the worm migrated to the eye just prior to being observed there. It may be noteworthy that nasal swabs taken at the time the worm was removed were negative for *Anatrichosoma* eggs. However, three of five other cynomolgus monkeys in the same group were positive for eggs by nasal swab. The parasite in question may have undertaken an aberrant migration in search of other worms.

The specimen was deposited in the National Parasite Collection as USNM Helm. Coll. 76603.

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Research Note

Contaminative Ability of *Baylisascaris procyonis* Infected Raccoons in an Outbreak of Cerebrospinal Nematodiasis

The ascarids, *Baylisascaris procyonis* and *B. columnaris*, of the raccoon (*Procyon lotor*) and skunk (*Mephitis mephitis*), respectively, are important causes of cerebrospinal nematodiasis and visceral larva migrans in various mammals and birds (Nettles et al., 1975, J. Am. Vet. Med. Assoc. 167:600–602; Sass and Gorgacz, 1978, J. Am. Vet. Med. Assoc. 173:1248–1249; Richardson et al., 1980, Avian Dis. 24:498–503). In naturally occurring cases of fatal central nervous system (CNS) disease due to these parasites few larvae were seen or recovered from the brain, despite the large number which were encysted elsewhere in the body. This further demonstrated the pathogenetic capabilities of individual larvae when they enter the CNS, as first determined by Tiner (1953, J. Infect. Dis. 92:105–113) and Sprent (1955, Parasitology 45:41–55). There is no information available, however, on the infective dose or length of exposure necessary to produce the clinical manifestations which were seen. Also, data on the fecundity of these parasites are lacking. This and the contaminative ability of infected animals are important from an animal and public health standpoint. In a recent extensive outbreak of fatal CNS disease in bobwhites (*Colinus virginianus*) (Reed et al., Avian Dis., in press), we had the unique opportunity to examine these parameters as they related to naturally occurring cerebrospinal nematodiasis. These considerations are the subject of this report.

In that outbreak 85 bobwhites had access to a 12 × 24 ft dirt-bottomed run.

Three young pet raccoons had been previously housed in the run for 3 mo. The birds were placed in the enclosure 6 weeks after the raccoons had been removed. The birds had access to the run for 2–3 mo, and all died of severe CNS disease beginning 3–4 weeks after introduction to the area. *Baylisascaris* larval migration was diagnosed histologically as the cause of death. Larvae were recovered from brains by Baermannization. The three raccoons were positive for *B. procyonis* eggs. Over 10,000 larvated *B. procyonis* eggs were recovered from 1,500 g of soil in the run (Reed et al., ibid.). These eggs caused 100% mortality by cerebrospinal nematodiasis in experimentally inoculated hamsters and mice (Kazacos, unpublished data).

To examine *B. procyonis* egg production in the raccoons, quantitative fecal flotations were performed (McMaster method). Individual fecal samples were collected and weighed. The raccoons were shedding *B. procyonis* eggs at rates of 5,400, 1,700, and 1,300 eggs per gram (epg) of feces, respectively (means of four determinations per animal). Eleven individual fecal samples were collected; the 5,400 epg animal averaged 106.4 g of feces per defecation (four samples), the 1,700 epg animal averaged 99.0 g (four samples), and the 1,300 epg animal averaged 93.5 g (three samples). The animals were fed a diet of dry dog food ad libitum. The owner stated that they defecated at least twice a day, which was similar to my experience with wild raccoons kept in the laboratory on a similar diet.

Using these rates and a twice daily defecation as a basis, the animals were shedding 1,149,120 eggs, 336,600 eggs, and 243,100 eggs per day, respectively (total 1,728,820 eggs per day). Over the 3-mo period, they would thus have contaminated the run with a total of 155,593,800 eggs. All of the eggs would have been potentially infective to the birds, since infectivity (second stage larva) is reached in 30 days (Lindquist, 1978, Am. J. Vet. Res. 39:1868–1869; Kazacos et al., 1981, J. Am. Vet. Med. Assoc. 179, in press). Based on this exposure, and the pathogenicity of the parasite, it was essentially inevitable that the birds would develop clinical disease in this situation. Following anthelmintic treatment using piperazine monohydrochloride at a dosage of 170 mg/6 lb body weight, 15 female and 10 male *B. procyonis* were recovered from the raccoons. Based on the total egg production, average fecundity of one *B. procyonis* female in this case approximated 115,000 eggs per day. These estimates are based on the assumption that egg production was constant, which is probably not true. However, egg production per animal was probably higher than this prior to the time they were examined, since some loss of worms took place over the 3-mo period (Reed et al., loc. cit.).

This report indicates that infected raccoons can shed very large numbers of *B. procyonis* eggs. Other animals may thus be exposed to extremely high numbers due to accumulation. The potential for accidental human infection must also be considered when large numbers of eggs are present. Although no human cases with *B. procyonis* have as yet been identified, human infection has been predicted by others (Sprent, 1965, Trans. R. Soc. Trop. Med. Hyg. 59:365–366; Beaver, 1969, J. Parasitol. 55:3–12). Kazacos et al. (1981, loc. cit.) recently reported 100% mortality in squirrel monkeys experimentally inoculated with *B. procyonis* eggs. Extensive visceral and ocular larva migrans and cerebrospinal nematodiasis oc-

curred in the subhuman primates in that study, indicating probable human susceptibility to infection by *B. procyonis*.

The present outbreak involved artificial concentration of raccoons and their feces, which is potentially a very serious situation for both animals and man. In the natural situation with wild raccoons, however, similar levels of contamination undoubtedly occur in certain areas, especially in areas with high population densities of infected raccoons. In an outbreak of CNS disease in commercial poultry linked to wild raccoons, Richardson et al. (1980, loc. cit.) documented the death of 622 birds following a single exposure. Jacobson et al. (1976, J. Wildl. Dis. 12:357–360), investigating an outbreak of CNS disease in rabbits and woodchucks, found a massive number of larvae in the viscera of one affected woodchuck, and a heavily infected wild raccoon shedding 25,750 epg. The interaction of these parasites and their hosts with wild and domestic animal populations and man deserves further attention.

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Research Note

Polydelphis (Nematoda: Ascarididae) Larvae Encysted in a Feral African Green Monkey (*Cercopithecus aethiops*)

An adult feral African green monkey (*Cercopithecus aethiops*) from east Africa, that had been housed at the Delta Primate Center for 1 year, presented with an apparent intestinal obstruction. The animal was subjected to an exploratory laparotomy for removal of the obstruction. Incidental to this, numerous (25–50) small nodules scattered diffusely throughout the mesentery and omentum were observed. They were irregularly round, 1–2 mm in size, white and firm. A small piece of mesentery containing nodules was removed and fixed in 10% formalin.

Microscopically, the nodules consisted of granulomas which were composed of a thin fibrous capsule, a band of lymphocytes, and an inner zone of large foamy macrophages. Occasional multinucleate giant cells and neutrophils were present. In the center of each granuloma was a single nematode larva (Fig. 1A). In tissue section the worms were observed to have a muscular esophagus, an intestine