. schroederi</i>	F (5'-CGCGGATCCTTCGATATTCGTGG CCT-3') R (5'-CGCAAGCTTCTAATATGGTGTCTT CGG-3')	Xie et al., 2015
cytochrome oxidase c subunit I (<i>CO1</i>)	413bp	<i>B. procyonis</i> , <i>B. transfuga</i> , <i>B. columnaris</i>	F: (5'-TTTTTGGGCATCCTGAGGTTTAT-3') R: (5'-TAACGACATAACATAATGAAAATG-3')	Franssen et al., 2013
cytochrome oxidase c subunit 2 (<i>CO2</i>)	1578bp	<i>B. schroederi</i>	F (5'-TTTAGAGGTTGGAATGTAGGGT-3') R (5'-CCATCCCTTAATCTGCAAT-3')	Xie et al., 2015
	483bp	<i>B. procyonis</i> , <i>B. transfuga</i> , <i>B. columnaris</i>	F: (5'-AATTTTAAITGTAGTCTTTTGTGG-3') R: (5'-CTATGATTAGCACCACAATC-3')	Franssen et al., 2013
mitochondrial cytochrome b <i>cytb</i>	~1500bp	<i>B. schroederi</i>	<i>Cytb</i> -1 (5'-GGTGCTATGCTCGTTACG-3') <i>Cytb</i> -2 (5'-CCACTAAGACCTCCATT-3')	Zhou et al., 2013
12s rRNA	499bp	<i>B. schroederi</i> , <i>B. ailuri</i> , <i>B. transfuga</i> , <i>B. procyonis</i>	F (5'-AGCGGAGGAAAAGAACTAA3') R (5'-TGATCCTTCTGCAGG TTCACCTAC-3)	Li et al., 2012
18s rDNA	1708bp	<i>B. schroederi</i> , <i>B. ailuri</i> , <i>B. transfuga</i> , <i>B. procyonis</i>	F (5'-AGCGGAGGAAAAGAACTAA-3') R (5'-TGATCCTTCTGCAGG TTCACCTAC-3')	Li et al., 2012
28s rDNA	751bp	<i>B. schroederi</i> , <i>B. ailuri</i> , <i>B. transfuga</i> , <i>B. procyonis</i>	F (5'-CCCATTGATTCTGTCCGC-3') R (5'-TGATCCTTCTGCAGG TTCACCTAC-3')	Li et al., 2012
	718bp	<i>B. procyonis</i> , <i>B. transfuga</i> , <i>B. columnaris</i>	F: (5'-CGAGGATTCCTTAGTAAC-3') R: (5'-TCGGATAGGTGGTCAACG-3')	Franssen et al., 2013
ITS1-5.8S-ITS2	630-650bp ^a	<i>B. procyonis</i> , <i>B. transfuga</i> , <i>B. columnaris</i>	F: (5'-ATAGTGAGTTGCACACTAATGT-3') R: (5'-TTATATGCTTAAATTCAGCGGG-3') ITS2-F: (5'-GCCATTTATGAATTTTCAACATGG-3') ITS2-R: (5'-AGTTATATGCTTAAATTCAGCGG-3')	Franssen et al., 2013
Complete mt genome- (<i>atp6</i> , <i>CO1</i> – <i>CO3</i> , <i>cytb</i> , <i>nad1</i> – <i>nad6</i> <i>nad4L</i> , 22 transfer RNA (<i>trn</i>) Genes (small (<i>rrnS</i>) and large (<i>rrnL</i>) subunits)		<i>B. schroederi</i> , <i>B. ailuri</i> , <i>B. transfuga</i> , <i>B. procyonis</i>	See reference for numerous sets of primers.	Xie et al., 2011a; Xie et al., 2011b
ITS2 rDNA	301 bp	<i>B. transfuga</i>	F (5'-TTATGAATTTTCAACATGGC-3') R (5'-GTTAGATGCTTAAATTCAGC-3')	De Ambrogi et al., 2011
3' end of the ITS-1, complete 5.8S and ITS-2, and the 5' end of 28S rDNA	700bp	<i>B. schroederi</i>	<i>zghu</i> (5'-AAGGTGGAGAGAAAGCTCTC GT-3') NC2 (5'-TTAGTTTCTTTTCTCCGCT-3')	Zhao et al., 2012
ITSs (18s and 28S)	1177bp	<i>B. transfuga</i>	F (5'-ACTGCTGTTTCGAGACCTTTCGAG-3') R (5'-TAGCACCTTCTTGGACTATAGCC-3')	Testini et al., 2011

^a Different length in different parasites due to insertions and tandem repeat.

4. Conclusion

The genus *Baylisascaris* is diverse with importance to wildlife, domestic animal, and public health. However, many knowledge gaps exist regarding species other than *B. procyonis*, which is largely driven by a scarcity of contemporary surveys and application of molecular tools to investigate the ecology of these parasites. Field studies elucidating important life history characteristics are critically lacking for these other, “neglected” *Baylisascaris* spp. Ideally, these future field efforts will incorporate modern molecular approaches along with traditional morphologic examination to better ascertain species diversity, species validity, host range, and disease caused by these parasites in wild definitive and paratenic hosts.

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