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RESEARCH NOTES/CASE REPORTS

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Serologic Survey for Selected Viruses in a Population of Raccoons, *Procyon lotor* (L.), in the Great Smoky Mountains

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Several viral pathogens of humans and domestic animals have been detected in raccoons within the United States of America. This has generated concern since raccoons continue to thrive despite human modification of the environment (Johnson, 1970, Agric. Exp. Sta. Auburn Univ. Bull. 402, 148 pp.) and in some instances do as well in suburban and residential communities as in rural and forested areas (Hoffman and Gottschang, 1977, J. Mammal. 58: 623–646). Epizootics of viral diseases have been reported in both urban (Hoff et al., 1974, J. Wildl. Dis. 10: 423–428) and rural (Keeler, 1978, M.S. Thesis, Univ. of Tennessee, Knoxville, Tennessee, 81 pp.) raccoon populations.

This study was conducted in Cades Cove, an area of the Great Smoky Mountains National Park in Tennessee which is protected, unmanaged and relatively isolated by mountains on three sides. There are tourist facilities and a campground on the extremities of the cove, thus the raccoons maintain some degree of contact with humans and domestic pets. In addition, 50–100 cattle are permitted to graze within the cove. The area also has a wildlife community which includes striped skunks (*Mephitis mephitis*), opossums (*Didelphis virginiana*), black bears (*Ursus americanus*), white-tailed deer (*Odocoileus virginianus*), feral swine (*Sus scrofa*), bobcats (*Felis rufus*), muskrats (*Ondatra zibethicus*), mink (*Mustela vison*), red foxes (*Vulpes vulpes*) and gray foxes (*Urocyon cinereoargenteus*). Our objective was to determine by serologic survey whether certain viral infections were present which would affect the raccoon population and could serve as potential sources of infection for other wildlife, humans and domestic animals.

The five viruses selected for this study have been reported to infect raccoons and included rabies virus (RV) (Kappus et al., 1970, J. Wildl. Dis. 6: 507–509), canine distemper virus (CDV) (Robinson et al., 1957, J. Am. Vet. Med. Assoc. 13: 276–278), canine hepatitis virus (CAV1) (Cabasso, 1970, *In* Infectious Diseases of Wild Mammals, 1st ed., Davis et al. (eds.). Iowa State Univ. Press, Ames, Iowa, pp. 134–139), pseudorabies virus (PRV) (Kirkpatrick et al., 1980, J. Wildl. Dis. 16: 601–615), and a parvovirus related antigenically to canine parvovirus (CPV) (Nettles et al., 1980, J. Am. Vet. Med. Assoc. 177: 787–789).

Raccoons were live-trapped from September 1979 through October 1980 and anesthetized with ketamine hydrochloride (Bristol-Myers Co., Syracuse, New York 13201, USA). Ages were determined by wear of the molars as described by Grau et al. (1970, J. Wildl. Manage. 34: 364–372). Blood samples were drawn from the femoral vein of each raccoon and were allowed to clot at room temperature for several hours before serum was harvested.

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Serum samples were stored at −70 C until tested.

The cell cultures, antigens and antisera used for tests with CAV, PRV and CPV have been described by Potgieter et al. (1980, Am. J. Vet. Res. 41: 978–980; 1981, Can. J. Comp. Med. 45: 212–216). A modified live, Flury strain of rabies virus (Endural-R, Norden, Lincoln, Nebraska 68501, USA) was used. Antiserum to this virus was obtained from a dog 4 wk after intramuscular inoculation. Canine distemper virus (Bu strain) was obtained from M. J. G. Appel (Baker Institute, Cornell University, Ithaca, New York 14850, USA). Antiserum to this virus was prepared in rabbits as described previously (Potgieter et al., 1980, op. cit.).

The indirect fluorescent antibody test (IFAT), described previously, was used for detecting antibodies to these viruses (Potgieter et al., 1977, Am. J. Vet. Res. 38: 1341–1343). The antigens used in the test were acetone-fixed virus-infected cell cultures. Pseudorabies virus was grown in primary bovine embryonic lung cells, CDV was grown in VERO cells, CPV was grown in CRFK cells, RV and CAV1 were grown in MDCK cells. Brain impression smears made from moribund mice 14 days after intra-cerebral inoculation with RV were used also. The fluorochrome indicator used in the test was fluorescein (FITC)-labelled staphylococcal protein A (Pharmacia Inc., Piscataway, New Jersey 08854, USA).

One hundred and seventeen raccoon serum samples were tested for antibodies to five viruses. Forty-nine samples originated from male raccoons and 68 from females. Forty-three animals were 0–14 mo old; 33 were 15–38 mo old; 28 were 39–57 mo old; seven were older than 86 mo, and six were of undetermined age.

Antibodies to RV, PRV and CDV were not detected. Brain impression smears from infected mice were preferred to infected cell cultures for detecting rabies antibody since greater virus concentrations were detectable by IFAT in the former, which facilitated reading the test.

Two raccoons had low levels of serum antibody to CPV (1:20 and 1:40) and three raccoons had low serum antibody levels to CAV1 (1:20). None of these five (or the other 112) animals had signs of disease at capture. One of the five and 13 other raccoons were radiocollared and tracked for 3–12 mo and appeared healthy throughout the study.

The data from this study as well as other ecological and physiological data (Rabinowitz, 1981, Ph.D. Dissertation, Univ. of Tennessee, Knoxville, Tennessee, 133 pp.), suggested that this population was healthy and that younger animals predominate. The latter observation coincides with that made in other raccoon populations (Johnson, 1970, op. cit.). However, it could be the result of a population recovering from an epizootic if all age groups of the population had been affected by the disease. An epizootic of CDV occurred in this population in 1973 (Keeler, 1978, op. cit.). However, the virus apparently did not become enzootic within this population since none of the raccoons tested had antibodies. A rapidly and uniformly fatal virus infection is not likely to spread readily in a population nor is the virus likely to become enzootic. Virus transmission from infected animals is not likely in an infection with a very short course before death, and if the infection is always fatal, virus transmission by recovered "carriers" could not occur. This has been shown to be the case with PRV in raccoons (Kanitz et al., 1974, Proc. 78th Annu. Meet. U.S. Anim. Health Assoc., pp. 354–356). Raccoons are highly susceptible to CDV (Robinson et al., 1957, op. cit.). Therefore, it is not surprising that antibodies to CDV and PRV were not detected.

Although CDV infection has been mistaken for rabies in raccoons (Helmboldt and Jungherr, 1955, Am. J. Vet. Res. 16: 463–469), it has become evident that rac-

coons may be of increasing importance as reservoirs for RV particularly in the southeastern USA (Kappus et al., 1970, op. cit.). Absence of clinical disease and RV antibody suggests that rabies does not frequently occur in this population. Rabies virus antibody develops relatively soon in animals after exposure to the virus and is readily detectable by the IFAT (Coe and Bell, 1977, Infect. Immun. 16: 915–919).

The significance of low antibody titers found in a few animals to CPV and CAV1 is not clear. The former is associated with severe disease in dogs but antibody titers in convalescent dogs are usually high (Potgieter et al., 1981, op. cit.). Low antibody levels can be observed very early after infection (Potgieter et al., 1981, op. cit.) and conceivably after an interval of several months or years after infection. Since the IFAT does not discriminate between strains of CPV or distinguish this virus from feline panleukopenia virus (Potgieter et al., 1981, op. cit.), our results suggested that the latter was not prevalent in this population.

Studies of CAV1 in dogs and ranch foxes have been extensive but little is known about this virus in other wildlife (Cabasso, 1970, op. cit.). However, serologic surveys suggest that this virus occurs naturally in raccoons (Jamison et al., 1973, J. Wildl. Dis. 9: 2–3; and Parker et al., 1961, J. Am. Vet. Med. Assoc. 138: 437–440). Apparently the raccoons in Cades Cove are not exposed frequently to CAV1.

The low prevalence and titers of antibodies suggested that the viruses which were surveyed were probably not endemic in this raccoon population. Thus, this raccoon population did not appear to serve as an important reservoir for these viruses at the time studied.

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Microbiological Observations on Two Stranded Live Whales[1]

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Much has been written regarding strandings of cetaceans in different areas (Hall et al., 1971, J. Wildl. Dis. 7: 324–327; Duguy, 1978, Aquat. Mamm. 6: 9–13; Irvine et al., 1979, Fish. Bull. 77: 511–513) but causes for the phenomenon remain unclear (Geraci, 1978, Oceanus 21: 38–47). Microbial disease is often suggested. Unfortunately, little information is available on the types of microorganisms associated with healthy cetaceans to compare with data from debilitated animals. Clearly, more studies are needed to define which microbes are associated with both wild and recently stranded animals. It is also necessary to know if microorganisms associated with healthy or diseased animals are potentially zoonotic (Johnston and Fung, 1969, J. Occup. Med. 11: 276–277;

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