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BAYLISASCARIS PROCYONIS AND RELATED SPECIES

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Larva migrans (LM) refers to the prolonged migration and persistence of helminth larvae in the organs and tissues of humans and animals (Beaver 1969; Kazacos 1997). In these hosts, the larvae behave as they would in their natural intermediate or paratenic hosts, which are usually small mammals or birds. During their migration, the larvae may produce extensive tissue damage and inflammation, leading to diverse clinical disease. Larva migrans is separated clinically and pathologically into visceral (VLM), ocular (OLM), neural (NLM) and cutaneous larva migrans (CLM), based on the main organ systems involved (Kazacos 1997). A large number of helminth parasites of lower animals, particularly carnivores, are potential causes of larva migrans affecting the deeper tissues of man and other animals, that is, visceral, ocular, and neural larval migrans (Beaver 1969; Beaver et al. 1984; Kazacos 1991, 1996, 1997, 2000; Smyth 1995). These include various ascarids, hookworms, gnathostomes, *Spirometra, Alaria,* and others. Of these, the ascarids are the most important group, with *Toxocara* and *Baylisascaris* accounting for the majority of cases in humans and animals.

Baylisascaris procyonis, the common raccoon ascarid, is the most commonly recognized cause of clinical larva migrans in animals and affects a wide variety of wild and domestic species. Infection with *B. procyonis* is best known as a cause of fatal or severe neurologic disease (NLM, cerebrospinal nematodiasis), which has been seen in > 90 species of mammals and birds in North America. *Baylisascaris procyonis* is also an important zoonosis, producing damaging visceral, ocular, and neural laval migrans in humans. Infection has important health implications for free-ranging and captive wildlife, zoo animals, domestic animals, and human beings, on an individual as well as population basis. This chapter will focus on *B. procyonis* as a cause of animal and human disease, with some reference to other *Baylisascaris* species also capable of producing clinical larva migrans; it is dedicated to Drs. Jack D. Tiner and John F.A. Sprent, for their pioneering research on *Baylisascaris* larva migrans.

INTRODUCTION. The ascarids or large roundworms (Superfamily Ascaridoidea) are some of the most common and well-known parasites of mammals. Because of their large size, in heavy infections they may interfere with digestion and cause partial or complete obstruction of the small intestine, negatively affecting animal health. Those ascarids that undergo liver and lung migration in intermediate, paratenic or definitive hosts cause hepatic, pulmonary, and other migration-related damage, with further deleterious effects on the host. Those species whose larvae enter the central nervous system, including *B. procyonis* and relatives, produce some of the most devastating clinical diseases in animals and humans.

Ascaridoids are basically heteroxenous, and most species utilize intermediate or paratenic hosts in their life cycles (Anderson 2000). For ascaridoids of carnivores, these hosts are usually small vertebrates (rodents, rabbits, birds) in the food chain of the definitive host, and transmission is via predation or scavenging. The single most important factor in the success of ascaridoids of terrestrial animals is the marked resistance and longevity of their infective eggs in the environment, which ensures eventual transmission to susceptible hosts. In intermediate or paratenic hosts, larval ascarids commonly undergo somatic migration, entering various organs and tissues where they will become encapsulated and persist, for later transmission to carnivores (Sprent 1952a, 1953a,b; Tiner 1953a,b; Sprent et al. 1973; Sheppard and Kazacos 1997). Thus, ascarids of carnivores, and *B. procyonis* in particular, are excellent examples of helminths that produce larva migrans.

The remarkable disease-producing capability of *B. procyonis* in animals and humans is one of the most important aspects of ascaridoid biology to come to light in recent years. Strikingly nonspecific in their infection of animals, *B. procyonis* larvae undergo aggressive somatic migration in a broad assortment of potential intermediate hosts, in which most of the larvae become encapsulated in various internal organs and tissues. A small percentage of larvae enter the brain, where they produce marked traumatic damage and inflammation that often results in clinical central nervous system disease. *Baylisascaris procyonis* affects a wide variety of birds and mammals, including humans, and is receiving increased attention in North America and Europe. Other closely related ascarids (e.g., *B. columnaris* of skunks and *B. melis* of badgers) are also potential causes of clinical larva migrans in animals and humans.

ETIOLOGIC AGENT. *Baylisascaris procyonis* and relatives are large roundworms in the nematode Order Ascaridida, Superfamily Ascaridoidea. Members of the superfamily are mainly medium- to large-size worms possessing three lips that may be separated by interlabia (Hartwich 1974; Gibson 1983). The superfamily contains five families, with *Baylisascaris* and most other ascarids of terrestrial hosts included in the family Ascarididae (Anderson 2000). Within the Ascarididae, *Baylisascaris, Ascaris, Toxascaris, Parascaris,* and *Lagochilascaris* are in the subfamily Ascaridinae, and *Toxocara* and *Porrocaecum* are in the subfamily Toxocarinae.

The genus *Baylisascaris* was defined by Sprent (1968) to include several ascarids previously named as members of *Ascaris* or *Toxascaris,* but which possess cervical alae with cuticular bars reaching the surface of the cuticle, and characteristic pericloacal roughened areas (area rugosa) in the males (McIntosh 1939; Sprent 1952b, 1970; Hartwich 1962). Dorsal and subventral labial papillae are distinctly double, and males possess stout, uniform spicules, usually < 1 mm long, and discrete precloacal and postcloacal groups of papillae on the tail. The genus currently contains eight recognized (Table 11.1) and two provisional species (Sprent 1968, 1970).

Members of *Baylisascaris* occur primarily in carnivores (Table 11.1), with one species (*B. laevis*) occurring in rodents. Similar to other ascarids of terrestrial carnivores, transmission of most *Baylisascaris* species involves ingestion of larvae in small mammal intermediate hosts; direct infection by eggs also occurs for some species, particularly in young definitive hosts (Tiner 1952a, 1953a; Sprent 1953b; Sprent et al. 1973; Kazacos 1983b; Kazacos and Boyce 1989). *Baylisascaris laevis* of rodents is morphologically similar to *B. columnaris* and *B. procyonis,* and probably arose as a transmission "capture" derived from the heteroxenous forms occurring in carnivores (Berry 1985; Anderson 2000).

Baylisascaris procyonis was first reported (as *Ascaris columnaris*) from raccoons in the New York Zoological Park (McClure 1933) and on a fur farm in Minnesota (Olsen and Fenstermacher 1938). It was later described as a new species (*Ascaris procyonis*) from raccoons in Europe and subsequently included within *Baylisascaris* (Sprent 1968). Synonyms of *B. procyonis* include *A. columnaris* (Leidy 1856) in raccoons, *A. procyonis* (Stefanski and Zarnowski 1951), and *Toxascaris procyonis* (Stefanski and Zarnowski 1951; Sprehn and Haakh 1956). Common names of the parasite include raccoon ascarid, raccoon roundworm, and, in German, Waschbärenspulwurm.

TABLE 11.1—Recognized species of *Baylisascaris* [a]

Parasite	Primary Definitive Host(s)
B. procyonis (Stefanski and Zarnowski 1951)	Raccoons
B. columnaris (Leidy 1856)	Skunks
B. melis (Gedoelst 1920)	Badgers
B. devosi (Sprent 1952)	Martens, fishers
B. transfuga (Rudolphi 1819)	Bears
B. schroederi (McIntosh 1939)	Giant pandas
B. tasmaniensis (Sprent 1970)	Tasmanian devils, quolls, native "cats"
B. laevis (Leidy 1856)	Marmots, ground squirrels

[a]From Sprent (1968, 1970).

Adult *B. procyonis* are large, tan-colored nematodes, the female reaching 20–22 cm long and the male 9–11 cm long (Hartwich 1962; Sprent 1968; Overstreet 1970; Gey 1998; K.R. Kazacos, unpublished.) Cervical alae are vestigial and inconspicuous, the vulva is located one-fourth to one-third the body length from the anterior end, and males possess pericloacal roughened areas (Sprent 1968; Overstreet 1970; Berry 1985; Averbeck et al. 1995). Morphological features have been examined by scanning electron microscopy (Kazacos and Turek 1982; Snyder 1989). The eggs of *B. procyonis* are ellipsoidal in shape, brown in color, contain a large single-celled embryo, and have a thick shell with a finely granular surface (Fig. 11.1a); they range in size from 63–88 x 50—70 µm, with most averaging 68–76 x 55–61 µm (Overstreet 1970; Kazacos and Turek 1983; Kazacos and Boyce 1989; Sakla et al. 1989; Miyashita 1993; Averbeck et al. 1995; Van Andel et al. 1995; Conboy 1996; Gey 1998).

LIFE HISTORY AND TRANSMISSION. Adult female worms in the small intestine of raccoons produce an estimated 115,000–179,000 eggs/worm/day, so that infected raccoons shed millions of eggs/day in their feces (Kazacos 1982; Snyder and Fitzgerald 1987). Naturally infected raccoons shed an average of 20,000–26,000 *B. procyonis* eggs/g feces, with higher shedding rates in juvenile raccoons than in adults (Kazacos 1982, 1983a; Snyder and Fitzgerald 1987). The highest reported shedding is 256,700 eggs/g feces (Kazacos 1983a). With adequate temperature and moisture, *B. procyonis* eggs can reach infectivity (second-stage larva) in 11–14 days (Sakla et al. 1989) (Fig. 11.1b) and can remain infective in the environment for years (Kazacos and Boyce 1989).

Young raccoons become infected by ingesting infective eggs, whereas older raccoons become infected by ingesting third-stage larvae (L_3's) in intermediate hosts, usually rodents (Fig. 11.2) (Tiner 1953a,b; Kazacos 1983a,b; Kazacos and Boyce 1989). Young raccoons become infected at an early age by ingesting eggs from their mother's contaminated teats or fur, from the contaminated den, or from raccoon latrines near their den. In young raccoons, larvae hatching from eggs enter the mucosa of the small intestine and develop there several weeks before reentering the intestinal lumen to mature, the worms reaching patency in 50–76 days (mean, 63). . In older raccoons, larvae from intermediate hosts develop to adults in the intestinal lumen, reaching patency in 32–38 days (mean, 35) (Kazacos 1983b; Kazacos and Boyce 1989). More extensive migration

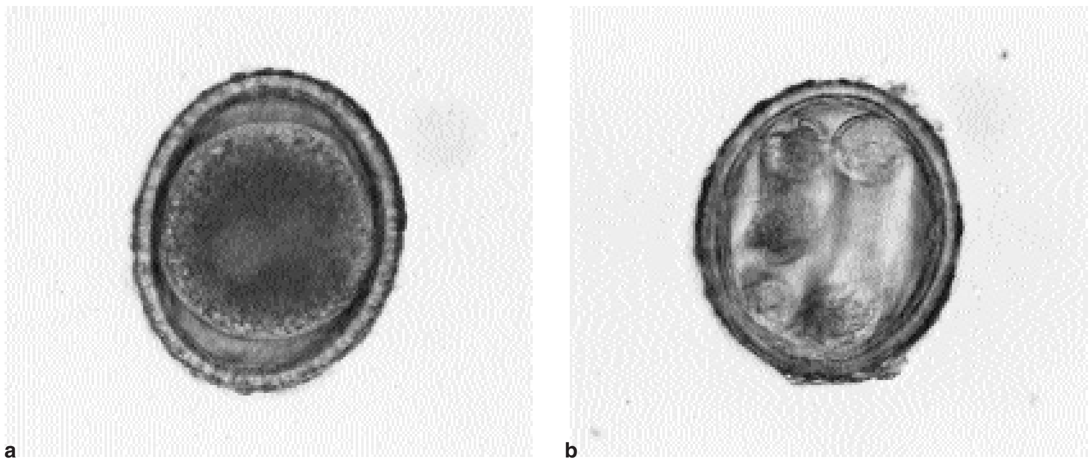

FIG. 11.1—**(a)** Undeveloped *Baylisascaris procyonis* egg from fresh raccoon feces. Note ellipsoidal shape, large single-celled embryo, and finely granular surface. **(b)** The other egg is infective and contains a second-stage larva. [Figure 1b reprinted from Kazacos (1983a) with the permission of Purdue Research Foundation.]

FIG. 11.2—Life cycle of *Baylisascaris procyonis.*

of *B. procyonis,* including somatic migration, does not appear to occur in raccoons, and although transmammary and transplacental transmission have not been investigated, based on the above their occurrence would be doubtful.

Tiner (1949, 1951, 1952a, 1953a,b) reported that *B. procyonis* larvae would produce fatal central nervous system disease in various experimentally infected rodents (white mice, house mice, cotton rats, hamsters, guinea pigs, gray squirrels). He also recovered similar larvae from the tissues of wild-caught fox squirrels and white-footed mice from raccoon-infested woodlots in Illinois (Tiner 1952a, 1953a). Based on counts of granulomas and larvae in wild-caught mice, Tiner (1954) estimated that 5% of *P. leucopus* mortalities in these woodlots were caused by *B. procyonis.* Since that time, *B. procyonis* has become well known as an important cause of morbidity and mortality in individuals and populations of small vertebrates sharing or frequenting the habitat of infected raccoons. *Baylisascaris* larvae have been recovered from numerous species of mammals and birds, most of which were suffering from clinical central nervous system disease (Kazacos and Boyce 1989; Sheppard 1995).

In intermediate hosts, *B. procyonis* larvae undergo aggressive somatic migration, similar to the larvae of several other carnivore ascarids (Sprent 1952a, 1955; Tiner 1953a,b). After ingestion, eggs hatch in the small intestine, and larvae quickly penetrate the intestinal wall and migrate through the liver to the lungs, presumably via the portal circulation and associated vascular channels. Pulmonary hemorrhages are evident by 12–48 hours postinfection, caused by larvae breaking out of capillaries in the lungs (Sprent 1952a, 1953b, 1955; Kazacos 1986). The larvae then enter the pulmonary veins, thereby gaining access to the left heart and systemic arterial circulation, which distributes them throughout the body but especially to the head and anterior carcass (Sprent 1952a, 1955; Tiner 1953b; Sheppard and Kazacos 1997). A few larvae probably migrate locally in the abdominal viscera after initial infection, and in the thoracic cavity once they reach the lungs; some others undergo tracheal migration, being swallowed and reentering the gastrointestinal tract. In white mice infected with *B. procyonis,* larvae enter the somatic tissues, eyes, and brain as early as 3 days postinfection (Tiner 1953b; Kazacos et al. 1985; Kazacos 1986), and clinical central nervous system disease is evident by 9–10 days postinfection (Tiner 1953a; Sheppard and Kazacos 1997). Larvae in visceral and somatic tissues become encapsulated in eosinophilic granulomas, where they will remain until ingested by raccoons. Host differences exist in the relative distribution and encapsulation of larvae in various tissues, but larvae entering the central nervous system of different hosts appear to be equally pathogenic (Tiner 1953a,b; Wirtz 1982; Sheppard and Kazacos 1997).

Larvae migrating in the brain produce traumatic damage and inflammation, resulting in progressive central nervous system disease, the onset and severity of which are dose related. The number of *B. procyonis* larvae entering the brain varies with animal species and dose and may be influenced by prior exposure or other factors (Kazacos and Boyce 1989; Sheppard and Kazacos 1997). A single *B. procyonis* larva in the brain of a mouse or small bird is usually fatal (Tiner 1953a,b; Sheppard and Kazacos 1997), and in natural cases one to five or more larvae are often recovered (Tiner 1953a; Armstrong et al. 1989; Van Andel et al. 1995; K.R. Kazacos, unpublished). In nature, the production of central nervous system disease in intermediate hosts has survival value for *B. procyonis,* because debilitation or death of intermediate hosts would result in increased transmission of *B. procyonis* back to raccoons, via predation or scavenging (Tiner 1953a,b; Kazacos and Boyce 1989; Sheppard and Kazacos 1997). Raccoons are opportunistic carnivores, so it is likely that increased pathogenicity of *B. procyonis* in intermediate hosts has been selected for over time.

Sources of *B. procyonis* infection for intermediate hosts include any areas or articles contaminated with the feces of feral or pet raccoons. In nature, most transmission to intermediate hosts occurs at raccoon latrines, preferred sites of raccoon defecation where their feces and *B. procyonis* eggs accumulate (Fig. 11.3) (Cooney 1989; Page 1998; Page et al. 1999). Raccoon latrines are found most often at the base of trees, in raised crotches of trees, and on large logs, stumps, rocks, tree limbs, and other horizontally oriented structures (Yeager and Rennels 1943; Stains 1956; Cooney 1989; Kazacos and Boyce 1989; Page 1998; Page et al. 1998). They are also found in barn lofts and garages and on woodpiles, decks, roofs, and other locations in the domestic environment (Kazacos et al. 1983; Kazacos and Boyce 1989). Large numbers of *B. procyonis* eggs occur at raccoon latrines, and these areas become important long-term sources of infection. As first alluded to by Tiner (1952a), there is mounting evidence that intermediate hosts, particularly granivorous rodents, become infected with *B. procyonis* by foraging for undigested seeds and other materials present in raccoon feces at latrines (Wirtz 1982; Kazacos and Boyce 1989; Sheppard and Kazacos 1997; Page 1998; Page et al. 1999). Recently, Page (1998) and Page et al. (1999) documented visitation to raccoon latrines by 16 species of mammals and 15 species of birds, with active foraging by white-footed mice (*Peromyscus leucopus*), eastern chipmunks (*Tamias striatus*), fox squirrels (*Sciurus niger*), opossums (*Didelphis virginiana*), white-breasted nuthatches (*Sitta carolinensis*), and hermit thrush (*Catharus guttatus*). Visitation by white-footed mice was significantly greater when corn, their most highly preferred seed type, was present in raccoon feces. Caching of raccoon feces by white-footed mice (Page 1998) and Allegheny woodrats (*Neotoma magister*) (McGowan 1993; K.R. Kazacos and S.A. Johnson, unpublished) has also been documented. Such behavior and subsequent infection with *B. procyonis* have been linked to extirpation of *N. magister* from parts of its

FIG. 11.3—Raccoon latrine on a log in an Indiana woodlot. The raccoon feces contain corn fragments and seeds, and are in various stages of weathering/decomposition.

northeastern range (McGowan 1993). Animals could also become infected while investigating a latrine site, or indirectly through grooming, after having become contaminated at a latrine (Sheppard and Kazacos 1997; Page et al. 1999).

Infection with *B. procyonis* has also been linked to the use of straw, hay, feed, and enclosures contaminated by wild raccoons (Richardson et al. 1980; Kazacos et al. 1982a, 1983, 1986; Armstrong et al. 1989; Sanford 1991; Van Andel et al. 1995; Campbell et al. 1997; Pessier et al. 1997; C.L. Eng and K.R. Kazacos, unpublished; A.M. Lennox and K.R. Kazacos, unpublished; J.C. Martin, unpublished), and cages or enclosures previously used to house raccoons (Schueler 1973; Church et al. 1975; Koch and Rapp 1981; Reed et al. 1981; Larson and Greve 1983; Myers et al. 1983; Dixon et al. 1988; Medway et al. 1989; Fitzgerald et al. 1991; Coates et al. 1995; Garlick et al. 1996; K.R. Kazacos, unpublished; M.A. Nieves et al., unpublished). In zoos, raccoon latrines in open exhibits or on the tops of "roundhouse" enclosures, and contaminated logs or tree limbs placed into exhibits have resulted in infections with *B. procyonis* (Armstrong et al. 1989; Kazacos and Boyce 1989; Stringfield and Sedgwick 1997; K.R. Kazacos, unpublished). An extensive outbreak involving *B. columnaris* infection in three species of marmosets and tamarins was linked to two infected skunks kept in the same exhibit, presumably to make it more "natural" (Huntress and Spraker 1985; K.R. Kazacos and P.L. Wolff, unpublished). Marmosets and tamarins spend much time on the ground foraging, which could lead to infection with *Baylisascaris* (Pessier et al. 1997). At least ten cases of fatal or severe central nervous system disease due to *B. procyonis* have occurred in young children following contact with contaminated areas or articles in the domestic environment.

EPIDEMIOLOGY

Distribution and Prevalence of *B. procyonis* in Raccoons. *Baylisascaris procyonis* is indigenous in raccoons in North America, Europe, and parts of Asia. In North America, it is more common in the midwestern and northeastern United States and along the west coast, where prevalences reach 68%–82% (Kazacos and Boyce 1989) (Table 11.2). The annual prevalence of *B. procyonis* appears to be stable in endemic areas (Table 11.3). In Wisconsin, the prevalence of *B. procyonis* decreases from south to north (75% in the southern half versus 18% in the northern quarter) and correlates with relative raccoon abundance (Amundson and Marquenski 1986). Interestingly, the prevalence of *B. procyonis* also decreases from northern to southern United States, so that the parasite is less common or absent in raccoons in the deep south (Table 11.2). In the southeast, *B. procyonis* appears to be found primarily in mountainous areas, not in the coastal regions or on coastal islands, an exception being south coastal Texas (Kerr et al. 1997). Its farthest known southeastern distribution is central Georgia (Babero and Shepperson 1958). It is important to note that local prevalences of *B. procyonis* may vary, so it is unwise to discount its occurrence in a particular area until an adequate sample of raccoons has been examined. Also, with the influx or translocation of infected raccoons (Lotze and Anderson 1979; Schaffer et al. 1981) and/or changes in their population density, prevalence in an area may change over time. Although *B. procyonis* was not found in east or central Texas by Chandler (1942) or Schaffer et al. (1981), it was identified recently in raccoons in east Texas and south coastal Texas (Table 11.2). Limited data are available on the occurence of *B. procyonis* in other southwestern states, and no data are available from the Rocky Mountain states.

Raccoons are native to North and Central America (Lotze and Anderson 1979) but have been introduced elsewhere, taking *B. procyonis* with them. Raccoons have become well established in major areas of Europe and Asia, following their escape or release decades ago. For example, it is estimated that > 100,000 wild raccoons occur in Germany (C. Bauer, personal communication, 1991), with a prevalence of *B. procyonis* infection of 71% (Gey 1998). The increase in raccoons in Europe has been accompanied by *B. procyonis*–induced larva migrans in various species, including humans (Kelly and Innes 1966; Koch and Rapp 1981; Küchle et al. 1993), the extent of which deserves further investigation. Over 20,000 raccoons have been imported into Japan as pets since 1977, and many have escaped and now inhabit wild areas of central Honshu and Hokkaido. Recently, *B. procyonis* was found in raccoons in Japan (Table 11.2) (Miyashita 1993). Even

TABLE 11.2—Geographic distribution, prevalence, and intensity of intestinal *Baylisascaris procyonis* in raccoons (*Procyon lotor*)

Geographic Location	No. Examined	Percent Infected	Intensity: Mean (Range)	Reference/Source
1. United States: Midwest				
Illinois	6	4 of 6	27 (2–71)	Leigh 1940
Central Illinois	1	1 of 1	~120	Tiner 1952a
Central Illinois/Wisconsin	na[a]	na	na	Tiner 1953a
Southern Illinois	36	64	na	Barnstable and Dyer 1974
NE Illinois (Chicago)	26	42	21 (1–101)	Pigage et al. 1983
Illinois	310	82	52 (1–328)	Snyder and Fitzgerald 1985
Illinois	100	86	52 (1–241)	Snyder and Fitzgerald 1987
Southern Illinois	60	5	na	Birch et al. 1994
Indiana	25	28	na	Robinson et al. 1957
Central Indiana	4	3 of 4	na	Reed et al. 1981
Indiana	95[b]	20	na	Jacobson et al. 1982
	218[c]	29	na	Jacobson et al. 1982
Indiana:	1425	72	na	Kazacos and Boyce 1989
1982 sample	391	74	43 (1–283)	Kazacos, unpub.
NW Indiana	219[c]	15	na	Cooney 1989
Iowa	1	1 of 1	(quite abundant)	Morgan and Waller 1940
Iowa	10	20	na	Waller 1940
Iowa	24[c] and 22[c]	13 and 73	na	Greve 1985
Iowa	25[b]	48	na	Hill et al. 1991
Kansas	na	na	na	Lindquist 1978
NE Kansas:	128	44	(1–263)	Robel et al. 1989
Fort Riley	36	33	14 (1–124)	Robel et al. 1989
Rural Manhattan	92	75	21 (1–263)	Robel et al. 1989
Eastern Kansas	8	5 of 8	na	Ball et al. 1998
Michigan	256	0	na	Stuewer 1943
Michigan, Wisconsin, and Ohio	25	32	(1–18)	R.L. Rausch, unpub. (1944–48); in Hoberg and McGee 1982
SE Michigan	33	58	27 (1–110)	Schultz 1962
Michigan	1	1 of 1	na	Thomas 1988
Minnesota	1	1 of 1	(numerous)	Olsen and Fenstermacher 1938
Minnesota	1	0	na	Larson and Scharf 1975
Minnesota:	163	61	na	S.A. Schmit, G.A. Averbeck, and B.E. Stromberg, unpub.na
Twin Cities Metro Area	109	66	na	S.A. Schmit, G.A. Averbeck, and B.E. Stromberg, unpub.
Nebraska	4	3 of 4	na	Armstrong et al. 1989
Southern Ohio	1	1 of 1	na	Rausch 1946
Central Ohio	28	25	na	Dubey 1982
Ohio and Northern West Virginia	10	20	3 (?–5)	Schaffer et al. 1981
South Dakota	250	12	na (?–46)	Boddicker and Progulske 1968
Wisconsin:	213	51	na (1–241)	Amundson and Marquenski 1986
Southern 1/2	114	75	48	Amundson and Marquenski 1986
Northern 1/2	99	23	28	Amundson and Marquenski 1986
2. United States: Northeast/Middle Atlantic				
Connecticut	1	1 of 1	(1321)	Carlson and Nielsen 1984
Maryland	19	5	(many)	Habermann et al. 1958
Maryland	304[b]	30	na	K.R. Kazacos and N.P. Garner, unpub.
New Jersey	21[b]	24	na	LoGiudice 1995
	137[c]	34	na	LoGiudice 1995
New York (New York City)	1	1 of 1	(10)	McClure 1933
New York (New York City)	na	+++[d]	na	Herman 1939
Eastern New York	2	2 of 2	389 (141–636)	Stone 1983
Western New York	429	68	48 (1–480)	Ermer and Fodge 1986
Southern New York (Ithaca)	277[b]	20 (4–42)	na	Kidder et al. 1989
SE New York (Long Island)	49[c]	39	na	Feigley 1992
SE Pennsylvania	1	1 of 1	5	Dubey et al. 1992
Washington, DC	23	35	na	Tecec 1987
	21[c]	52	na	Tecec 1987

TABLE 11.2 (*continued*)

Geographic Location	No. Examined	Percent Infected	Intensity: Mean (Range)	Reference/Source
3. United States: Southeast				
Alabama	371	0	na	Johnson 1970
Arkansas	30	0	na	Richardson et al. 1992
Florida	19	0	na	Harkema and Miller 1964
Central Florida	51	0	na	Schaffer et al. 1981
Southern Florida (Miami)	90	0	na	K.R. Kazacos et al., unpub. [1997]
Central Georgia	6	+++	na	Babero and Shepperson 1958
Eastern Georgia (Ossabaw Is.)	100	0	na	Jordan and Hayes 1959
Georgia	22	0	na	Harkema and Miller 1964
Northern Georgia	110	1	na	V.F. Nettles, unpub. [1976–77] in Kazacos and Boyce 1989
Northern Georgia/ Western North Carolina	23	0	na	Schaffer et al. 1981
SE Georgia	10	0	na	Schaffer et al. 1981
Eastern Georgia (St.Cath.Is.)	32	0	na	Price and Harman 1983
Western Kentucky	70	30	(?–61)	Cole and Shoop 1987
North Carolina:				
Coastal	61	0	na	Harkema and Miller 1964
Inland	148	0	na	Harkema and Miller 1964
SE North Carolina	10	0	na	Schaffer et al. 1981
South Carolina (Cape Is.)	16	0	na	Harkema and Miller 1962
South Carolina:				
Cape Island	17	0	na	Harkema and Miller 1964
Coastal	16	0	na	Harkema and Miller 1964
Inland	31	0	na	Harkema and Miller 1964
South Carolina	128	0	na	Yabsley and Noblet 1999
Tennessee	253	8	(1–221)	Bafundo et al. 1980
NE Tennessee/Virginia	20	0	na	Schaffer et al. 1981
NW Tennessee/ SW Kentucky	145	3	(?–83)	Smith et al. 1985
Virginia	6	0	na	Harkema and Miller 1964
Western Virginia	7	5 of 7	(17–93)	Jacobson et al. 1976
Eastern Virginia (coastal)	10	0	na	Schaffer et al. 1981
Virginia:				
Eastern	38	0	na	Jones and McGinnes 1983
Western (mountains)	34	56	16 (?–129)	Jones and McGinnes 1983
4. United States: West/Southwest				
Southern California	na	+++	na	Voge 1956
Southern California	na	+++	na	Overstreet 1970
Northern California	12	67	na	Goldberg et al. 1993
Northern California	26	58	na	Park et al. 1998
Northern California	56	70 (52 and 18[b])	(1–tremendous nos.)	W.J. Murray et al., unpub. [1999]
Northern California (coastal)	15	100	na	W.J. Murray et al., unpub. [1998]
Western Nevada	1[c]	1 of 1	na	G.N. Cooper and R.D. Anderson, unpub.
Oklahoma	1	1 of 1	(large nos.)	Campbell et al. 1997
Oregon	1	0	na	Senger and Neiland 1955
Eastern Texas	13	0	na	Chandler 1942
Central Texas	37	0	na	Schaffer et al. 1981
Eastern Texas	62[b]	23	na	S,C, Waring and D.D. Dingley, unpub.; in Kazacos and Boyce 1989
Southern Texas (coastal)	33	70	6 (1–28)	Kerr et al. 1997
SW Washington	29	3	1	McNeil and Krogsdale 1953
Washington	62	79	na	W.J. Foreyt, unpub. [1998]
5. Canada				
SW British Columbia	82	61	27 (1–226)	Ching et al. 2000
Nova Scotia	219	8	na	Anderson and Mills 1991
Nova Scotia	236	7	(1–46)	Smith 1992
Nova Scotia	491	7	na	J.K. Mills, unpub. [1993]
Ontario	na	+++	na	Sprent 1968
Ontario (Toronto)	23	43	(numerous)	Cranfield et al. 1984
Southern Ontario (Guelph)	41	51	(1–40)	Berry 1985
Prince Edward Island	50	2	na	G.A. Conboy, unpub. [1998]
Quebec	21	57	(?–86)	Mackay et al. 1995
Southern Saskatchewan	31	0	na	Hoberg and McGee 1982

(*continued*)

TABLE 11.2 (*continued*)

Geographic Location	No. Examined	Percent Infected	Intensity: Mean (Range)	Reference/Source
6. Germany				
Brandenburg	41	0	na	Lux and Priemer 1995
Hessen	185	71	(1–232)	Gey 1998
7. Poland	1	1 of 1	50	Stefanski and Zarnowski 1951
8. Czech and Slovak Rep.	1	1 of 1	2	Tenora et al. 1991
	1	1 of 1	28	Tenora and Stanek 1990
9. Japan	291[b]	27	na	Miyashita 1993
Zoos (n=21)	178[b]	40	na	Miyashita 1993
Animal dealers (n=6)	37[b]	8	na	Miyashita 1993
Pets	39[b]	8	na	Miyashita 1993
Wild	37[b]	0	na	Miyashita 1993

[a]na = not available
[b]Examination of raccoon feces for eggs.
[c]Examination of raccoon feces from latrines for eggs.
[d]+++ = positive, no numbers given.

TABLE 11.3—Prevalence of *Baylisascaris procyonis* in raccoons in Indiana, 1981-1986 [a]

Year	No. Examined	Percent Infected
1981	157	72
1982	391	74
1983	145	68
1984	259	73
1985	308	72
1986	165	67
Total	1425	72

[a]Animals collected in November and December.

though no cases of *B. procyonis*–induced larva migrans have been reported from Japan, its occurrence there can be predicted with certainty based on what is known from North America and Europe. *Baylisascaris procyonis* was also recovered from a kinkajou (*Potos flavus*) in Colombia (Overstreet 1970), thereby extending the geographical range of the parasite into South America. The parasite may occur in raccoons in Central America and in other procyonids in the Americas, although this has not been studied.

Prevalence and Intensity of *B. procyonis* in Raccoons. In areas where *B. procyonis* is common in raccoons, it has much higher prevalence in juvenile raccoons (> 90%) than in adults (37%–55%). Average parasite intensity ranges from 43 to 52 worms, with juvenile raccoons having a higher mean intensity (48–62, range 1–480) than adult raccoons (12–22, range 1–257) (Table 11.4) (Snyder and Fitzgerald 1985; Ermer and Fodge 1986; K.R. Kazacos, unpublished). The highest reported worm numbers (636 and 1321) occurred in juvenile raccoons that died of intestinal obstruction due to *B. procyonis,* with resulting starvation and emaciation (Stone 1983; Carlson and Nielsen 1984). The age distribution of *B. procyonis* correlates with what is known about the life cycle (Fig. 11.2), namely that young raccoons in their first season are susceptible to egg infection, whereas adult raccoons become infected via intermediate hosts (Kazacos 1983b; Kazacos and Boyce 1989). Thus, the parasite appears to be recruited into the raccoon population mainly through the young, which have higher worm burdens and prevalence of infection. Age resistance and/or intestinal immunity with self-cure may also contribute to the lower prevalence of *B. procyonis* in older raccoons.

There is mounting evidence that *B. procyonis* may undergo a yearly cycle in raccoons in temperate regions, with self-cure occurring in winter months (January-February). New infections are recruited into the raccoon population in late spring and summer, and the overall prevalence peaks in the fall (September-November) (Schultz 1962; Smith et al. 1985; Kidder et al. 1989; K.R. Kazacos, unpublished). Based on necropsies, Schultz (1962) first found *B. procyonis* in 3-month-old raccoons, with highest intensities at 5–6 months of age (September-October). Kidder et al. (1989) examined raccoon fecal samples from July 1986 to May 1987 and found the highest prevalence of patency (42%) in September-November, vs. 6% in December-August; this correlated with higher prevalences in both juvenile (61%) and adult (23%) raccoons in September-November as compared to the rest of the year (10% and 4%, respectively). Based on necropsies, Smith et al. (1985) in Kentucky/Tennessee, K.R. Kazacos (unpublished) in Indiana, and M.W. Dryden (unpublished) in Kansas have identified sharp declines in the prevalence of *B. procyonis* in raccoons in winter, occurring suddenly in January-February in Indiana and Kansas. As suggested by LoGiudice (1995), this sudden loss of worms may be related to the dramatic reduction in food intake by raccoons at this time of year in northern temperate regions, leading to as much as 50% reduction in their body weight (Folk et al. 1968) and negatively impacting worm survival.

TABLE 11.4—Prevalence of *Baylisascaris procyonis* in raccoons in Indiana, November-December 1982

Age/Sex Class (no.)	No. (% Infected)	Parasite Intensity +/- SE	Range
All animals (391)	289 (74)	42.6 +/- 2.6	1–283
Juvenile male (125)	113 (90)	49.3 +/- 4.6[a]	1–283
Juvenile female (139)	129 (93)	47.4 +/- 3.9[a]	1–256
Juvenile (male+female) (264)	242 (92)[b]	48.3 +/- 3.0[ac]	1–283
Adult male (71)	29 (41)	14.9 +/- 3.9	1–81
Adult female (56)	18 (32)	13.0 +/- 3.7	1–56
Adult (male+female) (127)	47 (37)[b]	14.2 +/- 2.8[c]	1–81
Male (juvenile+adult) (196)	142 (73)	42.05 +/- 3.91	1–283
Female (juvenile+adult) (195)	147 (75)	43.04 +/- 3.55	1–256

[a] Based on 108 juvenile males and 124 juvenile females.
[b,c] Significantly different ($P < 0.0001$); one way ANOVA ($F=8.54$; $df=3, 278$).

TABLE 11.5—Intestinal *Baylisascaris procyonis* in nonraccoon hosts

Host	Geographic Location	Natural (N) or Experimental (E)	Number Infected	Intensity: Mean (Range)	Reference/Source
Potos flavus	Colombia	N	na[a]	(13)	Overstreet 1970
	Indiana	N	2	(20–25+)	K.R. Kazacos, unpub.
Bassaricyon gabbii	na	na	1	1	Overstreet 1970
Canis familiaris	Iowa	N	2	(2–3)	Greve and O'Brien 1989
	Missouri	N	2	13 (na)	G.A. Averbeck et al. 1995 and unpub.
	Indiana	N	12	8 (3–13)[b]	K.R. Kazacos, unpub.
	Michigan	N	7+5[c]	2 (1–3)[d]	D.D. Bowman, unpub.
	Japan	E	1/3[e]	6	Miyashita 1993
		E	3/4[f]	(4–5)	Miyashita 1993
Didelphis virginiana	Georgia	E	0/1[e]	na	V.F. Nettles, unpub.; in Kazacos and Boyce 1989
		E	1/1[f]	13	

[a] na = not available.
[b] Based on ten dogs.
[c] Probable infections; based on single fecal exam.
[d] Based on four dogs.
[e] Fed infective eggs.
[f] Fed third-stage larvae.

Host Range of Adult *B. procyonis*. *Baylisascaris procyonis* is restricted primarily to raccoons but has also been found in related procyonids (kinkajous) and other hosts (Table 11.5). Intestinal infections could be expected to occur in coatimundis (*Nasua* spp.) and ringtails (*Bassariscus* spp.). Over two dozen cases of patent *B. procyonis* infection have been identified in domestic dogs (*Canis familiaris*) from the midwestern United States (Table 11.5). In several of these, *B. procyonis* occurred as a mixed infection with *Toxocara canis* and other helminths. It is not known how these dogs became infected with *B. procyonis,* however, 1 of 3 dogs fed infective eggs and 3 of 4 dogs fed L_3's from mice developed intestinal *B. procyonis* infections; no infections were seen in cats fed eggs or larvae (Miyashita 1993). The biological relationship of *B. procyonis* in dogs is particularly interesting when one considers that several dogs have died from severe neural larva migrans due to this parasite (Snyder 1983; Thomas 1988; Rudmann et al. 1996). Whether dogs with patent infections also have larvae in their somatic tissues is not known. Because of their indiscriminate defecation habits, dogs infected with *B. procyonis* would produce more widespread contamination with eggs, posing a particular zoonotic threat. It is likely that canine infection with adult *B. procyonis* is more common and widespread than is currently known.

Partial or complete development of *B. procyonis* probably takes place in other hosts, but the extent is unknown and not easily determined. Berry (1985) showed limited cross-transmission of *B. procyonis* to skunks, but found no genetic evidence that this occurred in nature. The author has anecdotal reports of patent *Baylisascaris* infections naturally occurring in opossums. This is supported by experimental evidence of a patent infection in a young opossum fed mice containing *B. procyonis* larvae (V.F. Nettles, unpublished: cited in Kazacos and Boyce 1989).

Environmental Limitations. In most areas where raccoons occur, there should be no environmental limitations on the presence of *B. procyonis,* although conditions for optimal egg development and survival will vary based on temperature and humidity. *Baylisascaris procyonis* eggs become infective in 11–14 days at 22°C–25° C and 100% humidity (Sakla et al. 1989), similar to eggs of *B. columnaris* (11–16 d) (Berry 1985). Under natural conditions, with cooler and/or fluctuating temperatures, egg development will be slower and will take several weeks to months. For example, *B. procyonis* eggs deposited in spring in Indiana do not develop until ambient temperatures increase, at which time the eggs embryonate slowly and later reach infectivity (K.R. Kazacos, unpublished). Under sufficiently warm but fluctuating temperatures (*e.g.,* cooler nights), most eggs should reach infectivity in 3–4+ weeks.

Embryonated *B. procyonis* eggs stored 9–12 years at 4° C retained their infectivity and central nervous system pathogenicity for mice (Lindquist 1978; W.D. Lindquist, personal communication in Kazacos et al. 1982b). Given adequate moisture, embryonated eggs will last years in the soil, including through harsh winters (Kazacos 1986, 1991; Kazacos and Boyce 1989). Conditions of extreme heat and dryness, as occur in barn lofts and attics in summer months, will kill *B. procyonis* eggs by desiccation, probably in a few weeks or months (Kazacos and Boyce 1989).

It is doubtful that the lack of *B. procyonis* in the deep southeastern United States is based on environmental limitations, since *Toxocara* and other ascarids do very well there. Rather, it is probably a result of the parasite's absence in raccoons colonizing those areas or its failure to establish due to inadequate host/parasite densities. With current high raccoon densities, translocation of raccoons by hunting clubs, pet owners, and others could introduce *B. procyonis* into new areas, where it could establish and pose a threat to indigenous birds and mammals, including humans (Kazacos and Boyce 1989).

Host Range for Clinical Neural Larva Migrans Caused by *B. procyonis* and Relatives. Few other parasites are as indiscriminate as *B. procyonis* in causing neurologic disease in wild, zoo, and domestic animals as well as human beings (Table 11.6). As would be expected, the geographic distribution of animals and humans clinically affected by *B. procyonis* parallels the occurrence and prevalence of the parasite in raccoons in different areas. Wherever raccoons occur or are introduced, the potential exists for disease caused by *B. procyonis,* a situation that should be taken very seriously.

Susceptibility to *Baylisascaris* larva migrans varies among animal groups and species (Wirtz 1982; Sheppard and Kazacos 1997). Animal groups particularly susceptible to *Baylisascaris* NLM include rodents, rabbits, primates, and birds, based on the number of cases and species affected (Table 11.6). Some animal groups and species are only marginally susceptible, with limited migration occurring in the intestinal wall or viscera; others appear to be resistant (Kazacos and Boyce 1989). For example, no cases of *B. procyonis* NLM have been documented in opossums, which are commonly exposed through foraging at raccoon latrines (Page 1998; Page et al. 1999), or in adult domestic livestock or zoo hoofstock, which are commonly exposed through contaminated hay. Very limited or no migration was seen in sheep, goats, and swine experimentally infected with *B. procyonis* (Dubey 1982; Snyder 1983; Kazacos and Kazacos 1984). No cases have been documented in cats or raptors, which eat rodents possibly contaminated with eggs and/or containing L_3's. Shrews (*Blarina brevicauda*) are resistant to experimental infection with *B. procyonis,* at dosages much higher than are lethal to mice (Sheppard 1996; K.R. Kazacos et al., unpublished); the reasons for this resistance are unclear, but may include unique or potent gastrointestinal enzymes and failure of egg hatching or larval survival. Unless complete necropsies are performed, including a thorough examination of the brain in all cases of central nervous system disease, then apparent species limitations to *Baylisascaris* infection should be regarded with caution.

Special circumstances, including prior exposure, concurrent infections (Sheppard and Kazacos 1997), and hormone fluctuations during pregnancy, may also influence infection. Although ruminants appear to be poorly susceptible to infection, a newborn lamb was diagnosed with *Baylisascaris* NLM and could only have been infected prenatally (Anderson 1999). It is hypothesized that pregnancy hormones increased the susceptibility of the ewe to infection and/or stimulated larval migration, with resultant transplacental transmission of larvae to the fetus. Reminiscent of *Toxocara canis* transmission in dogs, but previously unrecognized for *Baylisascaris,* this finding has important potential health implications for other pregnant mammals, including women.

The susceptibility of poikilothermic vertebrates and invertebrates to *Baylisascaris* is for the most part unknown, but infection would not be expected based on typical host stimuli necessary for larval hatching and migration. Berry (1985) was unable to infect northern leopard frogs (*Rana pipiens*), earthworms (*Eudrilus eugenioe*), cockroaches (*Blatta orientalis*), or African crickets (*Acheata domesticus*) with *B. columnaris,* or northern leopard frogs or African crickets with *B. procyonis,* by feeding infective eggs. However, even though egg hatching apparently doesn't occur, invertebrates could possibly serve as paratenic hosts for *Baylisascaris* eggs recently ingested from raccoon or skunk feces.

In much of the geographic range of *B. procyonis,* skunks infected with *B. columnaris* and, in more limited areas, badgers infected with *B. melis* also are found. Both of these parasites are potential causes of clinical NLM (Kazacos and Boyce 1989); however, based on differences in definitive host ecology,

TABLE 11.6—Host range for clinical neural larva migrans (cerebrospinal nematodiasis) caused by *Baylisascaris procyonis* (Bp) and *B. columnaris* (Bc)

Host	Geographic Location	Parasite[a]	Natural (N) or Experimental (E)	Number Affected[b]	Reference/Source[c]
O. Rodentia					
Mus musculus	Illinois	Bp	N	Several	Tiner 1949, 1953a
	Illinois	Bp	E	3/3	Tiner 1952a
Mus musculus (white,lab)	Illinois	Bp	E	12/12; 15/21; 8/9	Tiner 1949, 1952a, 1953a
	Illinois	Bc	E	3/9; 7/11	Tiner 1952a, 1953a
	Australia	Bc	E	4/9; 12/32; [2/9][d] [1/5][d]	Sprent 1953a, 1955
	Minnesota	Bc	E	54/70	Clark et al. 1969
	Indiana	Bc	E	0/12	Boyce et al. 1988b
	Missouri	Bp	E	6/6	Lindquist 1978
	Indiana	Bp	E	25/25	Kazacos 1981
	Ohio	Bp	E	66/66	Dubey 1982
	Indiana	Bp	E	10/10	Wirtz 1982
	Indiana	Bp	E	12/12	Boyce et al. 1988b
	Japan	Bp	E	70/75	Miyashita 1993
	Indiana	Bp	E	24/25	Garrison 1996
	Indiana	Bp	E	28/30	Sheppard and Kazacos 1997
Peromyscus leucopus	Illinois	Bp	N	[1][d]	Tiner 1953a,b, 1954
	Illinois	Bp	E	na; 7/10	Tiner 1949, 1953a
	Illinois	Bc	E	0/6; 1/4	Tiner 1953a
	Indiana	Bp	E	17/30	Sheppard and Kazacos 1997
	Indiana	Bp	N	1+[1][d]	Sheppard and Kazacos 1997 and unpub.[c]
	Indiana	Bp	N/E	10/46	Page 1998
Peromyscus maniculatus	California	Bp/Bc	N	1	R.H. Evans, unpub.
Peromyscus boylei	California	Bp/Bc	N	1	R.H. Evans, unpub.
Chaetodipus californicus	California	Bp/Bc	N	1	R.H. Evans, unpub.
Reithrodontomys megalotis	Indiana	Bp	E	3/3	Sheppard 1996
Zapus hudsonius	Indiana	Bp	E	1/1	Sheppard 1996
Microtus pennsylvanicus	Indiana	Bp	E	29/30	Sheppard 1996
	Ontario	Bc	E	[8][d]	Berry 1985
Microtus ochrogaster	Indiana	Bp	E	3/4	Sheppard 1996
Mesocricetus auratus	Illinois	Bp	E	na	Tiner 1949
	Indiana	Bp	E	32/32	Kazacos 1981
	Indiana	Bp	E	23/23	Wirtz 1982
Thomomys bottae	California	Bp	N	2	R.H. Evans, unpub.
	California	Bp/Bc	N	2	K.R. Kazacos and F.H. Dunker, unpub.
Neotoma magister	New York	Bp	N/E	10/10	McGowan 1993
	New Jersey	Bp	N/E	1	K. LoGiudice, unpub.
	Indiana	Bp	N	1	K.R. Kazacos and S.A. Johnson, unpub.
	Pennsylvania	Bp	N	1	J. Wright et al., unpub.
	Indiana	Bp	E	26/26	K.R. Kazacos, unpub.
Neotoma fuscipes	California	Bp/Bc	N	2	R.H. Evans, unpub.
Sigmodon hispidus	Illinois	Bp	E	6/6; 7/8; 3/3	Tiner 1949, 1952a, 1953a
	Illinois	Bc	E	0/4	Tiner 1952a
Rattus norvegicus (white,lab)	New Jersey	Bp	E	[2/3][d]	Tiner 1954
	Indiana	Bp	E	12/19	Wirtz 1982
Tamias striatus	Indiana	Bp/Bc	N	1	K.R. Kazacos and S.A. Johnson, unpub.
	Indiana	Bp	E	1	K.R. Kazacos, unpub.; in Kazacos and Boyce 1989

(*continued*)

TABLE 11.6 (*continued*)

Host	Geographic Location	Parasite[a]	Natural (N) or Experimental (E)	Number Affected[b]	Reference/Source[c]
Sciurus carolinensis	Illinois	Bp	E	4/6	Tiner 1949, 1952a, 1953a
	Indiana	Bp	E	10/10	Wirtz 1982
	Indiana	Bp	N	3	K.R. Kazacos, unpub.
	Washington	Bp	N	11/16	Tseng 1997
Sciurus niger	Indiana	Bp/Bc	N	2	K.R. Kazacos, unpub.
	California	Bp/Bc	N	5	Stringfield and Sedgwick 1997, and unpub.
Sciurus granatensis	Maryland	Bp	N	1	Schueler 1973
Sciurus sp. (*griseus* or *carolinensis*)	California	Bp	N	2	R.H. Evans, unpub.
Tamiasciurus douglasii	Br. Columbia	Bp	N	1	Coates et al. 1995
Spermophilus tridecemlineatus	Illinois	Bp/Bc	N	28	Fritz et al. 1968
	Illinois	Bp/Bc	N	1	Pigage et al. 1983
	Illinois	Bp/Bc	N	1	J.I. Everitt and S.E. McDonald, unpub.
Spermophilus beecheyi	California	Bp/Bc	N	8	R.H. Evans, unpub.
	California	Bp/Bc	N	1	C.E. Stringfield and C.J. Sedgwick, unpub.
Cavia porcellus	Illinois	Bp	E	na	Tiner 1949, 1953b
	Pennsylvania	Bp	E	na	Donnelly et al. 1989
	Missouri	Bp	N	30/50	Van Andel et al. 1995
	Nova Scotia	Bp	N	2	Craig et al. 1995
Chinchilla lanigera	Pennsylvania	Bp/Bc	N	6	Richter and Kradel 1964
	Ontario	Bp	N	100	Sanford 1991
Cynomys ludovicianus	Iowa	Bp	N	na	Greve 1985
	New York/ Wisconsin	Bp	N	3/52	Dixon et al. 1988
	Illinois	Bp	N	1	K.R. Kazacos, unpub.
Ondatra zibethicus	New York	Bp/Bc	N	4	W.B. Stone, unpub.
Marmota monax	Pennsylvania	Bp/Bc	N	4/4	Richter and Kradel 1964
	Connecticut	Bp/Bc	N	na	Swerczek and Helmboldt 1970
	Connecticut	Bc	E	1/5; [4/5][d]	Swerczek and Helmboldt 1970
	Virginia	Bp	N	3/3	Jacobson et al. 1976
	New York	Bp/Bc	N	6	Fleming and Caslick 1978
	New York	Bp/Bc	N	5/5	Fleming et al. 1979
	Indiana	Bp/Bc	N	3	Kazacos et al. 1981a; and K.R. Kazacos, unpub.
	New York	Bp/Bc	N	12/12	Roth et al. 1982
	Iowa	Bp/Bc	N	na	Greve 1985
	Indiana	Bp	E	3/3	K.R. Kazacos, unpub.
Myocastor coypus	Michigan	Bp/Bc	N	20/35	Dade et al. 1977
	Germany	Bp	N	65	Koch and Rapp 1981
Erethizon dorsatum	Pennsylvania	Bp	N	3	Medway et al. 1989
	Indiana	Bp	N	2	Fitzgerald et al. 1991
	New York	Bp/Bc	N	2	W.B. Stone, unpub.
Castor canadensis	Ireland	Bp/Bm	N	na	Kelly and Innes 1966
	New York	Bp/Bc	N	2	W.B. Stone, unpub.
Dolichotis patagonum	Illinois	Bp	N	4	K.R. Kazacos et al., unpub.
Hydrochaeris hydrochaeris	Illinois	Bp	N	1	K.R. Kazacos et al., unpub.

(*continued*)

TABLE 11.6 (*continued*)

Host	Geographic Location	Parasite[a]	Natural (N) or Experimental (E)	Number Affected[b]	Reference/Source[c]
O. Lagomorpha					
Sylvilagus floridanus	Illinois	Bp	E	na	J.D. Tiner, unpub., in Tiner 1954
	Illinois	Bp/Bc	N	1	Ferris et al. 1960
	Virginia	Bp	N	16/60	Nettles et al. 1975
	Connecticut	Bp/Bc	N	1	Church et al. 1975
	Virginia	Bp	N	18/72	Jacobson et al. 1976
	Virginia	Bp	E	1/1	Jacobson et al. 1976
	Iowa	Bp/Bc	N	na	Greve 1985
	Indiana/Illinois	Bp/Bc	N	3	K.R. Kazacos, unpub.
	Illinois	Bp/Bc	N	1	R.H. Evans, unpub.
Sylvilagus audubonii	California	Bp/Bc	N	3	R.H. Evans, unpub.
Oryctolagus cuniculus	Illinois	Bp	E	na	J.D. Tiner, unpub.; in Tiner 1954
	Connecticut	Bp	N	Several	Church et al. 1975
	Connecticut	Bc	E	3/4	Church et al. 1975
	Michigan	Bp/Bc	N	80	Dade et al. 1975
	Indiana	Bp	N	25	Kazacos et al. 1983
	Iowa	Bp	N	na	Greve 1985
	Indiana	Bp	N	3	K.R. Kazacos, unpub.; in Boyce et al. 1988a
	Indiana	Bp	N	15	Kazacos and Kazacos 1988b
	Indiana	Bp	E	1/1	Boyce et al. 1989
	Ontario	Bp	N	na	P. Lautenslager and S.E. Sanford, unpub.; in Sanford 1991
	Washington	Bp	N	4	Deeb and DiGiacomo 1994
	Illinois	Bp	N	2	K.R. Kazacos, unpub.
	Illinois	Bp	N	6	L.J. Hardy, unpub.
	Illinois	Bp	N	4	P.J. Didier, unpub.
	Illinois	Bp/Bc	N	2	R.H. Evans, unpub.
	Indiana	Bp/Bc	N	6	K.R. Kazacos, unpub.
	Indiana	Bp/Bc	N	3	N.A.Q. Mehdi, unpub.
	Indiana	Bp	N	1	D.D. Harrington, unpub.
	New York	Bp	N	3	L. Roth, unpub.
	New York	Bp	N	2	W.B. Stone, unpub.
O. Carnivora					
Vulpes vulpes	Iowa	Bp	N	4/4	Larson and Greve 1983
Canis familiaris	Illinois	Bp	E	3/5	Snyder 1983
	Michigan	Bp	N	1	Thomas 1988
	Indiana	Bp	N	1	Rudmann et al. 1996
Taxidea taxus	California	Bp	N	1	R.H. Evans, unpub.
Enhydra lutris nereis	California	Bp	N	1	N.J. Thomas et al., unpub.
Mustela putorius furo	Indiana	Bp	E	3/4	Kazacos 1981; Kazacos and Kazacos 1988a
Mustela nivalis	Indiana	Bp	E	1	K.R. Kazacos, unpub.
O. Primates					
Varecia variegata variegata	Oklahoma	Bp	N	2	Campbell et al. 1997
	Rhode Island	Bp	N	4/6	J.C. Martin, unpub.
	Tennessee	Bp	N	3	S.J. Barrett, unpub.
Varecia variegata rubra	Rhode Island	Bp	N	2/3	J.C. Martin, unpub.
Mirza coquereli	California	Bp	N	1	K.R. Kazacos and F.H. Dunker, unpub.
Callithrix geoffroyi	Texas	Bc	N	3	Huntress and Spraker 1985
	Illinois/Texas	Bc	N	1	K.R. Kazacos and P.L. Wolff, unpub.
Saguinus nigricollis	Texas	Bc	N	1	Huntress and Spraker 1985

(*continued*)

TABLE 11.6 (*continued*)

Host	Geographic Location	Parasite[a]	Natural (N) or Experimental (E)	Number Affected[b]	Reference/Source[c]
Saguinus midas	Texas	Bc	N	1	Huntress and Spraker 1985
Leontopithecus rosalia chrysomelas	Maryland	Bp	N	2	Pessier et al. 1997
	California	Bp/Bc	N	3	Stringfield and Sedgwick 1997; Pessier et al. 1997
Saimiri sciureus	Indiana	Bp	E	4/4	Kazacos et al. 1981b
Macaca fascicularis	Indiana	Bp	E	4/4	Kazacos et al. 1984b, 1985
Hylobates lar	Kansas	Bp	N	1	Ball et al. 1998
Ateles sp.	Maryland	Bp	N	1	Garlick et al. 1996
Cercopithecus neglectus	Indiana	Bp	N	2	C.L. Eng and K.R. Kazacos, unpub.
Homo sapiens	Missouri	Bp/Bc	N	1[e]	Anderson et al. 1975
	Pennsylvania	Bp	N	1	Huff et al. 1984
	Illinois	Bp	N	1	Fox et al. 1985
	New York	Bp	N	1[e]	Cunningham et al. 1994
	California	Bp	N	1	Rowley et al. 2000
	California	Bp	N	1[e]	Park et al. 2000
	California	Bp	N	1	W.A. Kennedy et al., unpub.
	Michigan	Bp	N	1[e]	J.M. Proos et al., unpub.
	Illinois	Bp	N	1[e]	M.B. Mets et al., unpub.
	Minnesota	Bp	N	2[e]	C.L. Moertel et al., unpub.
O. Marsupialia					
Macropus rufus	Michigan	Bp	N	11/20	Agnew et al. 1994
O. Artiodactyla					
Ovis aries	Idaho	Bp/Bc/Bm	N	1/3	Anderson 1999
O. Galliformes					
Gallus gallus (domesticated)	Indiana	Bp	N	622	Richardson et al. 1980
	Indiana	Bp	E	17/50	Kazacos and Wirtz 1983
Colinus virginianus	Indiana	Bp	N	85/85	Reed et al. 1981
	Iowa	Bp	N	na	Greve 1985
	Kansas	Bp/Bc	N	1	Williams et al. 1997
Callipepla californica	California	Bp/Bc	N	4	R.H. Evans, unpub.
Alectoris chukar	Maryland	Bp/Bc	N	1/30	Sass and Gorgacz 1978
Bonasa umbellus	New York	Bp/Bc	N	3	W.B. Stone, unpub.
Phasianus colchicus	Wisconsin	Bp	N	200-400	Kazacos et al. 1986
Alectura lathami	Indiana/Missouri	Bp	N	1	Kazacos et al. 1982a
Meleagris gallopavo	New York	Bp/Bc	N	2	W.B. Stone, unpub.
O. Columbiformes					
Columba livia	Oregon	Bp/Bc	N	10/45	Helfer and Dickinson 1976
	Illinois	Bp/Bc	N	1	Evans and Tangredi 1985
	Br.Columbia	Bp	N	2	Coates et al. 1995
	Nebraska	Bp	N	>15	V. Rinne and E.W. Pendleton, unpub.
Zenaida macroura	New York	Bp/Bc	N	2	Evans and Tangredi 1985
	Illinois	Bp/Bc	N	>25	C.U. Meteyer et al., unpub.
	California	Bp, Bp/Bc	N	9	R.H. Evans, unpub.

(*continued*)

TABLE 11.6 (*continued*)

Host	Geographic Location	Parasite[a]	Natural (N) or Experimental (E)	Number Affected[b]	Reference/Source[c]
O. Passeriformes					
Passer domesticus	California	Bp	N	2	R.H. Evans, unpub.
Psaltriparus minimus	California	Bp	N	3	R.H. Evans, unpub.
Serinus canarius	California	Bp/Bc	N	2	B.C. Barr, unpub.
Carpodacus mexicanus	California	Bp/Bc	N	2	R.H. Evans, unpub.
Pipilo maculatus	California	Bp/Bc	N	1	R.H. Evans, unpub.
Toxostoma redivivum	California	Bp/Bc	N	2	R.H. Evans, unpub.
Turdus migratorius	Illinois	Bp/Bc	N	1	Evans and Tangredi 1985
Cyanocitta cristata	Illinois	Bp/Bc	N	2	Evans and Tangredi 1985
Aphelocoma californica	California	Bp/Bc	N	2	R.H. Evans, unpub.
Mimus polyglottos	California	Bp/Bc	N	7	R.H. Evans, unpub.
Sturnus vulgaris	California	Bp/Bc	N	1	R.H. Evans, unpub.
Corvus brachyrhynchos	New York	Bp/Bc	N	1	B.P. Tangredi and K.R. Kazacos, unpub.
O. Psittaciformes					
Melopsittacus undulatus	California	Bp/Bc	N	10	B.C. Barr et al., unpub.
Nymphicus hollandicus	Iowa	Bp	N	3	Myers et al. 1983
Eolophus roseicapillus	California	Bp/Bc	N	3	Stringfield and Sedgwick 1997
Ara ararauna	Nebraska	Bp	N	3/4	Armstrong et al. 1989 and unpub.
Ara macao	Nebraska	Bp	N	3/4	Armstrong et al. 1989
	Iowa	Bp	N	2	M.A. Nieves et al., unpub.
Ara ararauna x *A. macao*	Nebraska	Bp	N	2/2	Armstrong et al. 1989
Amazona aestiva aestiva	California	Bp/Bc	N	1	B.C. Barr, unpub.
Amazona ochrocephala oratrix	Indiana	Bp	N	2	A.M. Lennox and K.R. Kazacos, unpub.
Aratinga acuticaudata	Indiana	Bp	N	5	A.M. Lennox and K.R. Kazacos, unpub.
Aratinga canicularis	Indiana	Bp	N	1	A.M. Lennox and K.R. Kazacos, unpub.
Aratinga solstitialis	Indiana	Bp	N	2	A.M. Lennox and K.R. Kazacos, unpub.
O. Strigiformes					
Tyto alba	California	Bp/Bc	N	1	R.H. Evans, unpub.
O. Ciconiiformes					
Nycticorax nycticorax	California	Bp	N	2	R.H. Evans, unpub.
O. Charadriiformes					
Calidris alba	California	Bp/Bc	N	1	R.H. Evans, unpub.
O. Anseriformes					
Anas platyrhynchos	California	Bp/Bc	N	2	R.H. Evans, unpub.
(domesticated)	Indiana	Bp	E	8/21	Wirtz 1982

(*continued*)

TABLE 11.6 (*continued*)

Host	Geographic Location	Parasite[a]	Natural (N) or Experimental (E)	Number Affected[b]	Reference/Source[c]
O. Casuariiformes					
Dromaius novaehollandiae	Indiana	Bc	N	2	Winterfield and Thacker 1978; Kazacos et al. 1982b
	Indiana	Bp	N	2	Kazacos et al. 1991
	Ontario	Bp/			
	Kansas	Bp/Bc	N	2/4	Suedmeyer et al. 1996
	Michigan	Bp	N	1	D.W. Agnew and K.R. Kazacos, unpub.
	Kansas, Missouri, Nebraska, New York	Bp	N	>15	K.R. Kazacos, unpub.
	California	Bp/Bc	N	1	L.W. Woods, unpub.
O. Struthioniformes					
Struthio camelus	Indiana	Bp	N	1	Kazacos et al. 1991
Probable *Baylisascaris* NLM, based on characteristic histopathologic lesions, clinical signs, and history of exposure, but larvae not found in histologic sections:					
O. Rodentia					
Neotoma magister	New York	Bp	N/E	1	McGowan 1993
Sciurus carolinensis	Indiana	Bp	N	2	K.R. Kazacos, unpub.
	Washington	Bp	N	4	Tseng 1997
O. Lagomorpha					
Sylvilagus floridanus	Indiana	Bp/Bc	N	1	K.R. Kazacos, unpub.
Sylvilagus audubonii	California	Bp/Bc	N	2	C.E. Stringfield and C.J. Sedgwick, unpub.
Oryctolagus cuniculus	Indiana	Bp	N	2	K.R. Kazacos, unpub.
O. Primates					
Homo sapiens	Oregon	Bp	N	1	M. Lahr and R.D. Jansen, unpub.; in Cunningham et al. 1994
O. Marsupialia					
Bettongia penicillata	California	Bp/Bc	N	1	J.E. Wynne, unpub.
O. Chiroptera					
Pteropus giganteus	California	Bp/Bc	N	1	Stringfield and Sedgwick 1997
O. Galliformes					
Pavo cristatus	Nebraska	Bp/Bc	N	1	Armstrong et al. 1989
O. Psittaciformes					
Agapornis sp.	California	Bp/Bc	N	1	B.C. Barr, unpub.
O. Casuariiformes					
Dromaius novaehollandiae	Oklahoma	Bp	N	3	Campbell et al. 1997
Probable *Baylisascaris* NLM, based on clinical signs and history of exposure, but unproven because animals survived or were lost to follow-up:					
O. Marsupialia					
Petrogale xanthopus	California	Bp/Bc	N	2	Stringfield and Sedgwick 1997
O. Primates					
Leontopithecus rosalia chrysomelas	California	Bp/Bc	N	2	Stringfield and Sedgwick 1997
O. Psittaciformes					
Calyptorhynchus magnificus	California	Bp/Bc	N	1	Stringfield and Sedgwick 1997
Rhynchopsitta pachyrhyncha	California	Bp/Bc	N	1	Stringfield and Sedgwick 1997

(*continued*)

TABLE 11.6 (*continued*)

Host	Geographic Location	Parasite[a]	Natural (N) or Experimental (E)	Number Affected[b]	Reference/Source[c]
O. Casuariiformes					
Dromaius novaehollandiae	Indiana	Bp	N	2	K.R. Kazacos, unpub.
O. Rheiformes					
Rhea americana	Indiana	Bp	N	2	K.R. Kazacos, unpub.
Baylisascaris larva migrans (VLM, OLM) identified, without recognition of concurrent NLM or clinical CNS disease:					
O. Rodentia					
Mus musculus (white, lab)	Great Britain	Bc	E	2/2	Goodey and Cameron 1923
	Australia	Bc	E	4/5	Sprent 1953a
Peromyscus leucopus	Illinois	Bp/Bc	N	13/67	Tiner 1953a, 1954
	Indiana	Bp/Bc	N	111/487	Page 1998
	Indiana	Bp	N/E	13/46	Page 1998
Sciurus carolinensis	Illinois	Bp	E	2/6	Tiner 1952a
	Indiana	Bp	N	3	K.R. Kazacos, unpub.
Sciurus niger	Illinois	Bp	N	8/12	Tiner 1951, 1953a
Castor canadensis	Kansas	Bp/Bc	N	1	McKown et al. 1995
O. Primates					
Homo sapiens	Kentucky	Bp	N	1	Raymond et al. 1978
	Michigan	Bp/Bc	N	1	Raymond et al. 1978
	Wisconsin	Bp/Bc	N	1	Williams et al. 1988
	California	Bp	N	1	Goldberg et al. 1993
	Germany	Bp	N	1	Küchle et al. 1993
	Massachusetts	Bp/Bc	N	1	Boschetti and Kasznica 1995

[a]Bp/Bc: species could not be determined (Bp or Bc); Bp/Bc: species involved most likely *B. procyonis;* Bm: possible *B. melis.*
[b]na = not available
[c]Unpublished cases have been confirmed by the author, with information and data on file at Purdue University.
[d]Positive for *Baylisascaris* NLM, but without clinical central nervous system disease
[e]Positive for *Baylisascaris* NLM based on serology.

defecation habits, other epidemiologic factors, and larval pathogenicity, their role in this syndrome is considerably less than that of *B. procyonis.* Since the third-stage larvae of these parasites are very difficult or impossible to differentiate, especially in histologic sections, species determination in cases and outbreaks often is based on epidemiologic findings, which indicate exposure to raccoon or skunk feces. In situations where both raccoons and skunks occur and epidemiologic studies are not done, a specific identification beyond *Baylisascaris* sp. cannot be made (although one or the other species might be considered more likely based on relative host abundance). In all but two cases and outbreaks where epidemiologic studies were done, *B. procyonis* was determined to be the parasite involved (Table 11.6), clearly indicating that the raccoon ascarid is the most likely cause of this disease syndrome.

CLINICAL SIGNS. Except in very heavy infections with intestinal obstruction, raccoons infected with *B. procyonis* appear clinically normal with no outward signs of infection. Similarly, other species with *Baylisascaris* larva migrans usually are asymptomatic if no larvae enter the brain. At high infecting dosages, individuals may become dull and anorexic, with dyspnea and increased respiratory rates 2–5 days postinfection, due to hemorrhagic pneumonitis from pulmonary migration (Kazacos et al. 1981b; Donnelly et al. 1989; Kazacos 1997). The severity and progression of central nervous system disease in NLM depends on the number of eggs ingested, the number of larvae entering the brain, the location and extent of migration damage and inflammation in the brain, and the size of the brain. Thus, clinical disease will vary from mild, insidious, slowly progressive central nervous system disease with subtle clinical signs to acute, fulminating, rapidly progressive central nervous system disease with marked clinical signs. Although larvae enter the somatic tissues, eyes, and brain of some species as early as 3 days postinfection, clinical central nervous system disease is not usually apparent before 9–10 days postinfection, and in many cases not until 2–4+ weeks postinfection, due to the lag time in causing central nervous system damage and inflammation (Kazacos 1997; Sheppard and Kazacos 1997). If the larvae leave the brain or

FIG. 11.4—Gray squirrel with encephalitis due to *Baylisascaris procyonis,* showing arching of the head and neck, and extensor rigidity of forelegs. [Reprinted from Kazacos (1983a) with permission of Purdue Research Foundation.]

FIG. 11.5—Woodchuck with encephalitis due to *Baylisascaris procyonis.* This animal was submitted as a rabies suspect because of ataxia, circling, and loss of fear of humans.

become encapsulated, clinical signs can stabilize, and the animal can survive and function with variable central nervous system deficits.

Initial clinical signs in rodents and other small mammals include depression, lethargy, or nervousness; rough hair coat; tremors in the front paws; slight head and/or body tilts; and circling or jumping when disturbed. These signs progress to various combinations of severe head and/or body tilts, ataxia, continuous circling, leaning, falling over, opisthotonos, lateral recumbency, rolling around the longitudinal axis, coma, and death (Kazacos and Boyce 1989; Sheppard and Kazacos 1997). Other clinical signs include “stargazing,” slow arching of the head and neck, blindness, nystagmus, various degrees of motor weakness or posterior paresis, hypotonia or extensor rigidity, and paddling movements while recumbent (Figs. 11.4, 11.5) (Kazacos and Boyce 1989; Sheppard and Kazacos 1997; references in Table 11.6).

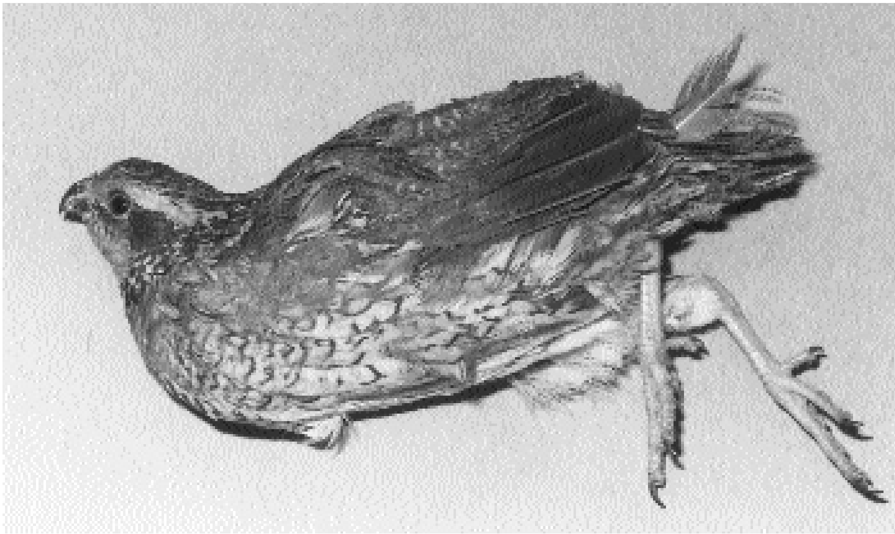

FIG. 11.6—Northern bobwhite with encephalitis due to *Baylisascaris procyonis,* from an outbreak linked to pet raccoons (Reed et al. 1981). Note lateral recumbency, torticollis, and extensor rigidity.

Monkeys experimentally infected with *B. procyonis* became less vocal, their activity declined, and they began to have problems with manual dexterity, with increasing difficulty grasping and handling food, climbing, and traversing the cage floor. These signs progressed rapidly to marked ataxia, loss of balance, inability to maintain an upright posture or to grasp food, torticollis, truncal ataxia, swaying and bobbing of the head, intention tremors of the head and forelimbs, and head pressing. Two monkeys became narcoleptic and a third had nondirected, unsolicited vocalizations. Finally, they became semicomatose and recumbent on the cage floor, with opisthotonos, extensor rigidity, and nystagmus, and were unresponsive to touching or prodding (Kazacos et al. 1981b). Many of these clinical signs and a similar progression were seen in marmosets, lemurs, a gibbon, and human infants infected with *Baylisascaris* (Huff et al. 1984; Fox et al. 1985; Huntress and Spraker 1985; Kazacos 1996, 2000; Campbell et al. 1997; Ball et al. 1998). Mildly affected primates suffered only subtle forelimb tremors (C.L. Eng and K.R. Kazacos, unpublished) or had slight head tilt and ataxia (Pessier et al. 1997).

Clinical signs in birds include ruffled feathers, disorientation, head tremors, torticollis, poor grip reflexes, incoordination, loss of balance, walking in circles, falling, rolling, inability to fly or loss of flight control, blindness, recumbency, extensor rigidity, and paralysis of one or both wings or legs (Fig. 11.6) (Richardson et al. 1980; Reed et al. 1981; Coates et al. 1995; references in Table 11.6). Clinically affected ratites exhibit varying degrees of incoordination, loss of equilibrium and balance, muscle weakness, wobbling, and progressive ataxia. They typically stagger, walk in circles, assume a wide, splay-legged stance with their head extended downward for balance, and walk rapidly backward, stumbling and falling; eventually they are unable to stand or walk and become increasingly emaciated (Kazacos et al. 1991; Kwiecien et al. 1993; Suedmeyer et al. 1996).

PATHOLOGY AND PATHOGENESIS. Somatic migration of *Baylisascaris* larvae causes mechanical damage and tissue necrosis and provokes vigorous inflammatory reactions. The principal pathologic alterations are those of eosinophilic and granulomatous inflammation, which may occur in a variety of organs and tissues, including the liver, lungs, heart, brain, and eyes. The most important lesions are in the brain and consist of focal to diffuse meningoencephalitis, necrosis, and spongiosis. Inflammatory reactions are directed against larval excretory-secretory antigens, which consist of enzymes, cuticular proteins, and metabolic wastes released by the larvae during migration. Eosinophils are a major component of host reactions, and toxic eosinophil proteins released in the tissues probably contribute to pathologic changes and clinical signs (Hamann et al. 1989; Kazacos and Boyce 1989; Kazacos 1996, 1997; C.L. Moertel et al., unpublished). A key factor in the pathogenesis of *Baylisascaris* NLM is the large size attained by the larvae, which when combined with their aggressive migration, results in considerable damage to the central nervous system. *Baylisascaris procyonis* L_2's are ~300 μm long (275–310 μm) when they hatch from ingested eggs (Berry 1985; Sakla et al. 1989). The larvae grow rapidly following infection, reaching a length of 1058 μm

(range, 625–1429 μm) at 10 days postinfection, 1573 μm (range, 1319–1888 μm) at 15 days postinfection, and 1750 μm (range, 1450–1850 μm) at 31 days postinfection (Tiner 1953b; Goldberg et al. 1993). Most *B. procyonis* larvae recovered from clinical cases are 1500–1900 μm long and 60–80 μm in greatest width (Kazacos 1997).

Early migration of *Baylisascaris* larvae through the liver and lungs and subsequent migration in other organs and tissues produce traumatic damage and inflammation. Rabbits naturally infected with *B. procyonis* had acute hemorrhagic tracks in the liver associated with focal necrosis and hepatitis. Also noted were multifocal eosinophilic myocarditis and myositis, with associated myofiber loss and fibrosis, interstitial pneumonitis, and focal nephritis. Inflammatory infiltrates consisted of eosinophils, macrophages, lymphocytes, and plasma cells (Kazacos et al. 1983). Similar lesions and others were seen in monkeys experimentally infected with *B. procyonis* (Kazacos et al. 1981b). The severe hepatic pathology associated with liver trapping of larvae in *Toxocara* infections has not been seen with *Baylisascaris,* reflecting differences in migration, antigenicity, and/or host responses between the two parasites (Kazacos 1997). *Baylisascaris* larvae migrate quickly through the liver and lungs, and the liver is not a major site of larval accumulation, before or after somatic migration.

Pulmonary migration of *Baylisascaris* can produce considerable lung damage, and in heavy infections clinical signs of verminous pneumonitis are noted within 2–5 days postinfection (Goodey and Cameron 1923; Sprent 1952a, 1953b, 1955; Kazacos et al. 1981b; Donnelly et al. 1989). Petechial and ecchymotic hemorrhages are evident in the lungs within 12–48 hours postinfection (Fig. 11.7), and within another day or two the lungs are often uniformly dark red. The hemorrhage subsides and is replaced by acute neutrophilic then eosinophilic inflammation (Kazacos 1997). Using bronchopulmonary lavage cytology to assess pulmonary migration of *B. procyonis* in mice, Wyand-Ouellette et al. (1983) saw a marked increase in erythrocytes on day 1, peaking on days 2–3 at up to 130X controls, and returning to normal by days 7–8. This was followed by a biphasic nucleated cell (leukocyte) response, which peaked on days 3 and 8, at 7–9X control mice; the early peak was due to neutrophils (210X control) and macrophages (3X control) and the latter peak to eosinophils (209X control) and macrophages (4X control). In affected lungs, alveolar septa are thickened by congestion, edema, and cellular infiltrates, and hemosiderosis develops in areas of resolving hemorrhage.

Following migration in extraneural tissues, larvae become encapsulated in well-circumscribed eosinophilic granulomas, usually 1–2 mm in diameter (Figs. 11.8, 11.9). Larval granulomas are often visible grossly (Fig. 11.8) and are found in many organs and tissues, including the liver, lungs, heart, diaphragm, pancreas, spleen, kidneys, mesentery, mesenteric lymph nodes, intestinal wall, skeletal muscles, brain, and eyes (Sprent 1952a; Kazacos et al. 1981b; Kazacos 1996, 1997). The development and distribution of *B. procyonis* larval granulomas varies with host species, which is an important consideration during necropsy examinations for this infection (Sheppard and Kazacos 1997). Tiner (1953a) found that gray squirrels had numerous larval granulomas primarily in the thorax, in the wall of the caval veins, heart, lungs, diaphragm, and intercostal muscles; the author has also noted this distribution in infected squirrels (K.R. Kazacos, unpublished). Naturally infected rabbits (*O. cuniculus*) had granulomas in the heart, lungs, diaphragm, liver, mesentery, intestine, and skeletal muscles (Kazacos et al. 1983; K.R. Kazacos, unpublished). In experimentally infected rats, most granulomas were in the wall of the intestine, with some in the heart and diaphragm (Wirtz 1982). Naturally infected woodchucks, lemurs, and marmosets (with *B. columnaris*) had numerous larval granulomas along the intestinal tract, in addition to other locations (Richter and Kradel 1964; Jacobson et al. 1976; Fleming and Caslick 1978; Huntress and Spraker 1985; K.R. Kazacos and P.L. Wolff, unpublished; K.R. Kazacos, unpublished). In children, granulomas were numerous in the heart, lungs, and mesentery; and in experimentally infected monkeys, they were abundant in these tissues as well as the anterior somatic musculature, diaphragm, and tissues of the head and neck (Kazacos et al. 1981b; Huff et al. 1984; Fox et al. 1985). Different species may react differently to *Baylisascaris* larvae, and host reactions may also be affected by prior infection. For example, previously unexposed *Mus musculus* showed a strong tendency to develop large granulomas, which were prominent in the heart (Fig. 11.8), diaphragm, body wall, and ante-

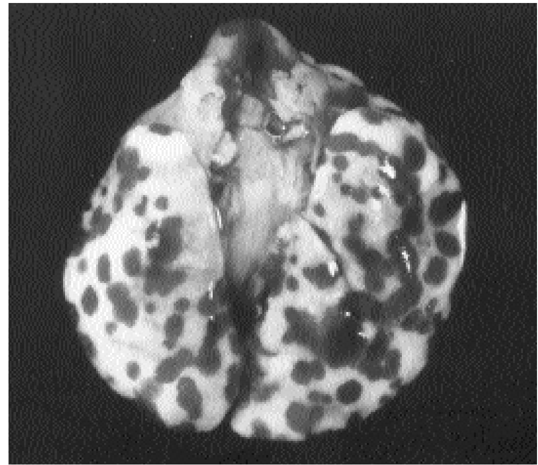

FIG. 11.7—Multifocal, coalescing hemorrhages in the lungs of a lab mouse, due to pulmonary migration of *Baylisascaris columnaris* larvae; 2 days postinfection with eggs.

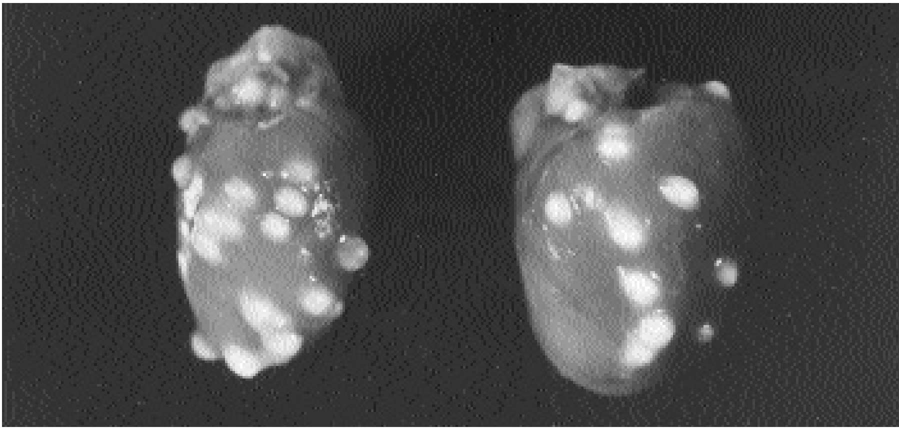

FIG. 11.8—Larval granulomas in the hearts of lab mice infected with *Baylisascaris columnaris;* 15 days postinfection. Similar granulomas develop in *B. procyonis* infections.

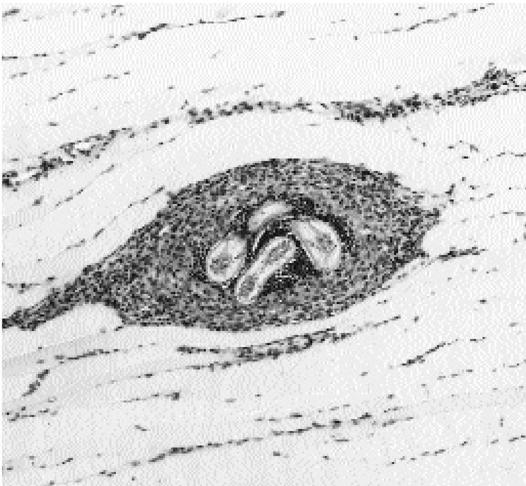

FIG. 11.9—*Baylisascaris procyonis* larval granuloma in skeletal muscle of a hamster. The larva is in a central pool of eosinophils. [Reprinted from Kazacos (1983a) with permission of Purdue Research Foundation.]

rior body musculature, but not along the gastrointestinal tract. In *P. leucopus,* granulomas were few, very small, rarely noted grossly, and found primarily in the heart and along the gastrointestinal tract. The large granulomas in *M. musculus* were characterized by a marked inflammatory reaction and contained significantly more eosinophils, whereas those in *P. leucopus* lacked an intense response and consisted primarily of macrophages (Sheppard and Kazacos 1997).

Unlike mammals, avian species infected with *Baylisascaris* typically lack gross lesions at necropsy, and histopathologic alterations are usually confined to the brain (Kazacos and Boyce 1989). Wirtz (1982) saw no gross larval granulomas in experimentally infected chickens and ducks and found only a single granuloma histologically, in an extrinsic ocular muscle. Solitary larval granulomas also were noted histologically in the lungs of a naturally infected brush turkey and bobwhite (Kazacos et al. 1982a; Williams et al. 1997). The comparative lack of gross lesions and paucity of larval granulomas in avian species with *Baylisascaris* NLM has been documented in various natural cases and outbreaks (Richardson et al. 1980; Reed et al. 1981; Myers et al. 1983; Evans and Tangredi 1985; Armstrong et al. 1989; Kazacos et al. 1991; Kwiecien et al. 1993).

In most clinical cases involving birds, including large ratites, few or no larvae are found in visceral or somatic tissues, even though 1–3 larvae are found in the brain. Thus, most avian cases appear to involve low-level infections, with a higher probability of larval migration to the brain; however, much higher infection levels are occasionally documented. For example, in a recent outbreak involving massive infection in a mixed collection of parrots and conures (Table 11.6), 17–150 (mean, 87) *B. procyonis* larvae were recovered from the brains and 47–285 (mean, 173) larvae from the viscera and carcasses of six birds dying from acute, severe central nervous system disease (A.M. Lennox and K.R. Kazacos, unpublished); interestingly, no gross lesions or larval granulomas were noted.

The most important pathological alterations due to *Baylisascaris* are in the brain. Gross lesions include hemorrhagic foci and tracks, congestion, swelling and softening, and cerebellar herniation. The leptomeninges may be congested and/or thickened and discolored (Sprent 1955; Kazacos et al. 1981b; Huff et al. 1984; Fox et al. 1985; Kazacos 1997). Early histopathologic lesions consist of focal migration tracks, with tissue disruption, hemorrhage, necrosis, and spongiosis, with a few inflammatory cells

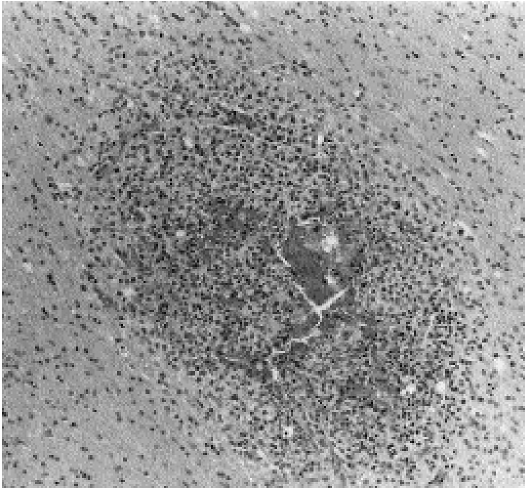

FIG. 11.10—Hemorrhagic migration track in the cerebral white matter of a squirrel monkey with *Baylisascaris procyonis* encephalitis, showing microcavitation, hemorrhage, necrosis, and influx of macrophages and other leukocytes. [Reprinted from Kazacos et al. (1981b) with permission of *JAVMA.*]

(Kazacos 1997). Migration tracks may be found in any portion of the brain and spinal cord, but are especially important in critical areas such as the cerebellum, midbrain, and medulla. Migration tracks quickly become infiltrated with inflammatory cells, primarily macrophages in the early stages, followed by eosinophils, lymphocytes, and plasma cells (Fig. 11.10). As the reaction progresses, perivascular cuffing becomes prominent, especially adjacent to areas of inflammation in the neuropil, and there is more extensive spongiosis and cavitation (Figs. 11.11–11.13). Depending on the numbers of larvae present, central nervous system lesions may be focal or diffuse and may extend to the leptomeninges. Lesions in heavy infections are typically more extensive and severe. Other central nervous system lesions include neuronal degeneration, swelling and degeneration of axons, demyelination, and prominent gliosis (Kazacos 1997). Larvae may be seen within migration tracks and areas of inflammation and necrosis (Fig. 11.14); however, they are also commonly found in normal-appearing neuropil away from obvious lesions, indicating their active migration in the central nervous system at the time of fixation. Encapsulation of larvae in the central nervous system lags far behind encapsulation in other tissues, resulting in prolonged larval migration in this site. If the animal survives long enough, however, encapsulation eventually occurs, and larvae become walled off in sharply demarcated foci of granulomatous inflammation. These foci resemble the granulomas in other tissues but are usually walled off by gliosis rather than fibrosis (Kazacos 1997). Eosinophilic deposits

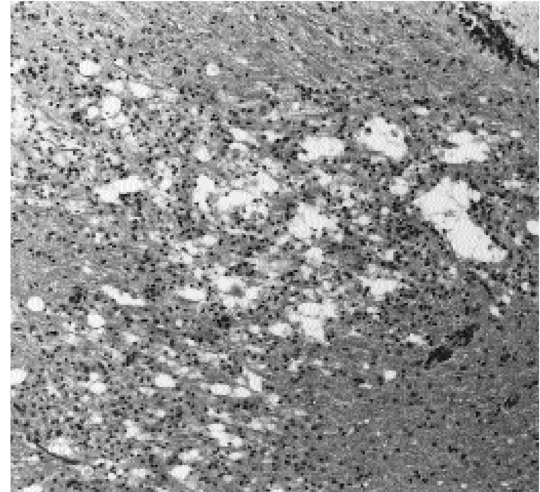

FIG. 11.11—Cerebellar peduncle of a rabbit with *Baylisascaris procyonis* encephalitis, showing extensive necrosis and inflammation. [Reprinted from Kazacos et al. (1983) with permission of *JAVMA.*]

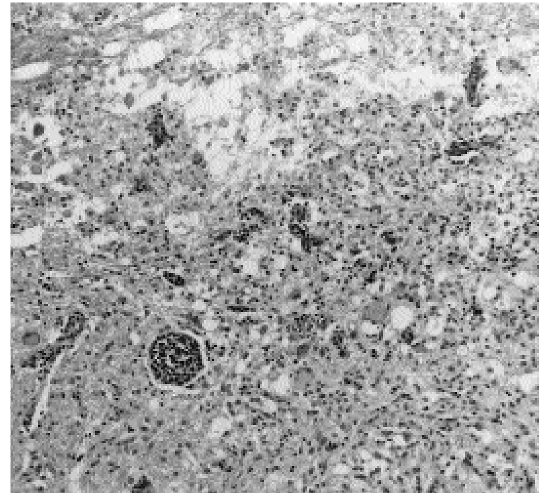

FIG. 11.12—Cerebrum of a rabbit with *Baylisascaris procyonis* encephalitis, showing extensive spongiosis, microcavitation, necrosis, swollen and degenerating axons, infiltration by inflammatory cells, and perivascular cuffing.

[Splendore-Hoeppli (SH) substance] may be prominent in areas of necrotic eosinophils, in migration tracks, and within granulomas adjacent to larvae. Immunofluorescence studies of fatal *Baylisascaris* infections in children indicated that these SH deposits consist of extracellular eosinophil major basic protein, originating from extensive eosinophil degranulation in the tissues (Hamann et al. 1989). Recently, two children with

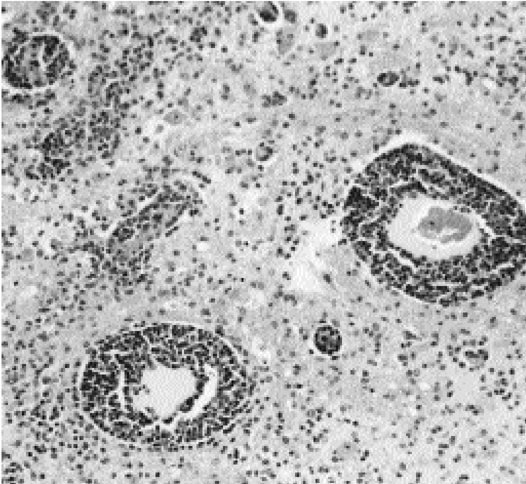

FIG. 11.13—Cerebrum of a ferret with *Baylisascaris procyonis* encephalitis, showing prominent perivascular cuffing with leukocytes.

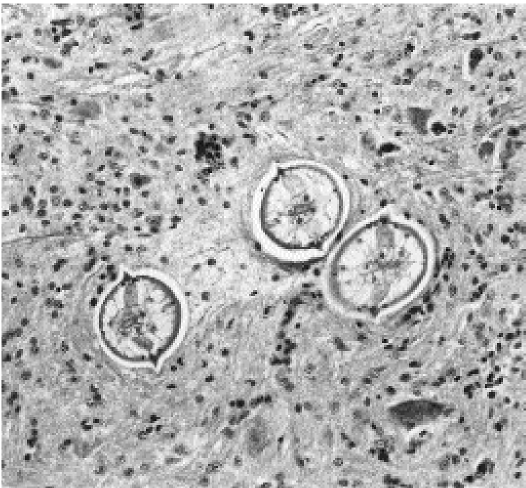

FIG. 11.14—Cross sections of *Baylisascaris procyonis* larva migrating in the cerebellar peduncle of a chicken with encephalitis (Richardson et al. 1980).

severe *Baylisascaris* NLM were found to have measurable levels of interleukin 5, eosinophil-derived neurotoxin, and major basic protein in their cerbrospinal fluid (C.L. Moertel et al., unpublished).

Different species of *Baylisascaris* vary in their central nervous system pathogenicity, based on differences in somatic migration and invasion of the brain, larval aggressiveness in the central nervous system, and the ability of the host to wall them off. *Baylisascaris procyonis* and *B. melis* are the most pathogenic, followed by *B. columnaris* and the others (Kazacos and Boyce 1989). *Baylisascaris procyonis* is clearly more pathogenic than *B. columnaris,* requiring fewer infective eggs ingested and fewer larvae in the brain to cause the same or worse clinical disease. A single *B. procyonis* larva in the brain of a mouse is usually fatal, whereas five to six or more *B. columnaris* larvae in the brain aren't necessarily fatal, even when central nervous system signs are present (Sprent 1952a, 1955; Tiner 1953a,b; Clark et al. 1969; Sheppard and Kazacos 1997). Clinical signs appear much earlier and progress much more quickly in mice infected with *B. procyonis* than with *B. columnaris.* In addition, *B. columnaris* larvae in the brain have a greater tendency to settle down and become encapsulated than do larvae of *B. procyonis* (Tiner 1953a,b; Sprent 1955; Clark et al. 1969). The author has confirmed these differences on several occasions, indicating the much greater pathogenicity of *B. procyonis* over *B. columnaris.* However, at sufficient dosages, *B. columnaris* has the ability to produce clinically significant NLM (Table 11.6) and should be treated with the same precautions as *B. procyonis* and *B. melis.*

In mice concurrently infected with eastern encephalitis or Colorado tick fever virus, migrating *B. columnaris* larvae induced mortality at rates considerably higher than those produced by virus or larvae alone or the sum of mortalities of each agent alone (Clark et al. 1969). The authors postulated that when sufficient migrating larvae breached the blood-brain barrier, it allowed increased amounts of virus to spill into susceptible neural tissue. Whether this pathogenic interaction occurs between *Baylisascaris* larvae and these or other neurotropic agents in natural populations is unknown.

Ocular larva migrans was seen in mice, hamsters, gray squirrels, woodchucks, and two species of monkeys experimentally infected with *B. procyonis* (Kazacos et al. 1984b, 1985); in rabbits and woodchucks experimentally infected with *B. columnaris* (Swerczek and Helmboldt 1970; Church et al. 1975); and in psittacines with naturally occurring *B. procyonis* infections (A.M. Lennox and K.R. Kazacos, unpublished). Ocular larva migrans due to *Baylisascaris* is an important disease in humans, in which *B. procyonis* has been identified as the primary cause of the large nematode variant of diffuse unilateral subacute neuroretinitis (DUSN) (Gass and Braunstein 1983; Kazacos et al. 1984a,b, 1985; Goldberg et al. 1993). Ocular disease due to *Baylisascaris* is manifested as unilateral loss of vision, usually seen in patients with no symptoms of visceral or neural larva migrans; this reflects the lower numbers of larvae typically present in patients with OLM and chance migration of a single larva into the eye (Kazacos 1997). In heavy primary infections, OLM may be seen in conjunction with NLM (Rowely et al. 2000; Park et al., 2000). In experimentally infected monkeys, *B. procyonis* larvae reached the eye as early as 7 days postinfection (Kazacos et al. 1984b, 1985). Larvae produce migration tracks in the retina and stimulate inflammatory reactions primarily involving the

retina, choroid, and vitreous. Early histopathologic lesions consist of retinal disruption, nuclear pyknosis, and hyperplasia and migration of the pigment epithelium. These progress to intense eosinophilic retinitis, with necrosis, vasculitis, vitritis, choroiditis, and abundant eosinophilic hyaline material (SH substance). In addition to producing these migration-related lesions, *Baylisascaris* larvae become walled off in granulomatous masses in the retina and choroid (Kazacos et al. 1984b, 1985; Kazacos 1997).

DIAGNOSIS. *Baylisascaris* infections in raccoons are diagnosed by finding ascarids in the small intestine at necropsy, or passed in the feces spontaneously or following anthelmintic treatment. Patent infections are diagnosed by finding characteristic *Baylisascaris* eggs in the feces. Differentiation of adults of several *Baylisascaris* species, including *B. procyonis* and *B. columnaris,* is difficult or impossible because of similarities and overlap of morphological characters. Although various morphological features have been described for separating *B. procyonis* from *B. columnaris,* including the shape of lip denticles, male pericloacal rough areas, and male tail (Tiner 1952a,b; Hartwich 1962; Sprent 1968), Berry (1985) concluded that these and other characters were variable enough that the two species could not be distinguished morphologically.

Distinct biological differences exist in the migration, development, and pathogenicity of *B. procyonis* and *B. columnaris* larvae in intermediate hosts (Sprent 1952a, 1955; Tiner 1952a, 1953a,b; Berry 1985; Sheppard and Kazacos 1997). In addition, Berry (1985) described morphological differences between the third-stage larvae of each parasite at 10 days, as well as electrophoretic differences in alleles for 6-phosphogluconate dehydrogenase. These data indicate that these parasites are closely related but distinct species. Although limited experimental cross-transmission of *B. procyonis* and *B. columnaris* between raccoons and skunks was possible, Berry (1985) found no evidence that cross-infection or hybridization by or of these parasites occurs in nature. Based on the apparent genetic isolation of these two species, in geographic areas where both parasites occur, the most useful criterion for separating them appears to be host (procyonid versus mephitid). The application of molecular genetic techniques should help answer the question of relatedness among these and other *Baylisascaris* species (Nadler 1992; Nadler and Hudspeth 2000).

Diagnosis of *Baylisascaris* larva migrans is based on clinical signs, a history of exposure, antemortem labratory findings (including serology, cytology, imagery, and examination of cerebrospinal fluids), postmortem gross and histopathologic lesions, and recovery and/or identification of larvae at necropsy, in biopsies, or in or from affected eyes (Huff et al. 1984; Kazacos et al. 1984a,b, 1985; Fox et al. 1985; Kazacos 1991, 1996, 1997, 2000; Goldberg et al. 1993). Of these, only identification of larvae in/from the tissues is confirmatory, although positive serology would be indicative of infection.

Prior to the mid-1980s, when *Baylisascaris* NLM was becoming widely recognized and better considered and sought by diagnosticians, many cases of NLM were misdiagnosed clinically. Most individuals exhibiting abnormal behavior were submitted as rabies suspects (Fig. 11.5) (Richter and Kradel 1964; Fleming and Caslick 1978; Kazacos et al. 1981a; Roth et al. 1982), despite the fact that the species involved (woodchucks, squirrels, rabbits, birds) were less likely candidates for rabies. The diagnostic focus on rabies in these cases masked the widespread occurrence of *Baylisascaris* NLM, because typically the entire head or brain was sent for rabies diagnosis, leaving no central nervous system tissues for examination for other possible causes. With the widespread occurrence of *Baylisascaris* NLM documented by our laboratory and others, this situation has changed, so that *Baylisascaris* NLM is now at or near the top of the differential list for central nervous system disease in these and related species. Of course, other possible causes of central nervous system disease, including rabies, must still be considered and appropriate samples taken, along with appropriate specimens for *Baylisascaris* NLM. Although clinical signs of NLM are nonspecific, in common target species they are still highly suggestive, especially in conjunction with compatible diagnostic findings and history.

Other possible causes of central nervous system disease in these animals would include protozoal (e.g., toxoplasmosis, sarcocystosis, free-living amebae), bacterial, or viral encephalitis, fungal infections, other migratory helminth larvae (e.g., *Alaria* mesocercariae, spargana, gnathostomes, filariids), helminth egg–induced encephalitis (e.g., *Dendritobilharzia* in waterfowl), aberrant migration of dipteran larvae (*Cuterebra* and others), as well as pesticide toxicoses and trauma. Causes of eosinophilic meningitis and meningoencephalitis are more limited and include migratory helminths, certain fungal agents, and some neoplasms and other noninfectious causes (Kuberski 1979; Weller and Liu 1993; Connor et al. 1997). Causes of eosinophilia are more diverse and include various other helminth infections, certain deep mycoses and bacterial infections, various allergies and hypersensitivities, myeloproliferative and neoplastic diseases, and some other conditions (Weller 1992). Multisystem granulomas also characterize some other diseases, including certain bacterial and deep mycotic infections (Connor et al. 1997; Kazacos 1997).

An important clinical and diagnostic finding in *Baylisascaris* NLM is eosinophilic pleocytosis of the cerebrospinal fluid, particularly in animals or humans with concurrent peripheral eosinophilia and progressive central nervous system disease. Eosinophil numbers appear to be higher in the cerebrospinal fluid during acute disease, and their levels would be related to the degree of central nervous system damage and

eosinophilic inflammation caused by migrating larvae. Since some other migratory helminths and pathologic conditions could cause eosinophilic pleocytosis, this finding is most useful in patients with typical clinical signs and a history and/or evidence of exposure. Antemortem imaging techniques, including computed tomography (CT) and magnetic resonance imaging (MRI), may yield important information on the location, severity, and progression of central nervous system lesions (Huff et al. 1984; Fox et al. 1985; Cunningham et al. 1994; Ball et al. 1998; Rowley et al. 2000). For example, MRI revealed diffuse white matter disease with deep periventricular involvement in children with severe *Baylisascaris* NLM (Rowley et al. 2000; Park et al., 2000), and a large, focal lesion in the frontal cortex of a gibbon with NLM (Ball et al. 1998). Important findings in *Baylisascaris* OLM/ DUSN include compatible lesions on ophthalmoscopy, eosinophils on intraocular cytology, positive serum and/or intraocular antibody levels, and intraocular larvae with characteristics of *B. procyonis* (Kazacos 1991, 1997; Goldberg et al. 1993).

Serologic methods, including indirect immunofluorescence, ELISA, and Western blotting, have been developed for *Baylisascaris* and applied primarily to human cases (Huff et al. 1984; Fox et al. 1985; Goldberg et al. 1993; Cunningham et al. 1994; Rowley et al. 2000; K.R. Kazacos, unpublished). Immunofluorescence assays use frozen sections of *B. procyonis* third-stage larvae, whereas ELISA and Western blotting use excretory-secretory antigens produced by larvae maintained in vitro (Boyce et al. 1988a,b, 1989). Children strongly positive for *Baylisascaris* antibodies in serum and cerebrospinal fluid were negative for *Toxocara* by ELISA, indicating that the two infections can be distinguished serologically. It was also possible to detect antibodies to *Baylisascaris* in experimentally and naturally infected rabbits, mice, and monkeys by Western blotting, but other than these studies, serologic testing has not been applied to nonhuman species. Development of similar serologic methods for other species is possible but would require animal species-specific reagents. The serologic diagnosis of *Baylisascaris* was possible only to genus (Boyce et al. 1988a,b, 1989). Thus, identification of the particular species of *Baylisascaris* involved in cases or outbreaks is best determined epidemiologically through an assessment of probable exposure.

Histopathologic lesions of *Baylisascaris* migration in the brain (i.e., eosinophilic meningoencephalitis, necrosis, spongiosis, cavitation) are characteristic and highly suggestive of this infection, particularly in typical target species. However, finding or isolating the larvae is important for confirmation. Routine histopathology may fail to detect larvae, particularly in low-level infections. In such cases, the likelihood of finding larvae is increased if numerous blocks and slides are examined and if suspect lesions are step or serially sectioned. The sensitivity of histopathology for detecting larvae in the central nervous system is increased in heavier infections; in two human cases larvae were identified in brain biopsies (Rowley et al. 2000; W.A. Kennedy et al., unpublished). In most cases, the majority of larvae occur in extraneural tissues, where they produce migration-related lesions and granulomas, which should be correlated with central nervous system findings. It is very important to thoroughly examine other organs and tissues for larval granulomas from which *Baylisascaris* larvae can be isolated by dissection or digestion, or identified using histopathology. Particular attention should be paid to the heart, lungs, and associated vessels, anterior somatic musculature, and the gastrointestinal tract, especially the mesentery and wall of the small and large intestines. For example, in a recent case involving a black-and-white ruffed lemur with central nervous system disease, the author isolated 2 larvae from the brain, 2 from the skeletal muscles of the head, 17 from the heart and lungs, and 118 from the intestinal wall. If characteristic lesions without larvae are seen in the brain, identification of larvae in other tissues gives strong support to *Baylisascaris* as a probable cause of the central nervous system lesions and clinical disease.

Considering the drawbacks of histopathology, larval isolation methods are more efficient and useful for detecting *Baylisascaris* larvae in the brain and other tissues, particularly in low-level infections. The best methods for isolating *Baylisascaris* larvae from the brain of affected hosts are brain squash, artificial digestion, and the Baermann technique. Of these, we usually use brain squash or digestion because there is less chance of missing or losing the larvae, especially in low-level infections. The Baermann technique will usually work but has some inherent problems, as described below. Digestion methods are also very useful for isolating larvae from other organs and tissues, even when granulomas are not readily apparent. Since other diseases must also be considered and sought in clinical cases, a good approach is to combine histopathology with one of these other methods. For example, several slices of brain may be taken for histopathology and the remainder processed for larvae; right and left halves of the brain may be examined by the respective methods; or, if sufficient numbers of affected animals are available, the whole brain of representative animals may be processed for larvae. These decisions are made by the diagnostician based on the particular situation and the differential list for the particular species. Each of the larval isolation methods can be performed with conventional laboratory equipment and is described in detail below. The brain is first removed with scissors or using a Stryker or hacksaw. The spinal cord of rodents and small birds is easily removed by ejection, using water pressure (Meikle and Martin 1981; Sheppard and Kazacos 1997), while that of larger animals is removed by laminectomy, using a Stryker saw, bone rongeurs, or other means.

Brain Squash. The brain or spinal cord to be examined is first rinsed with saline or water by squirt bottle

to remove any bone chips that may be present. This is done over a Petri dish or beaker that is also examined for larvae. Approximately 1-g pieces of brain or spinal cord are placed on round glass plates (12.7 mm diameter) and minced with fine forceps, second (top) plates are added, and the tissue is flattened to the periphery using steady hand pressure. We often separate the brain into right and left cerebral hemispheres, cerebellum, and midbrain-medulla and process each separately. The brain of a mouse or small bird or half the brain of a slightly larger animal can be processed on one to three plates. If small pieces of bone are present, they will prevent the tissue from being squashed and must first be removed by separating the plates. The squashed brain is examined using a dissecting microscope with bottom-transmitted light and 10–15X magnification. A fiberoptic light source works best, since it will transmit light through the brain tissue better than a standard light. When the light is adjusted properly, the larvae will refract the light and appear bright in the brain tissue, making them easier to find. It is important to examine the plate systematically and thoroughly, usually in two directions. This is aided by dividing the plate into numbered quadrants using a fine marker (Sharpie, Vis-a-Vis). When larvae are found, they are circled with the marker. Areas of inflammatory cell infiltration can also be seen, as focal, irregular, dark grainy areas, best noted in the clearer neuropil. When the exam is complete, any larvae present are isolated by carefully separating the plates, scraping and rinsing the tissue from the positive quadrant or half (both plates) into a Petri dish (100 x 20 mm) of saline, further mixing and macerating it with forceps, and examining it using an inverted or dissecting microscope until the larvae are found. An inverted microscope with a scanning objective (2.5–3.5X) works best since one can see "under" the debris. The dish is gently swirled in a circular motion to bring any larvae present into the center for easier detection. Larvae are removed with a Pasteur pipette to a small dish of saline and either placed on microscope slides for immediate identification or fixed in hot (65°C–70° C) fixative for later identification and storage. Appropriate fixatives include hot AFA (alcohol:formalin:acetic acid), 70% ethanol, or 5% or 10% formalin in saline, with long-term storage in glycerin-alcohol (9:1, 70% ethanol:glycerin). Fixatives are best heated using two beakers set up as a double boiler, on a hot plate on low heat, with the center (fixative) beaker capped with aluminum foil. We usually remove the larvae to a glass vial, draw off most of the saline under a dissecting microscope, then fill the vial with hot fixative. If necessary, larvae for microscopic examination can be cleared with phenol-alcohol or glycerin.

The advantage of this method is that it is nearly foolproof, assuming the squash is done properly and examined well; even if only a single larva is present, it will be found. The most common errors are using too much tissue, so that the squash isn't thin enough, and not doing a systematic, thorough search. Very dark areas of brain are harder to see through without a bright light and demand greater attention and a thin squash. The larvae can be lost in trying to retrieve them from the plates; however, since all the material is present, it can be comminuted and reexamined until they are found or processed further by digestion (see below). The main disadvantage of this method is that it is tedious and time-consuming when used on large brains (> 8–10 g), because many 1-g squashes must be examined. In such cases, we usually process the brain by digestion.

Artificial Digestion. The brain or other tissue is weighed, cut into pieces with scissors, then either pulverized in a small (100 ml) beaker (brain), finely minced with scissors, or comminuted in a Waring blender in warm (37° C) artificial digestive fluid (1% pepsin- 1% HCl-0.85% saline). Brain samples are thoroughly pulverized using a metal spatula (75 x 17 mm) to mash the brain pieces against the side of the beaker; other tissues are finely minced with scissors. Warm digestive fluid is then used to rinse the brain or other tissue into a flask for digestion. If a blender is used, the cut-up tissue is blended in warm digestive fluid. Prior to examination of the gastrointestinal tract, it is first opened along its length and all ingesta is removed and washed off to remove undigestible material and any confounding intestinal nematodes. We recommend using small glass blender jars (Eberbach #8470, 450 ml) with screw lids, filled about one-third with fluid. We also recommend using 3–4-second pulses until the tissue is comminuted to small bits. After processing, the samples are poured and rinsed into Erlenmeyer flasks at a final concentration of 20 ml digestive fluid per gram of tissue. The flasks are placed in a shaking incubator or oscillatory shaker at 37° C and 180–200 rpm or on a magnetic stirrer on low speed 2–2.5 hours. The digests are then coarsely filtered and rinsed through a single layer of cheesecloth into tall conical beer glasses (350–400 ml) (preferred) or graduated cylinders (250–500 ml), which are then filled with cold saline or water and allowed to stand 20–30 minutes for larvae to settle. If the digest is too large for two or three glasses, it is first sedimented in the flask (propped at an angle in a wire basket), drawn down to 400–600 ml by vacuum aspiration, and filtered into the glasses. Cold saline or tap water is used for all rinsing and filling, as it aids in flotation of lipids and fine debris. After sedimentation in the glasses, the surface ("floating") layer and two-thirds of the supernatant are removed by vacuum aspiration, and the glasses are refilled with cold saline or tap water and allowed to sediment again. This process may be repeated several times. The sediment is agitated and suspended in the remaining liquid, poured and rinsed into a Petri dish, and examined for larvae using a dissecting or inverted microscope. The dish is gently swirled to bring any larvae into the center. Larvae are removed and fixed as described previously.

This method is easy to perform and is very effective in recovering *Baylisascaris* larvae from different tissues (Fig. 11.15). Brain does not digest well, so it is

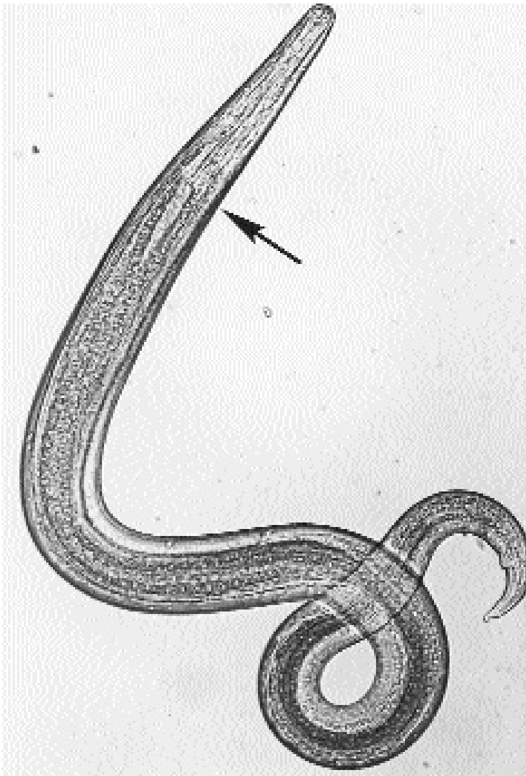

FIG. 11.15—*Baylisascaris procyonis* larva recovered by artificial digestion from the carcass of a conure with encephalitis. Note overall shape and proportions, smoothly rounded anterior end, sharply flexed tail tip, length of esophagus (arrow at esophageal-intestinal junction), prominent intestine, and transverse cuticular striations (seen at bend in midbody).

important to pulverize it thoroughly before digestion. Stock 1% HCl-0.85% saline can be stored in an incubator, and pepsin added (1 g/100 ml, on magnetic stirrer) just prior to use. Care should be taken to avoid blending the tissue for too long, or the larvae may be damaged. However, even if larvae are damaged (e.g., cut in half), the pieces are often recovered later. Digestion for 2 hours is usually sufficient, and overdigestion (> 3 hours) should be avoided, as larvae may be killed and digested. For sedimentation and later processing of larvae in Petri dishes, saline is preferred over water, which will eventually cause osmotic damage.

Baermann Technique. A Baermann funnel is a small- to medium-sized (4–5-inch diameter) glass funnel with a piece of tubing and a clamp on the stem; a piece of wire mesh is cut to fit the top third of the funnel and is used to support a double thickness piece of cheesecloth and the sample during processing. The Baermann funnel can be used on pulverized brain, finely minced lung, or other tissue. Just prior to loading the sample, the funnel is filled with warm (37° C) saline (preferred) or water to a level that will cover the sample, mesh, and cheesecloth. The funnel can also be filled with artificial digestive fluid (see above) as a combined method. The pulverized brain or minced tissue is scooped onto the cheesecloth, which is folded into the funnel to prevent wicking of liquid over the side. The funnel is allowed to stand undisturbed several hours or overnight (if overnight, use saline), preferably in a 37° C incubator. Stimulated by the warmth, the larvae migrate out of the tissue and down through the cheesecloth and mesh, then gravitate into the stem of the funnel. Later, the clamp is opened and fluid is drawn off into one or several 50-ml centrifuge tubes and gently centrifuged several minutes or allowed to stand 15–20 minutes. About two-thirds of the supernatant is removed by vacuum aspiration, and the sediment is resuspended in the remaining fluid, poured and rinsed into a Petri dish, and examined for larvae as described previously. In the case of cloudy samples, a saline wash step can be added.

The advantage of the Baermann technique is its ease of use. The disadvantage is that not all larvae may come down, so that in low infections, false negative results may be obtained (Richardson et al. 1980; Kazacos and Wirtz 1983). We have found that larvae can become trapped in the brain sludge on the cheesecloth, so that for negative samples it is a good idea to stir this material well and rerun the Baermann (Fox et al. 1985). Alternatively, one can remove the brain material to a Petri dish, macerate it further, and examine it thoroughly with an inverted microscope. The material may also be processed further by digestion (see above). Because of these limitations, we prefer brain squashes and digestion for the detection or recovery of *Baylisascaris* larvae.

Identification of *Baylisascaris* L_3's is based on morphologic characteristics of larvae recovered from tissues or seen in histopathologic sections. Other nematode larvae besides *Baylisascaris* may be seen in or recovered from the tissues of animals, especially wild species, although brain infection with other nematodes is much less frequent. The morphology of *B. procyonis* L_3's has been described in detail (Berry 1985; Bowman 1987; Donnelly et al. 1989), and their histologic identification by Kazacos (1986, 1997) and Bowman (1987). Histologic features of other ascarid and helminth larvae are described by Nichols (1956a,b), Chitwood and Lichtenfels (1972), Binford and Connor (1976), Bowman (1987), Connor et al. (1997), and Gutierrez (2000). *Baylisascaris procyonis* larvae are stout, 1500–1900 µm x 60–80 µm, with a smoothly rounded anterior end and three partially differentiated lips (Figs. 11.15, 11.16). Large, single lateral alae commence halfway between the anterior end and the nerve ring and extend to near the tip of the tail, and the cuticle has prominent transverse striations. The esophagus is clavate, strongyliform, ends in a pyriform bulb, and is ~13%–15% of the body length (Figs. 11.15, 11.16). The excretory cell nucleus is large (~20 µm), ovoid,

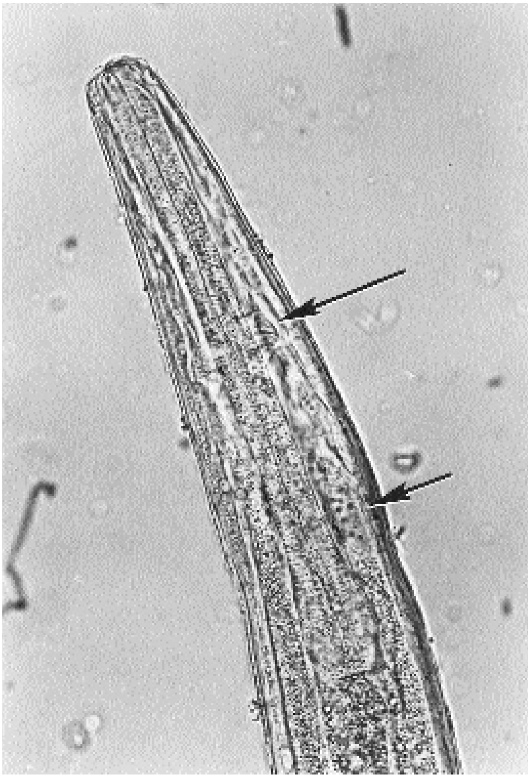

FIG. 11.16—Esophageal region of *Baylisascaris procyonis* larva recovered by the Baermann technique from the brain of a chicken with encephalitis (Richardson et al. 1980). Note partially differentiated lips, clavate strongyliform esophagus, nerve ring (long arrow), and large excretory cell nucleus (short arrow).

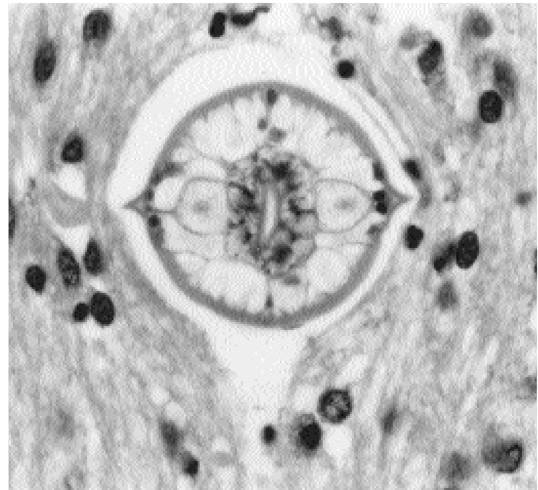

FIG. 11.17—Cross section through midbody/midintestinal region of a *Baylisascaris procyonis* larva in the brain of a ferret, showing characteristic diagnostic features. Note prominent, single lateral alae; large, centrally-located, laterally-compressed intestine with open lumen and microvillous border; and lateral excretory columns which are smaller than intestine, roughly triangular in shape, slightly dissimilar in size, and with prominent canaliculi. In histologic sections, larvae usually measure 60–70 μm in greatest width.

and seen in the left side (dorsal view) or right side (ventral view) of the excretory commissure near the esophageal bulb (Fig. 11.16). The intestine has a patent lumen, and the cells contain abundant granules. The tail tapers gradually from anus to tip, which is flexed sharply dorsad and ends in a tiny knob (Fig. 11.15) (Berry 1985; Bowman 1987; Donnelly et al. 1989; K.R. Kazacos, unpublished).

Histologically, the characteristic features of *Baylisascaris* larvae are best seen in transverse sections through the midbody/midintestinal region (Fig. 11.17) (Kazacos 1997). The larvae are usually 60–70 μm in greatest width and have prominent, single lateral alae, strongly pointed and flexed dorsad. The large, centrally located intestine has an open lumen and is laterally compressed in the mid to posterior regions; six to nine low columnar cells are usually visible, each with a thin microvillous border and numerous cytoplasmic granules. The intestine is flanked by prominent lateral cords supporting the lateral excretory columns, which are smaller than the intestine, roughly triangular in shape, slightly dissimilar in size, and with prominent central canaliculi. Three hypodermal nuclei are usually visible in the lateral cords, just below the cuticle. The shape and relative prominence of the excretory columns and intestine change as one progresses posteriorly from esophagus to anus (Fig. 11.18) (Kazacos 1997). The same structures can usually be seen in longitudinal or tangential sections, although they may be more difficult to identify (Suedmeyer et al. 1996).

IMMUNITY. It is likely that raccoons develop age resistance and/or intestinal immunity following infection with *B. procyonis.* This, combined with self-cure of intestinal infections, probably accounts for the lower prevalence of *B. procyonis* in adult raccoons.

It is well known that intermediate hosts infected with *B. procyonis* larvae develop strong antibody and inflammatory cell responses, directed at excretory-secretory antigens given off by the migrating parasites. The strong antibody responses form the basis for immunodiagnostic tests (immunofluorescence, ELISA, Western blotting) used for this infection (Fox et al. 1985; Boyce et al. 1988a,b, 1989; Goldberg et al. 1993; Cunningham et al. 1994). *Baylisascaris* also stimulates strong blood and tissue eosinophil levels, another indication that the parasite induces a strong T-helper type 2 cell response (Sheppard and Kazacos 1997). As might be expected, host species appear to

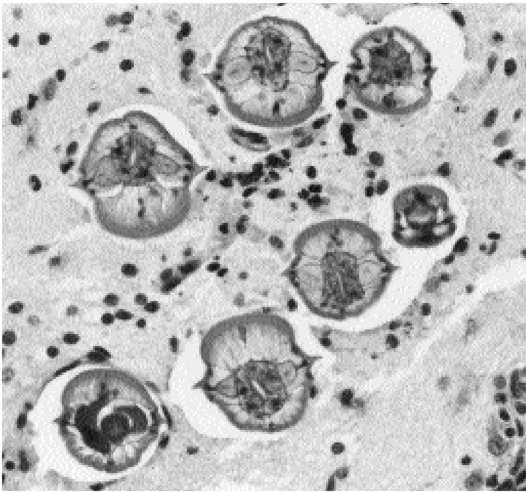

FIG. 11.18—Cross sections of a *Baylisascaris procyonis* larva in the brain of a squirrel monkey, showing changes in the shape and relative prominence of the excretory columns and intestine at different levels of the larva. The larva is coiled vertically from lower left to lower right and is sectioned at the following levels: (lower left) posterior esophagus at excretory commissure, showing excretory cell nucleus; (upper left and middle, lower second and third) midintestinal region, with upper middle section near midbody; (upper right) posterior intestinal region, beyond ends of excretory columns; (lower right) intestinorectal valve. [Reprinted from Kazacos (1983a) with permission of Purdue Research Foundation.]

vary in their specific responses to infection (Sheppard and Kazacos 1997).

Whether immune responses to *Baylisascaris* larvae in intermediate hosts are protective or not is less well known; some protective effect seems possible, especially in previously infected animals in which central nervous system invasion hasn't occurred. Reinfection of such animals might trigger immune responses directed at preventing infection or slowing down and walling off the larvae. This will continue to be difficult to test experimentally, because animals infected with *B. procyonis* often die from central nervous system disease (Boyce et al. 1988a,b, 1989). The only findings that may support this possibility come from a large epizootic of *Baylisascaris* NLM in rabbits in Michigan, in which only young or recently introduced rabbits were clinically affected. No disease developed in older breeder rabbits that had been on the farm for an extended period, suggesting that there was previous contact with the parasite and an acquired immunity (Dade et al. 1975). Whether this was indeed the case or not is unknown, and the question of protective immunity in *Baylisascaris* infections needs further study.

OTHER SPECIES OF *BAYLISASCARIS.* Several other species of *Baylisascaris,* including *B. melis* of badgers, *B. devosi* of fisher and marten, *B. transfuga* of bears, and *B. tasmaniensis* of marsupial carnivores, are potential causes of larva migrans disease if enough eggs are ingested. All four species undergo somatic migration in rodents, but with variations in larval growth, migratory behavior, and distribution of larvae to somatic and visceral tissues, as compared to *B. procyonis* and *B. columnaris* (Sprent 1952a, 1953a,b, 1955; Tiner 1953a,b; Sprent et al. 1973). Similar to *B. procyonis* and *B. columnaris, B. melis* larvae grow considerably and, in addition to invading other tissues and organs, enter the brain to cause NLM. *Baylisascaris melis* produced central nervous system disease in laboratory mice, deer mice (*P. maniculatus artemisiae*), ground squirrels (*Citellus armatus*), and rabbits (Tiner 1953a,b; Boyce et al. 1988b; K.R. Kazocos, unpublished) and entered the eyes of mice, producing OLM (K.R. Kazacos, unpublished). As pointed out by Tiner (1953a), *B. melis* is probably responsible for naturally occurring clinical NLM in ground squirrels and other rodents which share the habitat of badgers and constitute their normal food supply.

The larvae of the other species are smaller and/or grow more slowly and are distributed primarily to the anterior carcass musculature (*B. devosi*) and intestinal wall or mesentery (*B. transfuga, B. tasmaniensis*) of mice (Sprent 1952a, 1953a,b, 1955; Sprent et al. 1973). Although a few larvae also entered the brain, central nervous system disease was absent or rare with *B. devosi* and was not caused by *B. transfuga* or *B. tasmaniensis.* However, other research indicates that *B. transfuga* can produce clinical NLM as well as OLM in some infected mice (Papini and Casarosa 1994; Papini et al. 1996). Although they are potential causes of larva migrans disease in animals and humans, *B. transfuga, B. devosi,* and *B. tasmaniensis* are much less pathogenic than the other three *Baylisascaris* species discussed. Because of their much greater disease-producing capabilities, *B. procyonis, B. melis,* and *B. columnaris* are clearly the most dangerous members of this group and should be handled with the greatest precautions.

TREATMENT

Definitive Hosts. Many of the common anthelmintics used to treat ascarids in dogs, cats, and other species are effective against adult *Baylisascaris* in raccoons, skunks, dogs, and bears. We have successfully treated *B. procyonis*–infected raccoons with the following drugs (dosage x treatment day other than one): piperazine citrate (120–240 mg/kg), pyrantel pamoate (6–10 mg/kg), and fenbendazole (50–100 mg/kg x 3–5 days) (Kazacos et al. 1982a; Kazacos 1986); follow-up treatments are recommended to ensure removal of all worms. The following anthelmintics were 100% effective against *B. procyonis* in raccoons when fed in small amounts of moist cat food: pyrantel pamoate (20 mg/kg), ivermectin (1 mg/kg), moxidectin

(1 mg/kg), albendazole (50 mg/kg x 3 days), fenbendazole (50 mg/kg x 3 days), and flubendazole (22 mg/kg x 3 days) (Bauer and Gey 1995). Intramuscular ivermectin cleared *B. procyonis* from 11 of 12 raccoons, but 1 animal continued to shed eggs following treatment at 2 mg/kg (Hill et al. 1991).

Raccoons and skunks kept in captivity for any reason should be examined regularly (by fecal flotation) and strategically dewormed for *Baylisascaris,* in order to prevent or decrease environmental contamination with eggs and possible transmission to humans and other species (Kazacos and Boyce 1989). Newly acquired raccoons and skunks should be quarantined and dewormed immediately, with at least two follow-up treatments at 14-day intervals to ensure elimination of all developing worms. Young raccoons and skunks pose a particular threat because they have a higher prevalence of infection and are often acquired during the prepatent period (50–76 days for *B. procyonis,* via eggs), when they will be false-negative by fecal exam (Kazacos 1983b). These individuals can be fecal-negative many weeks, then suddenly begin shedding large numbers of eggs, resulting in extensive contamination. Thus, strategic deworming of young raccoons and skunks should be started at ~5–6 weeks of age and repeated on a regular basis (e.g., every 2 weeks for five or six treatments). It is recommended that all captive raccoons and skunks be housed away from other species, in clean dedicated cages or enclosures that can be decontaminated if necessary. Placing young raccoons or skunks in egg-contaminated cages or feeding raccoons and skunks meat from wild animals (rodents, rabbits, birds) can result in *Baylisascaris* infection (Kazacos and Boyce 1989). Based on the minimum known prepatent period of *Baylisascaris* in raccoons and skunks (32 days for *B. procyonis,* via larvae), once infections are eliminated, strategic deworming at monthly intervals should prevent future environmental contamination with eggs (Kazacos and Boyce 1989; Bauer and Gey 1995).

Intermediate Hosts (Neural and Ocular Larva Migrans). With or without treatment, NLM due to *Baylisascaris* carries a guarded to poor prognosis. The efficacy of anthelmintic treatment of NLM depends on drug pharmacokinetics and activity against larvae in the central nervous system; clinical efficacy also depends on the level and duration of central nervous system infection and the extent of central nervous system damage at the time of treatment. Treatment of early, low-level central nervous system infection appears possible, using larvicidal drugs which effectively cross the blood-brain barrier; presently, the best candidates appear to be albendazole and diethylcarbamazine (see below). Unfortunately, *Baylisascaris* NLM usually is not considered or diagnosed until central nervous system signs are pronounced, and extensive, irreparable central nervous system damage has already occurred; anthelmintic treatment at this stage is usually ineffective. Killing larvae in the central nervous system carries the added risk of exacerbating inflammatory reactions, due to the release of larval antigens (Kazacos and Boyce 1989). Controlling parasite-induced inflammation in the central nervous system with corticosteroids, and supportive maintenance of the patient are both very important. Any anthelmintic treatments should be started as early as possible, because larvae in early migration in extraneural sites are more amenable to treatment than after they have entered the central nervous system. Possible drugs for such treatment include albendazole, mebendazole, fenbendazole, thiabendazole, diethylcarbamazine, and levamisole.

Baylisascaris procyonis larvae enter the brain of mice as early as 3 days postinfection, and clinical central nervous system disease develops by 9–10 days postinfection (Tiner 1953a,b; Kazacos 1986; Sheppard and Kazacos 1997). Mice infected with 100 *B. procyonis* eggs and treated daily 1–10 days postinfection with albendazole (25 mg/kg), mebendazole (25 mg/kg), or thiabendazole (500 mg/kg) were protected 100%, 80%, and 80%, respectively, from development of central nervous system disease (Miyashita 1993). When treatment was 1–3 days postinfection only, central nervous system protection declined to 40%, 20%, and 20%. Several other anthelmintics had lower efficacy. Mice infected with 250 *B. procyonis* eggs and treated daily 1–10, 3–10, and 7–10 days postinfection with albendazole (50 mg/kg) +/- prednisone (1 mg/kg) were protected 100%, 95%, and 75%, respectively, from central nervous system disease (Garrison 1996). Mice treated similarly with diethylcarbamazine (100 mg/kg) +/- prednisone were protected at 100%, 100%, and 45%, respectively. In these experiments, inclusion of steroids did not significantly improve treatment efficacy.

Gradual improvement was seen in two black-and-white ruffed lemurs with probable *Baylisascaris* NLM at the Nashville Zoo, following extended treatment with albendazole (5 mg/kg 3 times per day for alternating 2-week periods and rest over 4 months) (S.J. Barrett, unpublished). The slowly progressing central nervous system disease was arrested after the first 2-week course, and the animals gradually improved, relearning some motor skills. These animals probably had low-level infections, as evidenced by their mild signs and low cerebrospinal fluid eosinophilia, making them good candidates for treatment. A California infant with *Baylisascaris* NLM was treated with albendazole (40 mg/kg x 28 days) and steroids (methylprednisolone, 20 mg/kg/day), but without obvious clinical improvement, probably due to the extent of central nervous system damage (Park et al., 2000).

Because of its efficacy against a variety of other parasites, ivermectin has received considerable attention for the treatment of *Baylisascaris* NLM. Unfortunately, ivermectin does not cross the blood-brain barrier well and has proved unsuccessful in all treatment attempts of which the author is aware. The following animals and humans with clinical NLM, when treated with ivermectin at the dosages indicated, failed to improve, continued to deteriorate, and died or were euthanized,

with living larvae subsequently recovered from the brain: rabbits (600 µg/kg) (Deeb and DiGiacomo 1994); gray squirrels (200 µg/kg every 2 weeks for three treatments) (Tseng 1997); rock doves and a Douglas squirrel (300 µg/kg) (Coates et al. 1995); macaws (400 µg/kg) (Armstrong et al. 1989); emus (200 µg/kg) (Suedmeyer et al. 1996); marmosets (100 µg/kg) (Huntress and Spraker 1985); and a child (175 µg/kg, total dose 1.5 mg) (Cunningham et al. 1994). Similarly, treatment of clinically affected rabbits with thiabendazole (25 mg/kg x 28 days) or tetramisole (8 mg/kg) (Dade et al. 1975), marmosets with fenbendazole (10 mg/kg x 10 days) (Huntress and Spraker 1985), and two children with thiabendazole (50 mg/kg x 6–7 days) (Fox et al. 1985; Cunningham et al. 1994) was unsuccessful.

Although there is a general lack of effective anthelmintic therapy for clinical NLM due to *Baylisascaris* (except for early treatment as noted above), several anthelmintics show great promise as preventatives for this infection. Currently, the best candidates are the pyrantel compounds, pyrantel tartrate and pyrantel pamoate, which prevent initial infection and thus subsequent central nervous system disease due to *Baylisascaris.* When administered continuously in the feed, pyrantel tartrate (Banminth, Pfizer) is a well-known preventative for *Ascaris suum* migration in swine. Experimentally infected mice given pyrantel tartrate at 0.25% and 0.5% and pyrantel pamoate at 0.2% concentration in their feed were fully protected against *B. procyonis* infection and central nervous system disease, which proved 100% fatal to untreated mice (Lindquist 1978). As pointed out by Kazacos and Boyce (1989, Addendum 1995) and Suedmeyer et al. (1996), pyrantel tartrate for swine could be formulated directly into ratite or other feed, or the pelleted formulation (Strongid C for horses) could be added as a top dressing; all would constitute extralabel usages of these drugs. Because of their high efficacy, acceptance, and ease of use, pyrantel drugs are recommended for prevention of *Baylisascaris* larval infection in mammals and birds. They should be used whenever animals are exposed to known or potentially contaminated environments, particularly on premises with an ongoing problem, where the sources of infection cannot be identified or effectively decontaminated. Pyrantel tartrate pellets (Strongid C) were well accepted by emus at the Kansas City Zoological Gardens, whereas acceptance of oral ivermectin was inconsistent (Suedmeyer et al. 1996). Pyrantel tartrate pellets are also being fed to lemurs and other species at various zoos in the United States.

Periodic treatment with ivermectin has also been used in an effort to kill *Baylisascaris* larvae in preneural migration, but with less consistent results. Miyashita (1993) found that only 20% of mice treated daily 1–10 days postinfection with ivermectin (1 mg/kg) were protected from central nervous system disease. In an outbreak of *Baylisascaris* encephalitis in emus, all remaining birds were placed on ivermectin (200 µg/kg) per os every 2 months. However, when an additional emu developed central nervous system disease 2 months later, the regimen was increased to monthly treatment, and no subsequent clinical cases were seen (Kwiecien et al. 1993). In another outbreak, three emus were treated with ivermectin (200 µg/kg per os) every 20–30 days, and all remained clinically normal for a year, whereupon one developed progressive central nervous system disease and was euthanized. The remaining two emus were then treated with ivermectin weekly and, a year later, were put on pyrantel tartrate pellets (50 mg/kg) in their feed (Suedmeyer et al. 1996).

Cases of OLM and DUSN due to *Baylisascaris* in humans have been successfully treated using laser photocoagulation to destroy the intraretinal larvae, thus preventing further migration damage (Raymond et al. 1978; Williams et al. 1988; Goldberg et al. 1993; Küchle et al. 1993). Visual improvement following treatment depends on the location and extent of intraocular damage and on successful resolution of intraocular inflammation using corticosteroids. The efficacy of oral anthelmintics against intraocular *Baylisascaris* larvae has not been evaluated.

PREVENTION AND CONTROL. Considering the seriousness of *Baylisascaris* infection in animals and humans, as well as the lack of effective treatment for NLM, prevention of infection with *Baylisascaris* is of utmost importance. The three key elements for preventing and controlling *Baylisascaris* infections in animals and humans are (1) reducing environmental contamination with infective eggs, (2) preventing contact with contaminated areas or articles, and (3) educating people about these parasites as causes of animal and human disease. These approaches are interrelated and should be carried out in concert as part of a comprehensive prevention and control program.

Reducing environmental contamination with eggs in an area can only be accomplished by treating, removing, or relocating infected raccoons and skunks. Keeping raccoons and skunks as pets should be strongly discouraged, especially in households with young children. Pet permittees and those involved in wildlife rehabilitation should have adequate knowledge of *Baylisascaris* and other zoonotic diseases or be provided such information. Anthelmintic treatment of raccoons and skunks kept as pets or for other reasons is readily accomplished but must be done adequately and properly (see above) in order to prevent contamination.

Feral raccoons and skunks are much more difficult to deal with and are involved in the majority of *Baylisascaris* infections. Feral raccoons and skunks can be a considerable nuisance in and around zoos and other animal facilities, on farms, and in the suburban domestic environment, where they are responsible for widespread fecal contamination. The excellent climbing ability of raccoons and the fact that they will establish latrines in elevated locations poses a particular problem for zoos using roundhouse enclosures, especially in or

near wooded areas. It should theoretically be possible to use baits containing anthelmintics to deworm feral raccoons and skunks in an area, similar to the use of baits for rabies vaccination of wildlife or the bait treatment of *Echinococcus* in foxes in Europe. The timing, frequency, and logistics of such treatments, as well as their effectiveness in decreasing prevalence of *Baylisascaris* in local populations, are not well known, although some encouraging results were seen in a recent study. LoGiudice (1995) baited raccoons with piperazine at two sites in central New Jersey and saw a significant reduction in egg-positive scats in latrines at one of the sites post-baiting, whereas positive scats increased at one of two control sites (the other treatment and control sites showed no differences). Although there was a significant overall reduction in positive scats at the treatment sites as compared to control sites, it was impossible to identify the contributions of individual animals at latrines. Two of three scats which contained a fluorescent marker (indicating bait consumption) also had adult *Baylisascaris* present, indicating successful treatment. Because too few raccoons were trapped and examined for eggs, nothing could be stated as to the effects of anthelmintic baiting on prevalence of the parasite. Additional studies are needed to better assess the usefulness of baiting for control of *Baylisascaris* in wild populations. With the very high population densities of feral raccoons and skunks in many suburban areas, as well as the likelihood of reinfection, this approach could prove costly and time-consuming and still have questionable effectiveness, especially if attempted on too large a scale. Depending on the situation, however, it may be advantageous to deworm a stable, localized resident population on a regular basis as part of overall control efforts, combined with latrine cleanup and decontamination. It is also very important to discourage people from intentionally feeding feral raccoons and to control other food sources (pet food, garbage) which serve to maintain high populations. This would not only help stabilize local raccoon populations but could also reduce the establishment of new latrine sites and levels of fecal contamination in the domestic environment.

The other, more straightforward method of dealing with this problem is through depopulation and removal of raccoons and skunks. This has the advantage of immediately reducing new environmental contamination in an area and is also best combined with latrine cleanup and decontamination. Trapped animals may be relocated to distant sites (thereby making them someone else's problem), or euthanized. Many suburban zoos have ongoing wildlife control programs in an effort to reduce and control nuisance wildlife and their diseases. The main impediments to such approaches are not technological or logistical, but political and social, primarily objections from animal rights and related groups (Stringfield and Sedgwick 1997). However, there is no question but that this is the most direct and effective method of reducing environmental contamination and transmission of *Baylisascaris* to all species, and it must be considered seriously wherever this disease problem occurs.

Dealing with contaminated areas is more problematic, because of the marked resistance of *Baylisascaris* eggs. Once in the environment, the eggs can survive for years. Eggs are resistant to all common disinfectants, including bleach, although certain solvent mixtures will kill them. Thus, small areas of contamination on resistant surfaces can be treated with 1:1 xylene:ethanol after most organic material has been removed (Kazacos and Boyce 1989). Treatment with 20% bleach (1% sodium hypochlorite) will remove the outer protein coat, making the eggs nonadherent and able to be washed away, but will not kill them. Chemical treatments which kill eggs are generally not practical for use in the environment (Kazacos and Boyce 1989; Kazacos 1991). Desiccants such as sodium borate broadcast onto latrine sites at a sufficient rate might hasten egg death, but this has not been studied.

Heat is by far the best method of killing *Baylisascaris* eggs. Boiling water, a propane flame gun, steam cleaner, autoclave, burning straw, or other means can be used for small or large areas of contaminated soil or concrete, metal cages, enclosures, holding pens, and contaminated tools and utensils (Kazacos and Boyce 1989; Kazacos 1991, 2000). Direct flame from a propane gun is the most effective method for destroying eggs. Our laboratory routinely uses such a device (VT 3-30 Red Dragon Vapor Torch; Flame Engineering, LaCrosse, Kansas) to decontaminate live traps, cages, and enclosures that have held *Baylisascaris*-infected raccoons or skunks. This method has also been used to decontaminate concrete-floored animal rooms, kennel runs, and raccoon latrine sites in zoos and around homes (Pegg 1977; Abdelrasoul and Fowler 1979; Kazacos and Boyce 1989; Kazacos 1991; K.R. Kazacos, unpublished). Surface soil can be flamed, broken up, and turned over several times with a shovel or rake and reflamed each time to ensure decontamination. Obviously, appropriate care should be taken when using this method, particularly in or around buildings and other flammable materials.

For heavily contaminated areas, it may be desirable to remove and discard the top several inches of soil and replace it; this may be combined with heat treatment of the area (e.g., with a flame gun). Dried raccoon feces and other contaminated material (hay, straw, leaves) in exhibits or buildings should be carefully removed and properly disposed of (e.g., by incineration). Residual material in buildings can then be treated with steam or boiling water or removed using a canister-type vacuum cleaner containing a disposable filter bag. Personnel cleaning contaminated areas should wear disposable coveralls, rubber gloves, washable rubber boots, and a particulate face mask to prevent the inhalation or ingestion of any eggs and fecal fungi stirred up in dust (Kazacos and Boyce 1989). When finished, disposable items should be incinerated, autoclaved, or otherwise properly disposed of. The presence of eggs in soil or environmental debris, as well as the effectiveness of

their destruction or removal, can be assessed using centrifugal sedimentation-flotation methods on detergent-washed samples (Kazacos 1983c).

It is important to prevent contact with known or suspected contaminated areas or articles until they can be properly assessed and effectively decontaminated, or removed or destroyed. This would include cages or enclosures which previously housed raccoons or skunks, areas or articles contaminated by feral animals, and raccoon latrine sites in and around the domestic or zoo environment. Care should be taken to prevent raccoon fecal contamination of hay, straw, and feed, and contaminated materials should not be used. Fallen timber, large tree limbs, and rocks from the wild should be carefully inspected, washed, and heat-treated before use in animal enclosures or exhibits.

Education of individuals and groups about the health hazards associated with *Baylisascaris* is perhaps the most important aspect of prevention and control (Kazacos 1991). Efforts should be made to inform a wide spectrum of people about these parasites, including wildlife biologists, natural resources personnel, animal care directors and staff, wildlife rehabilitators, animal damage control officers, public health personnel, veterinarians, physicians, and the general public. The diseases caused by *Baylisascaris* are preventable through simple, straightforward measures, but these will not be taken unless people understand and appreciate the problem.

PUBLIC HEALTH CONCERNS. Extensive opportunities exist for contact and infection of human beings with *Baylisascaris,* especially *B. procyonis* from raccoons. Raccoons are extremely common and well adapted to coexistence with human beings in urban, suburban, and rural environments. Some of the highest recorded densities of raccoons are from suburban residential areas, particularly in and around wooded parks and neighborhoods; in these areas, humans are very likely to encounter raccoon fecal contamination (Hoffman and Gottschang 1977; Greve 1985; Kidder 1990; Rosatte et al. 1991; Feigley 1992; W.J. Murray et al., unpublished; Park et al. 2000). Because of their engaging qualities, raccoons are often encouraged through feeding to frequent peoples' yards and homes, where they will establish latrine sites. In addition, raccoons and skunks are frequently kept as pets, increasing the likelihood of human contact and infection with *Baylisascaris.*

Human infection with *Baylisascaris* was anticipated in the 1960s (Beaver 1969), based on experiments done in rodents by Tiner (1951, 1952a, 1953a,b) and Sprent (1951, 1952a, 1955). The zoonotic importance of *B. procyonis* was indicated by Kazacos and associates in the 1980s, based on epidemiologic studies of infected animals and experimental infection of subhuman primates and other species (see Kazacos 1981, 1983a, 1986, 1991; Kazacos et al. 1981b, 1984a,b, 1985; Kazacos and Boyce 1989). Human deaths caused by *B. procyonis* were first documented in children in 1984–85 (Huff et al. 1984; Fox et al. 1985), at which time the parasite was also implicated as a cause of human OLM (Kazacos et al. 1984a,b, 1985). To date, there have been documented fatalities from *Baylisascaris* NLM in infants in Pennsylvania (Huff et al. 1984), Illinois (Fox et al. 1985), and Minnesota (C.L. Moertel et al., unpublished), and cases of severe, disabling central nervous system disease in infants in New York (Cunningham et al. 1994), Michigan (J.M. Proos et al., unpublished), Illinois (M.B. Mets et al., unpublished), Minnesota (C.L. Moertel et al., unpublished), and California (Rowley et al. 2000; Park et al. 2000), with a probable case in Missouri (Anderson et al. 1975). The parasite also produced central nervous system disease in a 21-year-old man in Oregon (cited in Cunningham et al. 1994) and a 17-year-old boy in California (W.A. Kennedy et al., unpublished) and was considered the cause of death from an eosinophilic intracardiac mass in a 10-year-old boy in Massachusetts (Boschetti and Kasznica 1995). *Baylisascaris* infection was demonstrated serologically in clinically normal individuals in New York (Cunningham et al. 1994) and Germany (Conraths et al. 1996), indicating that asymptomatic, low-level infection also takes place. In cases involving infants, infection was linked to contact with raccoon feces in open fireplaces in the home, coming from raccoons living in the chimneys (Huff et al. 1984), chewing on pieces of bark from contaminated firewood brought into the home (Fox et al. 1985), and geophagia at or near raccoon latrines in the domestic environment (Cunningham et al. 1994; M.B. Mets et al., unpublished; C.L. Moertel et al., unpublished; Park et al. 2000).

In humans, *Baylisascaris* more commonly causes clinical OLM, with dozens of cases now recognized in North America and Europe (Gass and Braunstein 1983; Kazacos et al. 1984a,b, 1985; Kazacos 1991, 1997, unpublished; Goldberg et al. 1993; Küchle et al. 1993). Large nematode larvae were documented in human eyes as early as 1952 in the United States (Parsons 1952) and 1961 in Europe (Schrott 1961), and seven cases were assembled by Gass and Braunstein (1983) in their further description of DUSN, but the etiology was not determined. *Baylisascaris procyonis* was suggested as the probable cause, based on the pathogenesis of *B. procyonis* in animals and the compatible size, geographic location, and pathogenesis of the larvae in these patients; in addition, one of the patients had known raccoon contact (Kazacos et al. 1984a,b, 1985). With additional, well-documented cases linked morphometrically and serologically to *Baylisascaris* infection and raccoon exposure (Goldberg et al. 1993; Küchle et al. 1993), *B. procyonis* is now recognized as an important cause of human OLM and the primary cause of the large nematode variant of DUSN.

The fact that OLM and DUSN are usually related to low-level infection further indicates that human infection with *Baylisascaris* is probably common. Many more people will contract low-level infections than

heavy infections, and unless the larvae migrate to the eye or brain in sufficient numbers, these individuals will not develop clinically significant disease. Similar to infections with *Toxocara canis,* most human infections with *Baylisascaris* are probably asymptomatic. These cases would be characterized only by low serum antibody titer, with or without mild eosinophilia, and without signs of visceral, ocular, or neural larva migrans (Kazacos 1991, 1997; Cunningham et al. 1994; Conraths et al. 1996).

At the other end of the spectrum, infants 1–4 years old are at greatest risk of heavy infection with *Baylisascaris* because of their poor hygiene and propensity for pica and geophagia. Special attention should be paid to this age group and other children in order to prevent life-threatening infections with these parasites (Kazacos 2000). Children should be kept away from known or potentially contaminated areas and taught to recognize and avoid raccoon latrines they may encounter in the environment. They should also be monitored closely to prevent pica and geophagia and taught to wash their hands regularly, especially after contact with outdoor areas or animals and prior to eating.

DOMESTIC ANIMAL HEALTH CONCERNS. Ample opportunities exist for contact and infection of domestic animals with *Baylisascaris,* especially *B. procyonis.* Fatal central nervous system disease due to *B. procyonis* has been seen in pet dogs, rabbits, porcupines, and psittacines, as well as farm-raised rabbits, chinchillas, poultry, quail, pheasants, and ratites (Table 11.6). *Baylisascaris* appears to have only limited migration in large domestic livestock (Dubey 1982; Snyder 1983; Kazacos and Kazacos 1984), except perhaps during pregnancy; the only known case involved a lamb with transplacentally acquired infection (Anderson 1999). Infection of domestic animals is commonly associated with keeping raccoons on the premises, using cages or enclosures that previously held raccoons, using contaminated hay, straw, or feed, and exposure to fecal contamination from feral raccoons (Kazacos and Boyce 1989).

Two fatalities in hunting dogs were linked to wild-caught raccoons kept on the premises for training purposes (Thomas 1988; Rudmann et al. 1996). Many dogs, especially puppies, are coprophagic and will ingest raccoon feces they encounter in the domestic environment or surrounding woods. This could result in fatal central nervous system disease, or in some cases patent intestinal infection with *B. procyonis* (Greve and O'Brien 1989; Miyashita 1993; Averbeck et al. 1995; D.D. Bowman, unpublished; K.R. Kazacos, unpublished). In the largest outbreak of NLM recorded to date, 622 chickens died over a 7-week period, following the use of raccoon feces–contaminated straw litter in a poultry facility (Richardson et al. 1980). In another case, 100% mortality occurred in 85 bobwhite quail placed in a 12 x 24 foot dirt pen previously used to house three young pet raccoons (Reed et al. 1981). Over a 3-month period, these raccoons contaminated the pen with over 155,500,000 *B. procyonis* eggs, even though they were shedding at a low rate (1300–5400 eggs/g feces, mean 2800/g) (Kazacos 1982). In a recent dramatic outbreak, 10 pet psittacines in a mixed collection were killed acutely by massive *B. procyonis* infection acquired from contaminated feed. Feral raccoons had contaminated a stored seed mixture kept in a bin in the owner's garage (A.M. Lennox and K.R. Kazacos, unpublished). These examples graphically illustrate the health hazard posed by *B. procyonis* to a variety of domestic and farm-raised animals.

MANAGEMENT IMPLICATIONS. Without management and control of raccoon and skunk populations in an area or particular situation, it would be very difficult or impossible to reduce or eliminate potential transmission of *Baylisascaris.* It is well established that wildlife populations can increase to the point of serious nuisance, particularly in urban and suburban areas, with damage to buildings and vegetation, predation on domestic and zoo animals, widespread fecal contamination, and transmission of infectious and parasitic diseases. Raccoons and skunks are foremost examples of this problem, and in addition to causing property damage and other losses, pose the real threat of *Baylisascaris* transmission to animals and humans in an area.

An excellent example of the problems and management implications associated with nuisance wildlife and *Baylisascaris* was recently described by Stringfield and Sedgwick (1997) at the Los Angeles Zoo. What they depict is not unlike the situation at many other suburban zoos, and much can be learned from it: "The Los Angeles Zoo sits in the middle of Griffith Park, which is a large wild area. Previous nonmanagement of pests had allowed the zoo to become overrun with these animals, and problems had reached epidemic proportions in 1995. Coyotes living in the zoo were hunting gerenuk and flamingos, skunks were everywhere, and raccoons had free-roam of the zoo. In the past 3 yr at the Los Angeles Zoo, we have seen numerous cases of central nervous system disease secondary to *Baylisascaris*". A change in zoo management brought an immediate and aggressive, multifaceted response to these problems. This included trapping and removal of the resident raccoon, skunk, and coyote populations, repairing gaps in the perimeter fence to prevent future influx, trimming trees and overhanging foliage, installing wire mesh along the bottoms of exhibits to prevent animal access, repairing garbage bins, rehabilitating contaminated exhibits, and instituting ongoing surveillance and control measures, coupled with a program of staff education. Although the scope of the task was daunting, with thoughtful planning and implementation the program was successful, and nuisance wildlife and *Baylisascaris* transmission to zoo animals were both brought under control.

A second example involves the impact of *Baylisascaris* on indigenous wildlife, including threatened and

endangered species, following introduction of the parasites and/or increases in raccoon or skunk populations in an area. The Allegheny woodrat (*Neotoma magister*) was extirpated from New York state and Connecticut and continues to decline in other parts of its northeastern range. In the Hudson Highlands region of New York, declines were linked to an increase in the raccoon population and to NLM caused by *Baylisascaris.* In Mohonk Preserve, New Paltz, New York, abnormal behavior was documented in various animals including gray squirrels and deer mice found circling or unable to climb (Smiley and Huth 1986). Ward Stone, Associate Wildlife Pathologist for the state, suggested *B. procyonis* NLM as the probable cause and also hypothesized its role in the extirpation of woodrats from the preserve between 1959 and 1977. Previously, Smiley (1977a,b) had astutely noted declines in the woodrat population as well as increases in the raccoon population and wondered if the two were related. Sighting a raccoon at Mohonk was a rarity from 1923 to 1932, but by the end of 1949 and subsequently they were abundant (Smiley 1977a).

The New York Department of Environmental Conservation (DEC) began surveying historic woodrat sites in 1978 and noted marked declines in the species, which became extirpated from the state in 1987. The DEC undertook release studies at Mohonk in 1991, which indicated that *B. procyonis* was the likely cause of extirpation of *N. magister* from the region, in combination with other factors (McGowan 1993). The steep rock and boulder talus slopes preferred by woodrats are also very attractive to raccoons, which use them for den and latrine sites. Uninfected woodrats from West Virginia released into these areas, as well as their offspring, died of *Baylisascaris* NLM, which was documented in all 11 woodrats recovered and examined; 4 woodrats had exhibited abnormal behavior when live-trapped (McGowan 1993). In addition, *Baylisascaris* NLM was identified recently in Allegheny woodrats in southern Indiana (K.R. Kazacos and S.A. Johnson, unpublished) and southcentral Pennsylvania (J. Wright et al., unpublished), and in a woodrat released in New Jersey (K. LoGiudice, unpublished). In New York and Indiana, the caching of raccoon feces by woodrats also was documented, indicating a direct behavioral link to *B. procyonis* infection in this species. The plight of the Allegheny woodrat demonstrates that *Baylisascaris* can have a significant impact on indigenous wildlife. Because of *Baylisascaris,* the reintroduction and/or long-term survival of particular animal species may be difficult or impossible without management of raccoon and skunk populations in an area.

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