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National Sciences and Engineering Research Council of Canada. Specimens of the leech, trypanosome and piroplasm have been deposited in the Invertebrate Section of the National Museum of Canada in Ottawa, Ontario K1A 0M8, Canada, and assigned accession numbers NMCIC1984-0787 and NMCPC1984-0790 through NMCPC1984-0792. Marine Sciences Research Laboratory Contribution Number 536.

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Hepatic Capillariasis in African Giant Rats (*Cricetomys gambianus* Waterhouse)

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Diseases of wild rodents in Africa have not been studied extensively. There are only a few reports on parasitic diseases in the African giant rat (Dipeolu and Ajayi, 1975, East Afr. Wildl. J. 13: 85–89; Ikede and Ajayi, 1976, J. Nigerian Vet. Med. Assoc. 5: 63–65). Even though hepatic capillariasis has been reported in numerous species of wildlife from many countries including wild rodents (Reynolds and Gavutis, Jr., 1975, J. Wildl. Dis. 11: 13), the only report of the parasite in Nigerian wildlife was that of Ikede and Ajayi (1976, op. cit.) in a captive African giant rat. African giant rats are easily domesticated and have potential for supplementing the scarce protein supply for humans in Nigeria.

The purpose of this paper is to document the occurrence of and describe the lesions associated with hepatic capillariasis in free-living African giant rats trapped in Zaria, Nigeria.

Twenty-one young and adult wild African giant rats were captured within the period of 1 yr (January 1982 to January 1983) in live-traps set in various locations in Zaria, Nigeria. Within 12–24 hr of capture, each animal was examined at necropsy. Tissue specimens were taken from the liver and were fixed in 10% buffered formalin. After embedding in paraffin, sections were made at 5 μm and stained with hematoxylin and eosin and trichrome stain. Portions of the liver were teased, mixed with normal saline on a glass slide and examined with a light microscope.

Seven of the 21 rats showed hepatic lesions of similar pattern which ranged from mild to severe. Gross examination commonly showed an enlarged liver. The lesions consisted of white to yellow nodules which on measurement ranged from 1 to 5 mm in diameter on the liver surface. The areas of the liver showing depressed streaks were firmer and less easily sectioned with a knife than the apparently normal areas of the organ. Portions of the liver teased and examined as a wet preparation under the microscope showed the presence of numerous ovoid-shaped eggs with bipolar caps. The eggs measured between 55 and 57 μm in length and 30 μm at the widest diameter and showed radial

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striations on the shell, all of which are characteristic of *Capillaria hepatica* Bancroft, 1893.

On histological examination, the liver sections from all the affected rats showed multifocal granulomatous areas characterized by the presence of macrophages, lymphocytes and plasma cells and associated with fibrous connective tissue proliferation. In some granulomas foreign body giant cells formed part of the cellular exudate. In two cases sections of the liver showed in addition to the lesions described above several cross and tangential sections of adult *Capillaria* sp. and each of these was surrounded by numerous eggs of the parasite, macrophages, neutrophils and lymphocytes.

Most of the findings in the present case agree with those of Ikede and Ajayi (1976, op. cit.); however, in addition we detected adult *Capillaria* sp. in histologic sections of the livers of two rats. None of the wild rodents with capillariasis showed any visible ante-mortem clinical signs of disease even though in some the liver was involved extensively.

Voucher specimens have been deposited in the U.S. National Parasite Collection (Beltsville, Maryland 20705, USA) and assigned USNM Helm. Coll. No. 78173.

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Elaeophora schneideri Wehr and Dickmans, 1935 in White-tailed Deer from the Edwards Plateau of Texas

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The arterial nematode, *Elaeophora schneideri*, was first reported from white-tailed deer (*Odocoileus virginianus* (Zimmermann)) in Arizona (Hibler and Adcock, 1968, J. Parasitol. 54: 1095–1098). Since then it has been recovered from this host in Florida, Georgia, Oklahoma, and South Carolina (Prestwood and Ridgeway, 1972, J. Wildl. Dis. 8: 233–236; Hibler and Prestwood, 1981, Filarial nematodes of white-tailed deer, *In* Diseases and Parasites of White-tailed Deer, Davidson et al. (eds.), Tall Timbers Res. Sta., Tallahassee, Florida, pp. 351–362). In Texas, *E. schneideri* has been recovered from white-tailed deer (Foreyt and Foreyt, 1979, J. Wildl. Dis. 15: 55–56), Barbary sheep (*Ammotrogus lervia* Pallas) (Pence and Gray, 1981, J. Wildl. Dis. 17: 49–56), mule deer (*Odocoileus hemionus hemionus* (Rafinesque)) (Pence and Gray, 1981, op. cit.), and sika deer (*Cervus nippon* Temminck) (Robinson et al., 1978, J. Wildl. Dis. 14: 137–141).

Clinical disease due to arterial worm has been noted in Barbary sheep and sika deer (Pence and Gray, 1981, op. cit.; Robinson et al., 1978, op. cit.) and white-tailed deer have been suggested as a reservoir host for the infection in Texas (Robinson et al., 1978, op. cit.). This study was initiated to determine (1) the prevalence of *E. schneideri* in white-tailed deer from the Texas Edwards Plateau and (2) the potential for

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