1983

Prevalence of Baylisascaris procyonis in Northern Illinois and Larval Migration in the Mongolian Gerbil (Meriones unguiculatus)

Diane Mary Sudduth
Loyola University Chicago

PREVALENCE OF BAYLISASCARIS PROCYONIS IN NORTHERN ILLINOIS AND LARVAL MIGRATION IN THE MONGOLIAN GERBIL (Meriones unguiculatus)

by

Diane Mary Sudduth

A Thesis Submitted to the Faculty of the Graduate School of Loyola University of Chicago in Partial Fulfillment for the Degree of Master of Science

September 1983

ACKNOWLEDGEMENTS

The author wishes to express her gratitude to her advisor, Dr. Benedict Jaskoski of Loyola University, for his valuable guidance, patience and understanding throughout the course of this work and for his assistance in the preparation of this manuscript. Special thanks goes to Dr. Edward Palinscar and Dr. Clyde Robbins for their help and work as committee members. Sincere appreciation is also extended to Kurt Tompkins and Tompkins Fur Company, Waterman, Illinois, and to George Hubert, Fur Biologist, Illinois Department of Conservation for their help in providing raccoon viscera.

My special thanks also go to my parents for their help, encouragement and financial support throughout the course of my graduate studies.

VITA

Diane Mary Sudduth, daughter of Mary and Robert Hamel, was born on May 27, 1961.

After graduation from St. Theresa's Middle School in Yorkshire England in 1974, she attended Peru Central Sr. High School in Peru, New York from 1976-1978 and graduated in May 1978. She received a Bachelor's Degree in Chemistry in 1981 from the State University of New York at Plattsburgh. From 1981-1983 she pursued graduate studies at Loyola University at Chicago and was a teaching assistant in Biology.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Baylisascaris procyonis and B. columnaris are ascarids of raccoons and skunks, respectively, and are recognized producers of visceral larva migrans (VLM) and cerebrospinal nematodiasis (CN) in many animals in North American. Animals that have been reported as unnatural hosts of B. procyonis are; woodchucks by Kazacos (1981); rabbits by Jacobson, Scalon and Nettles (1976); squirrels by Schueler (1973); chickens by Richardson, Kazacos, and Thacker (1980); quail by Reed, Kazacos and Dhillon (1981); and in an Australian (Latham's) brush turkey to Kazacos, Render and Thacker (1982). Ingestion of infective eggs and fecal contamination of bedding or food are the causes of infection. Hoffmeister and Mohr (1972) have shown that the raccoon is present in every county of Illinois and is particularly common in parts of the state with extensive wooded areas. Scats or droppings of raccoons are found along margins of streams and lakes where great accumulations are often found because the raccoon commonly follows regular routes and leaves his droppings in a few chosen spots. B. procyonis VLM has been reported in man, it is a legitimate concern that man himself may become a host in places where man lives in close contact with raccoons or where fecal contamination is heavy. It is presently known that B. procyonis will hatch and migrate in man, Kazacos (1983) diagnosed the first case of fatal CNS injury in a 2-year old boy from Pennsylvania.

Upon experimental inoculation with Baylisascaris procyonis infective eggs, Kazacos (1981) found the following range of hosts susceptible to VLM and CN due to B. procyonis: rodents (mice, hamsters, rats, grey squirrels); avians

(chickens, ducks); carnivores (ferrets); primates (squirrel monkeys). This is the first study of Baylisascaris procyonis in the Mongolian gerbil (Meriones unguiculatus). Preliminary work indicated that B. procyonis infective eggs hatch and migrate in the gerbil and cause ataxia, loss of balance, and tremors in the forelimbs. These same symptoms have been reported in other animals known to harbor B. procyonis larvae. Studies of the minimum lethal dose, the smallest dosage of ascarid eggs that will cause death in the host, have been done with Ascaris lumbricoides var. suum in hamsters by Jaskoski (1960), in guinea pigs by Kerr (1938), and with B. procyonis in mice by Dubey (1982).

The objectives of the present investigation are:

1. To determine the prevalence of Baylisascaris procyonis in the natural host the raccoon (Procyon lotor) in a Northern Illinois area.
2. To orally inoculate infective Baylisascaris procyonis eggs into gerbils (Meriones unguiculatus) in order to determine if migration occurs and if so, the effects in the gerbil.
3. To determine the lethal dose of Baylisascaris procyonis eggs in the gerbil as compared to published reports on the minimum lethal dose of B. procyonis in the mouse and A. lumbricoides var. suum in the hamster and guinea pig.

LITERATURE REVIEW

Baylisascaris procyonis is an obligate parasite of the raccoon, Procyonis lotor. It belongs to the phylum Nemsthelminthes, class Nematoda. The genus Baylisascaris was named in 1922 after Dr. H. A. Baylis (formerly of the British Museum of Natural History), and defined by Sprent (1968) to include several species of ascarids previously categorized as members of the genera Ascaris or Toxascaris.

Baylisascaris can be differentiated from the genus Ascaris in three ways: (1) the pre- and post cloacal papillae found on the male tail are segregated in Baylisascaris but undivided in Ascaris, (2) cervical alae are reduced in both sexes of Baylisascaris but remarkable in Ascaris, and (3) the surface contact between the cuticle and the cuticular bars present in Baylisascaris is lacking in Ascaris.

The digestive system consists of a mouth surrounded by three well-developed trapezodial lips, a straight muscular esophagus, an intestine, rectum and anus. The nervous system is comprised of six longitudal nerves connected between an esophageal ring and a cloacal commissure. An H-shaped excretory system is also characteristic. The reproductive and excretory tracts in males open into the cloaca while the reproductive tract in females opens separately.

The first extensive review of the literature concerning the parasites of North American carnivores was published by Stiles and Baker (1935) and includes a list of the helminths reported from raccoons (taken from Barnstable and Dyer, 1974). Stains (1956) reported a subsequent list of raccoon helminths to include what is now known as Baylisascaris procyonis.

Snyder and Fitzgerald (1979) found 94% of 40 juvenile raccoons from central Illinois parasitized by Baylisascaris procyomis. Thirty-six of 121 (30.6%) raccoons positive for B. procyonis were collected from West Lafayette, Indiana by Kazacos (1980). Jacobson (1982) reported 104 of 147 (70.7%) raccoons taken from Tippecanoe County, Indiana positive. Seven of 29 (25%) raccoons collected from columbus, Ohio by Dubey (1982) were positive for B. procyonis.

Ascarids of the raccoon and skunk undergo modifications when ingested by unnatural hosts. Tiner (1952) found that these larvae, especially that of the raccoon ascarid (now known as Baylisascaris procyonis; Sprent, 1968; Stefanski and Zarnowski, (1951) tend to wander from the normal ascarid migration pathway (intestine to liver to right side of heart ot lungs) and invade the brain of rodents where they grow to 1 millimeter in length and cause fatailites. Sprent (1952) traced the migration of the raccoon ascarid in the mouse and found that the most conspicuous characteristic was the wide spread migration of the larvae. By the first day after infection, larvae had reached the lungs and many were recovered from the liver and intestinal wall. On the third and fourth days, the mice showed symptons of severe pulmonary disorder. A large number of larvae was recovered from the liver and lungs between the 4th and 28th day after inoculation. At ten days, encapsulated larvae were seen in the heart, kidneys and liver in white "spots" containing living larvae. On the 17th day after infection, the mice began to show signs of central nervous system (CNS) injury and died shortly after.

Kazacos, Wirtz and Burger (1981) sutdies Baylisascaris procyonis infection in subhuman primates and found larvae were most abundant in the CNS, heart, liver and lungs in squirrel monkeys (Saimiri sciureus) infecuted with 5000 or

10,500 B. procyconis eggs. He also found low numbers of larvae (2-4 larvae) in the brain which he believed caused significant, often fatal migration damage. Dubey (1982) fed 62 to 1000 infective B. procyonis eggs to Swiss white mice (outbred, laboratory) and observed that 40 of 60 mice died ten days or more after infection, and mice fed 2 B. procyonis eggs were fatal of 1 of 6 mice. He attributed this to fatal migration of larvae in the brain of mice.

MATERIALS AND METHODS

Baylisascaris procyonis Prevalence

In a two year survey conducted from November through January 1981 and 1982, raccoon heart, intestines, liver and lungs were obtained from Tompkins Fur Company in Waterman, DeKalb County, Illinois. Raccoons were collected within a fifty mile radius around Waterman, and weight, length and sex were recorded in 1981. In 1982 forty-nine raccoons were collected, but weight and length were not recorded. The small and large intestines were examined for Baylisascaris procyonis, and the hearts were examined for dog heartworm.

Preparation of Eggs

The uteri of worms were dissected out and the terminal ½ inch excised, ensuring the isolation of as many fertile eggs as possible. The uteri were placed in 0.9% sodium chloride solution and mixed in a Waring Blender for five seconds and then filtered through three layers of cheesecloth. The eggs were collected and refrigerated in a solution of 0.9% NaCl and 2% formalin as described for Ascaris suum by Jaskoski (1952). Eggs were incubated at 31 degrees C. in one liter Erlenmeyer flasks. After 21 days' incubation eggs were stored in the refrigerator for later use.

Gerbil Infection

Forty weanling gerbils, Meriones unguiculatus (24 females and 16 males) of 23-75 grams body weight were obtained from West Jersey Biological Supply, Wenonah, New Jersey. The gerbils were maintained on Gerbil Chow from

Scientific Animal Feed and were provided with water an libitum. Food and water were withheld for 48 hours before inoculating the gerbils. The same batch of eggs were used for all animal infections. The number of viable eggs per ml inoculation was counted with the aid of a dissecting microscope at 30X magnification. Gerbils were inoculated with a 20-gauge blunted needle attached to a 3-ml syringe into the stomach. An aliquot containing 6000, 4800, 3600, 3000, 2400, or 1200 infective eggs was given. Animals of both sexes were infected according to the protocol presented in Table 1. Three separate experiments were designed to trace the migration route of Baylisascaris procyonis in the gerbil and determine time of death at high dose rates.

Experiment 1. Twenty Mongolian gerbils were divided into 6 groups: 4 groups of 4, 1 group of 1 or 1 group of 2 gerbils. Gerbils in the 4 groups of 4 were infected with 1200, 2400, 3600 and 4800 infective B. procyonis eggs respectively. One gerbil was infected with 6000 B. procyonis eggs. The remaining 3 were not infected and served as controls. Symptoms of disease and short-term lethal dose observations were recorded up through the time of necropsy. Sequential necropsies were performed on various dates after inoculation from day 1 through day 17. Control gerbils were killed on day 18 or 19.

Experiment 2. Ten gerbils were infected with 3000 infective B. procyonis eggs based on the results of experiment 1 to confirm in more detail the migration route of B. procyonis and to determined symptoms of disease and possible lethal effects of this parasite at this dose level. At 12, 24 and 36 hours or 3 to 10 days after infection gerbils were sacrificed by chloroform anesthesia and examined for gross pathology and histopathology.

Table 1. Experimental Protocol for experiments on the Minimum l hal Dose of Baylisascaris procyonis in the Mongolian Gerbil (Meriones unquiculatus)

	Gerbil Tag Number	Sex	Body Weight	Number of Eggs	Time of Necropsy After Infection
EXPERIMENT 1:	1	F	38.7	1200	1 day
	2	M	28.7		2
	3	M	25.8		3
	4	F	39.4		4
	5	F	37.4	2400	5
	6	F	40.9		6
	7	F	46.8		7
	8	M	43.9		8
	9	F	37.4	3600	9
	10	F	33.9		10
	11	F	37.9		11
	12	F	43.3	4800	12
	13	F	37.4		13
	14	F	40.5		14
	15	F	40.9		15
	16	M	41.3	6000	17
	18	F	35.4	control	18
	19	M	37.3	control	19
EXPERIMENT 2:	20	F	31.9	3000	12 hr.
	21	M	37.5		24
	22	M	37.3		36
	23	M	35.9		3 days
	24	F	34.5		4
	25	F	29.0		5
	26	F	32.4		6
	27	M	35.9		7
	28	M	27.9		8
	29	M	33.1		9
	30	F	37.6		10

Table 1. Continued.

EXPERIMENT 3:	31	F	35.1	4800	min lethal dose predicted within 21 days
	32	M	35.1		
	33	F	40.3	6000	
	34	F	30.4		
	35	F	36.3		
	36	M	36.3		
	37	M	36.3	control	
	38	F	38.5	control	
	39	F	26.3	control	
	40	F	36.1	control	

Experiment 3. A third experiment was designed to further study lethal effects of B. procyconis at dose levels of 4800 and 6000 infective B. procyonis eggs. Ten gerbils were divided into 1 group of 2 and 2 groups of 4 gerbils. Gerbils in the group of 2 were infected with 4800 infective B. procyonis eggs. One group of 4 gerbils was infected with 6000 eggs. The other group of 4 was not infected and served as controls. Gerbils were held until death occurred.

Dissection

Approximately one-half of the brain, lung, liver and examined by making press preparations of the fresh tissue were removed. The other one-half were used for paraffin sections and were fixed in 10% formalin and examined after staining with hematoxylin and eosin. Both the press preparation and histolic sections were examined under high power (450X) magnification for the larvae of Baylisascaris procyonis.

Paraffin Infiltration and Embedding

After sections of the liver, lung and brain had remained in 10% formalin for 24 hours to allow complete penetration, they were cut into pieces of about 1 cubic cm. Two changes of 95% alcohol for one hour and twenty minutes each were used for dehydration of tissues. Two changes of acetone and chloroform for one hour and twenty mintues each were necessary to remove alcohol before saturating the tissue with paraffin.

Tissues were then placed in melted paraffin (melting point, 56 C.) with two changes after one hour and twenty minutes. After the last change of paraffin, a mold with new paraffin was cast and cooled in the refrigerator. The tissue was

sectioned at 5um and warmed in a tissue float bath filled with distilled water, then affixed to a previously albuminized slide.

Staining and Mounting

Paraffin was removed from the mounted sections by transferring to the following: (1) xylene (2 minutes), (2) xylene (2 minutes), (3) absolute alcohol (2 minutes), (4) 95% alochol (2 minutes), (5) 95% alcohol (2 minutes), (6) water. The tissue was then stained with Hematoxylin for three minutes and in Eosin for 60 seconds. Next, the section was rinsed in 95% alcohol and passed through the following: (1) 95% alcohol (2 minutes), (2) 95% alcohol (2 minutes), (3) absolute alcohol (2 minutes), (4) absolute alcohol (2 minutes), (5) xylene (2 minutes), (6) xylene (2 minutes). The section was mounted with a drop of Balsam and a Coverslip.

RESULTS

Baylisascaris procyonis Prevalence Studies

Fifty-eight raccoons were examined during the period November, 1981 through January, 1982 (Year I). Thirty-nine were positive (67%) for Baylisascaris procyonis. The infections were light and the mean worm count was 16. Twenty-five of the 39 raccoons found positive for B. procyonis contained less than 20 worms.

In the second survey year, thirty-nine of 49 (71.4%) raccoons collected in DeKalb County, Illinois, from November 1982 through January 1983 were positive for B. procyonis. The mean worm count found in Year II was 39.8 (range of 3 to 171).

In both study years the prevalence of B. procyonis for males was not statistically greater (P = 0.025)than for female raccoons. In Year II, of thirty females, 70% were positive for B. procyconis, while 64% of twenty-eight males were positive (Table 2). The prevalence of B. procyonis in raccoons collected in Year I and II from Northern Illinois increased from 67% in 1981 to 71.4% in 1982. Dog heartworm was not found in any of the hearts examined.

In Year I, a higher prevalence of B. procyonis was found in lighter weight raccoons and a gradual reduction in infection prevalence was associated with increasing body weight (Table 3). B. procyonis was found in 3 of 3 raccoons (100%) weighing less than 4260 grams, 11 of 14 (79%) weighing 4261 to 5861 grams, 9 of 11 (82%) weighing 5862 to 7462 grams, and 3 of 8 (37.3%) weighing greater than 7463 grams. The maximum count of 56 worms was found in two

Table 2. Comparison of Results of the 1981 and 1982 Surveys of the Prevalence of Baylisascaris procyonis in Raccoons in Northern Illinois

	Number Examined		Number Infected		Percent Infected	
	1981	1982	1981	1982	1981	1982
Male	28	27	18	21	64	77.7
Female	30	22	21	14	70	63.6
Total	58	49	39	35	67	71.4

males weighing 6560 grams and 7300 grams. Both measured greater than 580 millimeters in body length. A higher prevalence was found, 16 of 17 (94%), in raccoons with a body length of 506-571 millimeters (Table 4). Body weight and body length were measured in Year I only.

Gerbil Infection Studies

Experiment 1. Mortality at particular dose levels are shown in Table 5. The experimental design did not allow for the pathogenic effects of infection progress to death in gerbils infected with low doses of B. procyonis eggs. Only gerbils which showed symptoms of CNS injury died before the scheduled necropsy date. The only exceptions were Gerbil 15 which displayed CNS injury but was necropsied before spontaneous death, and gerbil 16, which died of pulmonary sumptoms at 3 days after infection. At dose levels of 3600, 4800 and 6000 eggs, 3 gerbils demonstrated CNS injury and death occurred within 2 weeks. There was no remission of signs or CNS injury once they appeared.

Obvious symptoms of disease was noted in 4 gerbils. There was a decrease in activity in all 4 of the gerbils beginning day 3 after infection. In 3 gerbils (No. 11, 13 & 17) loss of coordination and circling was observed on day 8. Gerbil 15 exhibited similar behavior patterns on day 14. On the 11th day after infection one gerbil was found dead and 2 showed symptoms of CNS injury, the latter not appearing until day 15 in gerbil 15. No obvious signs were noted in gerbils that did not die in spite of infection.

Experiment 2. Numerous small hemorrhagic foci on the small intestine and liver were observed in 3 gerbils examined twelve to thirty-six hours after infection with infective B. procyonis eggs. A few hemorrhagic foci were present on the lungs of one gerbil that was necropsied on day 3. The number and degree of

Table 3. Prevalence of Infection with Baylisascaris procyonis in Raccoons of Various Body Weights from Northern Illinois in November 1981 through January 1982*

Weight (gram)	Number Infected	Number Examined	Percentage Infected
2660-4260	3	3	100.0
4261-5861	11	14	79.0
5862-7462	9	11	82.0
7463-	3	8	37.3
Total	26	36	72.2

*There were 22 Raccoons of unknown body weight examined, of which 59% were found to be infected.

Table 4. Prevalence of Infection with Baylisascaris procyonis in Raccoons of Various Body Lengths from Northern Illinois in November 1981 through January 1982*

Length (mm)	Number Infected	Number Examined	Percentage Infected
440-505	2	3	67.0
506-571	16	17	94.0
572-637	10	16	63.0
Total	28	36	77.7

*There were 22 Raccoons of unknown body length examined, of which 59% were found to be infected.

Table 5. Summary of Mortality in Baylisascaris procyonis-infected Gerbils in Experiment 1.

Gerbil Tag Number	Number of Eggs	Ave. Eggs per Grams BW	Day signs of Disease Appeared	Day of Necropsy
1	1200	40	-	1
2			-	2
3			-	3
4			-	4
5	2400	61	-	5
6			-	6
7			-	7
8			-	8
9	3600	100	-	9
10			-	10
11			CNS-Day 11	11
12			-	12
13	4800	110	CNS-Day 12	Died Day 12
14				13
15			CNS-Day 15	15
16			Pulmonary	Died Day 3
17	6000	1600	CNS-Day 12	Died Day 12
18	control			18
19	control			19
20	control			

hemorrhagic foci in the lungs increased from the 4th to 8th day after infection. Larvae were found in sections of lung from 5 infected gerbils from day 6 through day 10. The larvae were oval in shape with a mean cross-sectional diameter of 20um by 17um (Figure 1). Hemorrhagic foci were visible on the surface of the brain of 5 gerbils from day 6 through day 10. Larvae were found on day 10 in the brain and had a mean cross-sectional diameter of 29um by 21um (Figure 2). No obvious symptoms of CNS injury were noted in any of 10 gerbils infected with 3000 B. procyonis eggs within 10 days after infection.

Experiment 3. All gerbils demonstrated CNS injury at high dose levels of 4800 and 6000 infective B. procyonis eggs. Death occurred at the 17th or 18th day after infection as shown in Table 6. Only symptoms of disease and day of death are recorded for the infected gerbils. Control gerbils showed no signs of disease.

All experimental animals lost between 0.7-26.2 grams body weight during infection periods. By contrast, body weight gain of controls ranged from 8.2-27.5 grams. Gross pathology and histological study were also performed on all control gerbils, no obvious signs of disease were noted as described for the infected gerbils.

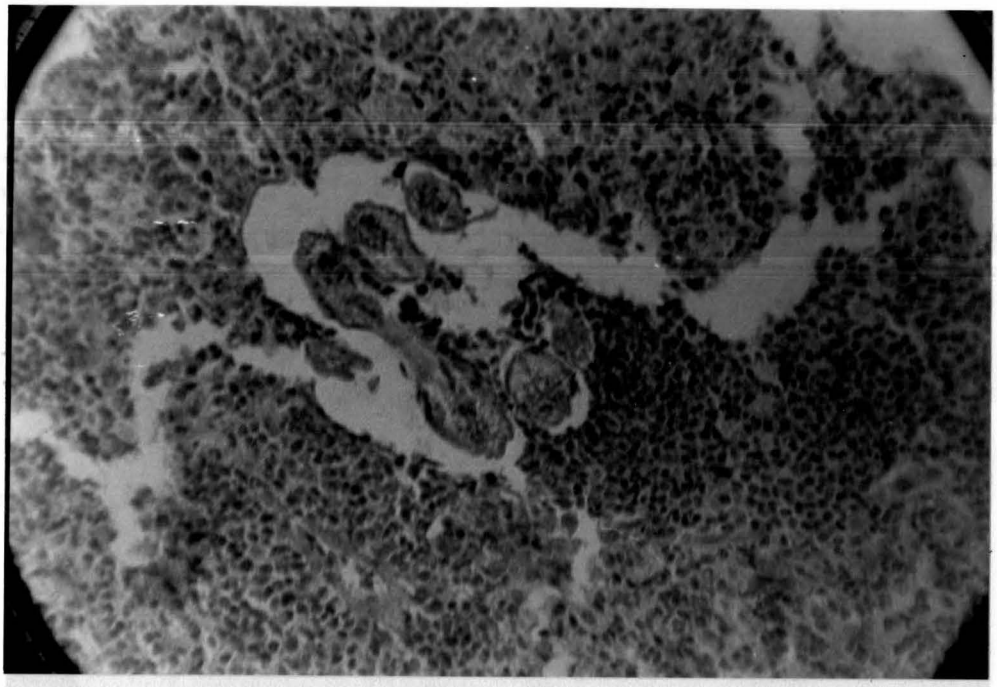

Fig. 1. Larvae of Baylisascaris procyonis in a section of the lung of a gerbil necropsied on the 6th day after infection. Notice increased cellularity in the spaces and influx of neutrophils and macrophages. H & E stain; 250X.

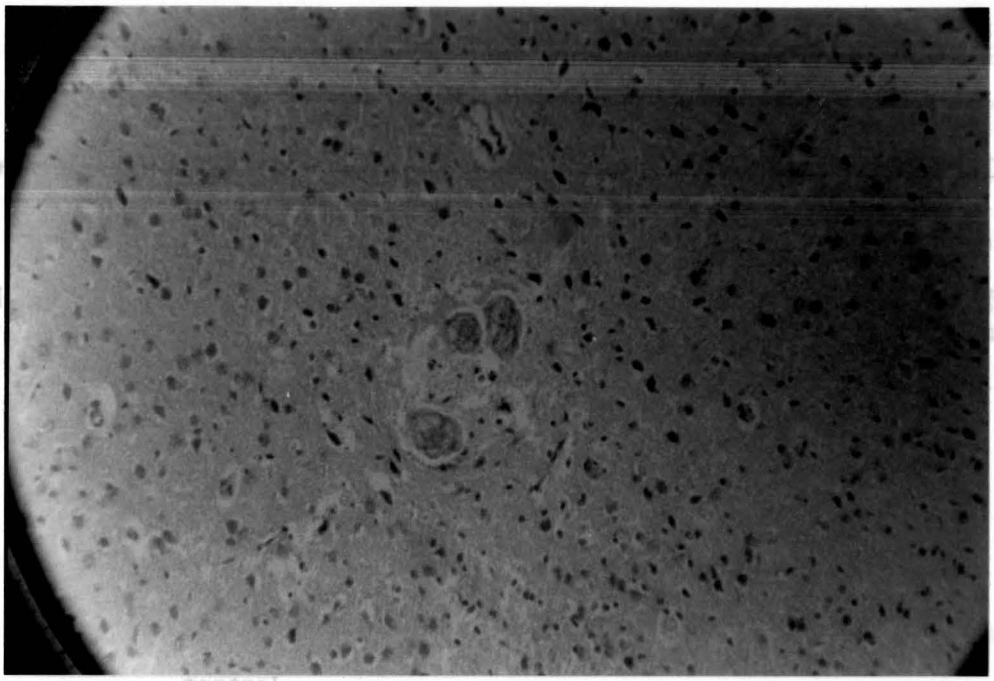

Fig. 2. Larvae of Baylisascaris procyonis in a section of the brain of a gerbil necropsied on the 10th day after infection. Notice malacia and influx of macrophages and neutrophils. H & E stain; 100X.

Table 6. Summary of Mortality in Baylisascaris procyonis-infected Gerbils in Experiment 3.

Gerbil Tag Number	Number of Eggs	Ave. Eggs per Grams BW	Day signs of Disease Appeared	Day of Spontaneous Death
31	4800	110	CNS-Day 13	17th
32			CNS-Day 14	18th
33	6000	116	CNS-Day 14	17th
34			CNS-Day 14	18th
35			CNS-Day 13	18th
36			CNS-Day 13	17th
37	control			
38	control			
39	control			
40	control			

DISCUSSION

Baylisascaris procyonis Prevalence Studies

Baylisascaris procyonis is a common parasite of raccoons, varying from about 35-90% prevalence in local populations. in this survey of more than 107 raccoons from DeKalb and surrounding counties in Illinois, about 69% were found to be positive. These and previous results suggest that the prevalence of infection is high in certain populations of raccoons and maybe increasing in certain areas with infected raccoons being found as often in urban areas as in rural localities.

Solomon (1969) pointed out that in many experimental nematode infections male animals are more susceptible than females. In both study years no difference in preferential susceptibility of male or female raccoons to B. procyonis was found.

In Year I more worms were recovered from lighter weight raccoons of medium body length. Possibly length and body weight are related to age as young raccoons become infected directly, by ingestion of infective eggs contaminating their mother's body, or from the local environment of the den since Snyder and Fitzgeral (1979) found more than 90% of juvenile raccoons infected with B. procyonis. Both greater age and a better nutritional state would be expected to increase resistance to parasitism in various ascarid host/parasite systems.

Gerbil Infection Studies

After ingestion of the eggs by the gerbils, the eggs hatch, the larvae penetrate the intestines and migrate to the liver. Grossly visible hemorrhages were seen on the surface of the small intestine and liver an necropsy beginning at 12 hours post infection. Lesions were found in lesser numbers beginning 36 hours. After 4 days hemorrhagic foci began to appear in the lungs and increased up to the 8th day. Larvae were found in two gerbils on the 6th day. From the lungs they migrate to the brain as early as the 6th day. The first appearance of symptoms of CNS injury in gerbils that ultimately died correlated with the appearance of hemorrhagic foci seen on the surface of the brain between the 6th and 10th days. However, the brain tissue cells showed little or no reaction toward the larvae. It would seem that host resistance might destroy larvae in the early days of infection when the larvae are small and not readily observed. Evidence is 6 larvae in a histologic section of one gerbil at 10 days.

Minimum Lethal Dose Studies

The Minimum lethal dose varies depending on the animal species, its size, and the migratory behavior of the parasite in it, especially to the brain. The lethal dose of B. procyonis in these experiments in gerbils was determined to be 100 eggs per gram of body weight (EPG), or four times more than the 400 EPG lethal dose of A. suum in hamsters as reported by Jaskoski (1960). Dubey (1982) reported that as few as 2 B. procyonis were fatal to one of 6 white mice. Although Dubey did not determine the lethal dose for mice, his results showed that infection with B. procyonis was a thousand times more virulent in mice than

in gerbils. In contrast, the lethal does of A. suum in guinea pigs was reported by Kerr (1938) to be 35 EPG, or eleven times more virulent in guinea pigs that the 400 EPG lethal dose of A. suum in hamsters.

SUMMARY AND CONCLUSIONS

Seventy-four of 107 (69.1%) raccoons (Procyon lotor) collected in Northern Illinois from November 1981 through January 1982 and November 1982 through January 1983 were positive for B. procyonis. A significant difference in preferential susceptibility of male or female raccoons to B. procyonis was not demonstrated (P = 0.025). The number of worms found ranged from 3 to 171, with a mean of 39.8 worms per host.

Three experiments were used to trace the migration route of B. procyonis in Mongolian gerbils (Meriones unguiculatus) and the possible lethal effects it had within 21 days post infection. In experiment one, 17 gerbils were infected with 1200, 2400, 3600, 4800 or 6000 B. procyonis eggs and one gerbil was sacrificed each day for 15 consecutive days and examined for larvae. In experiment two, 10 gerbils infected with 3000 eggs were used to confirm in more detail the migration route of B. procynois and observe histological changes in the tissues. In experiment three, 6 gerbils were given high dose levels of 4800 and 6000 eggs and monitored until death. Seven gerbils served as controls for all the experiments.

Results of these experiments indicated that larvae migration in gerbils was typical of that reported for B. procyonis in other abnormal hosts. Gerbils infected with dose levels of 3600 or more eggs developed CNS injury and died within 21 days post infection. Only those that showed symptoms of VLM died with one exception, one gerbil died at 3 days after showing pulmonary signs.

These results and previously published reports on the minimum lethal dose suggest that larger animal host species require larger dosages, in the range of 500 to several thousand or more eggs ingested. The lethal dose of B. procyonis in these experiments in gerbils was determined to be 100 EPG, or a thousand times more than the 2 EPG lethal dose in mice.

LITERATURE CITED

1. Barnstable, R.W., Dyer, W.G., 1974: Gastrointestinal helminths of the raccoons, Procyon lotor, in southern Illinois. Transactions, Ill. State Acad. Sci. 67: 451-460.

2. Dubey, J.P., 1982: Baylisascaris procyonis and eimerian infections in raccoons. J. Am. Vet. Med. Assoc. 181: 1292-1294.

3. Guyer, M.F., 1956: Animal Microbiology. Chicago : University of Chicago Press.

4. Hoffmeister, O.F., 1972, Mohr CO: Fieldbook of Illinois Mammals. New York : Dover Publications, Inc. p. 93-95.

5. Jacobson, H.A., Scanlon, PF, Nettles, VF, 1976, et al : Epizootiology of an outbreak of cerebrospinal nematodiasis in cottontail rabbits and woodchucks. J. Wildl. Dis. 12 : 357-360.

6. Jacobson, J.E., 1982: Prevalence of Baylisascaris Procyonis in harvested Indiana raccoons. In Proceedings. Ann. Midw. Conf. Parasitol, p. 13.

7. Jaskoski, B.J., 1960: Relation of age and fat reserve in swine ascarid eggs to infectivity in the hamster. Exp. Parasitol. 10 : 333-336.

8. Jaskoski, B.J., 1952: The protein coat in the development of A. lumbricoides eggs. Exp. Parasitol. 1 : 291-302.

9. Kazacos, K.R., 1981: Animal and public health implications of the nematode genue Baylisascaris. in Proceedings. 26th Meet. Amer. Assoc. Vet. Parasitol., p. 21.

10. Kazacos, K.R., 1983. Raccoon Roundworms (Baylisascaris procyonis). A cause of animal and human disease. Station Bulletin. 422: p. 13.

11. Kazacos, K.R., Appel GO, Thacker, H.L., 1981: Cerebrospinal nematodiasis in a woodchuck suspected of having rabies. J. Amer. Vet. Mad. Assoc. 179 : 1102-1104.

12. Kazacos, K.R., Reed, W.M., Thacker, H.L., Winterfield, R.W., Dhillon, A.S.,: 1982 Epizootiology of outbreaks of cerebrospinal nematodiasis in bobcats and rabbits. In Proceedings, 62nd CRWAD, p2.

13. Kazacos, K.R.; Wirtz, W.L., Burger, P.P., 1981 et al : Raccoon ascarid larvae as a cause of fatal central nervous system disease in subhuman primates. J. Amer. Vet. Med. Assoc. 179 : 1089-1094.

14. Kazacos, K.R., Kazacos, E.A., Render, J.A., Thacker, H.L., 1982: Cerebrospinal nematodiasis and visceral larve migrans in an Australian (Latham's) brush turkey. J. Amer. Vet. Med. Assoc. 181 : 1295-1298.

15. Kerr, J.B., 1938: The cellular response in acquired resistance in guinea pigs to an infection with pig Ascaris. Amer. J. Hygiene 27 : 28-51.

16. Richardson, J.A., Kazacos, K.R., Thacker, H.L., 1980, et al : Verminous encephalitis in commercial chickens. Avian Dis. 24 : 498-503.

17. Reed, W.M., Kazacos, K.R., Dhillon, A.S., 1982, et al : Cerebrospinal nematodiasis in bobwhite quail. Avian Dis. 25 : 1039-1046.

18. Schueler, R.L., 1973: Cerebral namatodiasis in a red squirrel. J. Wildl. Dis. 9 : 58-60.

19. Solomon, G., 1969. Host hormones and parasitic infection. Int. Rev. Trop. Med. 8 : 101-158.

20. Snyder, D.E., Fitzgerald, P.R., 1979: Helminths parasites of the raccoon from Illinois. 55th Meet. Amer. Soc. Parasitol.

21. Sprent, J.F.A., 1952: On the migratory behavior of the larvae of various Ascaris species in white mice. J. Inf. Dis. 90 : 165-176.

22. Sprent, J.F.A., 1968: Notes on Ascaris and Toxocaris, with a definition of Baylisascaris gen. nov. Parasitology 58 : 185-198.

23. Stains, H.J., 1956: The raccoon in Kansas -- Natural history, management and economic importance Univ. of Kansas Mus. Nat. His. and State Biol. Survey Misc. Publ. 10 : 76.

24. Tiner, T.D., 1949: Preliminary observations of the life history of Ascaris colunmaris. J. Parasitol. 35 (supplement) : 13.

25. Tiner, T.D., 1951: Observations on larval Carnivore ascarids in rodents. J. Parasitol. 37 (supplement) 21-22.

26. Tiner, T.D., 1952: The migration, distribution in the brain and growth of ascarid larvae in rodents. J. Inf. Dis. 92 : 105-113.

27. Tiner, T.D., 1954: The fraction of peromyscus Leucopus fatalities caused by raccoon ascarid larvae. J. Mamm. 35 : 589-592.

APPROVAL SHEET

The thesis submitted by Diane Mary Sudduth
has been read and approved by the following committee:

Dr. Benedict J. Jaskoski, Director
Professor, Biology, Loyola

Dr. Edward E. Palinscar
Professor, Biology, Loyola

Dr. Clyde Robbins
Associate Professor, Biology, Loyola

The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the thesis is now given final approval by the Committee with reference to content and form.

The thesis is therefore accepted in partial fulfillment of the requirements for the degree of Master of Science.

4/18/84
Date

B J Jaskoski
Director's Signature