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RESEARCH NOTES/CASE REPORTS

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Rabies Virus in Salivary Glands of Raccoons (*Procyon lotor*)

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Raccoons have assumed an increasingly important role in wildlife rabies in the United States in recent years. From the 1940's until 1978 rabid raccoons were recognized as a problem only in the southeastern states of Florida and Georgia (Bigler et al., 1973, Am. J. Epidemiol. 98: 326–335). That southeastern focus has since continued to expand and now involves, in addition to Florida and Georgia, Alabama and South Carolina. In 1977–1978 index cases of a second focus of raccoon rabies appeared along the border of Virginia-West Virginia (Centers for Disease Control, 1983, MMWR 31: 592–593). This focus has expanded in geographic area and intensity at an unprecedented rate. By December 1983 four mid-Atlantic states, West Virginia, Virginia, Maryland, Pennsylvania, and the District of Columbia were involved. The number of rabid raccoons reported in this area has increased dramatically during the 7-yr period (1977–1983), from several cases per year to 1,906 cases reported in 1983.

The close association of raccoons with humans has resulted in many persons being bitten or otherwise exposed to raccoons which were later proven rabid. Because of this human exposure potential we examined 105 pairs of mandibular salivary glands taken from raccoons diagnosed as rabid by the Fairfax, Virginia, Health Department Laboratory. Using standard virus inoculation techniques (Lennette and Schmidt, 1964, Diagnostic Procedures for Viral & Rickettsial Diseases, Am. Pub. Health Assoc., 814 pp.), 0.03 ml of a supernate from a triturated salivary gland suspension was inoculated intracerebrally (IC) into 3-wk-old Swiss white mice. Six mice were inoculated with each 10-fold dilution from $10^{0.7}$ to $10^{5.7}$. Mice were held for 3 wk, examined daily, and any mice dying were tested for rabies virus.

Table 1 shows the results of the raccoon salivary gland titrations. Raccoons from Virginia shed appreciable amounts of rabies virus in salivary glands, though not as much as previously reported for raccoons (McLean, 1975, *In* Natural History of Rabies, Vol. II, Academic Press, New York, pp. 53–77), or for red foxes (*Vulpes vulpes*) (Winkler, 1975, *In* Natural His-

TABLE 1. Quantity of rabies virus in salivary glands of raccoons from the mid-Atlantic rabies outbreak, 1982–1983.

Virus titer[a]	Number of raccoons	% of total
0	20	19
≤1	11	10
>1–2	10	10
>2–3	7	7
>3–4	7	7
>4–5	17	16
>5–6	17	16
>6	16	15
Total	105	

[a] Expressed as logarithm of mouse intracerebral 50% lethal dose ($MICLD_{50}$).

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tory of Rabies, Vol. II, Academic Press, New York, pp. 3–22) in the southeastern states during the 1960's. The geometric mean titer (GMT) of virus in salivary glands tested in these earlier studies was $10^{4.90}$ in raccoons and $10^{4.09}$ in red foxes. Among the raccoons from Virginia, 81% (85/105) shed measurable amounts of virus and the GMT of virus in the salivary glands was $10^{3.21}$.

It seems clear from these data that the salivary glands of many rabid raccoons contain substantial amounts of infectious rabies virus. Bites by such animals should be regarded as potentially capable of transmitting rabies.

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Avian Pox in a Red-tailed Hawk (*Buteo jamaicensis*)

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Avian pox has been reported in at least 60 species of birds belonging to 20 different families (Kirmse, 1967, Wildl. Dis. 49). However, poxvirus infection in birds of prey is apparently uncommon (Cooper, 1969, Vet. Rec. 85: 683–684; Greenwood and Blakemore, 1973, Vet. Rec. 93: 468–470; Halliwell, 1972, J. Wildl. Dis. 8: 104–105; Moffatt, 1972, J. Wildl. Dis. 8: 161–162; Pearson et al., 1975, J. Wildl. Dis. 11: 224–228; Thiele et al., 1979, Arch. of Virol. 62: 77–82; Tantawi et al., 1981, J. Wildl. Dis. 17: 145–146; Schmeling and Docherty, 1982, *In* Workshop on Raptor Management and Biology in Alaska and W. Canada, Ladd and Schempf (eds.), U.S. Fish and Wildlife Service, Alaska Regional Office, pp. 255–262). Attempts at isolating the causal virus in falcons were made by Cooper (1969, op. cit.) and Greenwood and Blakemore (1973, op. cit.), but were unsuccessful. Later, Thiele et al. (1979, op. cit.) and then Tantawi et al. (1981, op. cit.) were successful in isolating avian poxvirus from saker falcons (*Falco cherrug*) and from a European sparrow hawk (*Accipiter nisus*), respectively. This short communication reports a pox infection in a wild red-tailed hawk.

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On 18 May 1981, an adult male red-tailed hawk was found on the U.S. Department of Energy's "Arid Land Ecology Reserve" in Benton County, Washington. The bird was incapable of flight and was extremely thin. Nodular proliferations were noted on both feet and cutaneous scab-like lesions around the beak and eyes. No other lesions were seen during the field examination.

The bird was killed in the field and submitted promptly to the diagnostic laboratory for necropsy. Tissue sections were taken from the skin above the eye, skin adjacent to the beak, right kidney, liver, adrenal, peritoneal wall, heart, aorta, spleen and lung and preserved in 10% neutral buffered formalin (NBF). Direct smears from the foot lesion, pleura, kidney, peritoneal fluid, beak, lungwash and liver were Gram stained. Samples of foot lesions, pleura, peritoneal fluid, liver, kidney, lungwash and mouth lesions were cultured aerobically on 5% sheep blood agar and MacConkey agar.

Tissues fixed in 10% NBF were paraffin embedded, sectioned at 8 μm and stained

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