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RESEARCH NOTES/CASE REPORTS

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Malignant Catarrhal Fever in a Free-Ranging Black-tailed Deer (*Odocoileus hemionus columbianus*) in California

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Although well documented in domestic cattle, malignant catarrhal fever (MCF) was not reported in North American Cervidae until 1970 (Clark et al., 1970, J. Wildl. Dis. 6: 376–383). Since then, MCF has been diagnosed in non-native cervids including Pere David's deer (*Elaphurus davidianus*), red deer (*Cervus elaphus*), sika deer (*Cervus nippon*), moose (*Alces alces*), Sambar deer (*Cervus mariannus*), eld's deer (*Cervus eldi thamin*), rusa deer (*Cervus timoensis*), barasingha deer (*Cervus duvauceli*), axis deer (*Axis axis*), reindeer (*Rangifer tarandus*), and native white-tailed deer (*Odocoileus virginianus*) and mule deer (*Odocoileus hemionus*) (Huck et al., 1961, Vet. Rec. 73: 457–465; Reid et al., 1975, Res. Vet. Sci. 18: 269–273; Sanford et al., 1977, J. Wildl. Dis. 13: 29–32; Williams et al., 1984, J. Wildl. Dis. 20: 230–232; Heuschele, 1982, Proc. U.S. Anim. Health Assoc. 86: 552–570; Hatkin et al., 1980, J. Wildl. Dis. 16: 439–443; Clark et al., 1970, op. cit.; Clark et al., 1972, J. Wildl. Dis. 8: 72–74; Altmann et al., 1973, *In* Proc. 15th Int. Symp. Dis. Zoo Anim., Berlin, pp. 41–49; and Pierson et al., 1974, Am. J. Vet. Res. 35: 523–525). These cases occurred on game farms, in zoos or other captive situations, often where the affected animals were in contact with other exotic hoofstock or domestic sheep. A disease resembling MCF was reported in captive white-tailed deer where the reservoir host could not be determined (Wobeser et al., 1973, Can. Vet. J. 14: 106–109). Major losses of captive bison (*Bison bison*) to MCF have been documented and sheep were implicated in transmission (Ruth et al., 1977, J. Am. Vet. Med. Assoc. 171: 913–917).

Malignant catarrhal fever has been reported also from numerous exotic ungulates, notably greater kudu (*Tragelaphus strepsicorus*), Indian gaur (*Bos gaurus*), and blue wildebeest (*Connochaetes taurinus*), the latter have proven to be both carriers and perinatal shedders (Boever and Kurka, 1974, J. Am. Vet. Med. Assoc. 165: 817–819; Zimmer et al., 1981, J. Am. Vet. Med. Assoc. 179: 1130–1136; Plowright, 1981, *In* Infectious Diseases of Wild Mammals, Iowa State Univ. Press, Ames, Iowa, pp. 126–146.). The purpose of this report is to describe a case of MCF in a free-ranging black-tailed deer from Nevada County, California with no history of contact with sheep or exotic ungulates.

The animal (4-yr-old female) died shortly after arrival at the Wildlife Investigations Laboratory on 26 January 1982. It had been picked up on a large cattle ranch because it was obviously ill and unable to flee. Antemortem signs included extreme depression, torticollis, subnormal temperature, poor vascular perfusion, conjunctivitis, mucopurulent nasal discharge and diarrhea.

Upon gross necropsy the mucosa of the turbinates, oropharynx and tonsils were

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swollen and reddened. Numerous proliferative lesions with central ulceration occurred along the mucocutaneous junctions of the lips and four distinct ulcers on the dorsal to lateral surface of the tongue were noted. The gingiva was red and receding from the teeth. Several lymph nodes, particularly those draining the head and neck, were swollen and reddened.

The coronary band was red and swollen, and the wall of bulbs of the heel appeared to be separating from the underlying connective tissue. Small (1–2 mm) cutaneous proliferative crusting foci were most prominent above the coronary band, along the bridge of the nose, the backs of the ears and in the perineal region.

The trachea and bronchi contained blood-tinged fluid and froth; the lungs were edematous. The pericardial sac contained excessive straw-colored fluid and several fibrin clots. Numerous pale, pinhead sized proliferative lesions were seen on the pericardium. The serosal vasculature of the small and large intestine was prominent; the spleen was mildly enlarged; and three ecchymotic hemorrhages of the renal capsule were present. Other tissues appeared normal.

Lesions in this case suggestive of epitheliotropic foreign animal virus diseases caused the author to submit tissues to the Plum Island Animal Disease Center (PIADC) for virologic and histopathologic examination. Microscopic examination revealed fibrinoid necrosis of small to medium sized renal, myocardial and lingual arteries. Lymphocytic perivascular cuffing was present about small vessels in lung and skeletal muscle. Nonsuppurative myocarditis and sarcocysts in skeletal muscle and tongue were noted. Mild lymphocytic depletion and an absence of splenic periarteriolar sheaths and germinal centers were described by one pathologist (RJY). Another pathologist noted widespread necrosis in lymphoid tissues (NJM). Both noted that histologic lesions were characteristic of MCF.

Virus isolation attempts at PIADC in suckling mice, embryonated eggs, vero cells, bovine kidney, calf thyroid and lamb testis cells failed. An inoculum from several lymph nodes, sections of lung, spleen, blood clots and trachea was prepared in tris buffered normal saline at approximately 1:10 dilution with 2% penicillin/streptomycin, macerated in a Ten Broeck tissue grinder and sonicated. The inoculum was co-cultivated with VERO African green monkey kidney cells using MEM with 10% fetal bovine serum, 1% penicillin/streptomycin and 0.1% gentamycin. A similar inoculum was added to an 80% monolayer of baby hamster kidney (BHK) cells, in MEM with 10% tryptose phosphate broth and 10 mg/ml trypsin and allowed to adsorb for 1 hr at 37 C.

Although some cytopathic effect on VERO cells was evident within the first few days, on subsequent passage this was not seen. Three adult black-tailed deer were immobilized with etorphine and acepromazine. One received 5 cc inoculum of BHK cells and tissue culture fluid intranasally; the second, a similar 5 cc of VERO inoculum intravenously; the third 5 cc of VERO cells and culture fluid intranasally. One animal died of aspiration pneumonia 72 hr after inoculation. Lesions similar to the index case were not present. No signs of disease were seen in the other two deer which remained in captivity for over 1 yr.

Blue wildebeest have been shown to be reservoirs of the herpesvirus which causes African MCF. Sheep have been implicated as carriers of the unidentified virus of North American MCF, but the epidemiologic picture is rather unclear.

Several cases of MCF occurred in captive white-tailed deer when the nearest sheep were 30 m distant with no direct contact (Wobeser et al., 1973, op. cit.). The North American form of MCF is notoriously difficult to transmit, is thought to be cell-associated or quite fragile and has not been isolated and typed. The failures

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to isolate or transmit MCF in this case may suggest North American rather than African MCF etiology.

This deer was a free-ranging animal. No previous history of MCF on this premise or in this county could be found. Inspection of the premises and conversations with the ranchers did not reveal any contact with sheep or exotic ungulates. Until this report, MCF had not been described in free-ranging North American cervids or in black-tailed deer. The existence of endemic North American MCF in black-tailed deer must be considered.

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First Record of Viral Erythrocytic Necrosis and *Ceratomyxa shasta* Noble, 1950 (Myxozoa: Myxosporea) in Feral Pink Salmon (*Oncorhynchus gorbuscha* Walbaum)

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Adult, migrant pink salmon from the lower Fraser River, British Columbia, and five sites upstream were examined for certain disease agents, and for general signs of disease during autumn 1983. *Ceratomyxa shasta* Noble, 1950, was identified in these fish and evidence was obtained for the presumptive diagnosis of viral erythrocytic necrosis (VEN). These findings have not to our knowledge been reported previously in feral pink salmon, although Evelyn and Traxler (1978, J. Fish. Res. Board Can. 35: 903–907) found VEN in artificially reared pink salmon held in sea pens.

Viral erythrocytic necrosis is caused by one or more viruses tentatively assigned to the Iridoviridae and these are known to infect naturally or experimentally a diversity of species of marine and anadromous fish, and possibly certain terrestrial ectothermic vertebrates (Smail, 1982, Proc. R. Soc. Edinb. Sect. B. (Biol.) 81: 169–176). The most obvious gross sign of disease is severe or chronic anemia. The appearance of 0.8–4.0 μm diameter amorphous, pink or magenta colored inclusion bodies (usually one per cell) in the cytoplasm of Giemsa-stained erythrocytes by light microscopy is considered pathognomonic (Evelyn and Traxler, 1978, op. cit.; MacMillan and Mulcahy, 1979, J. Fish. Res. Board Can. 36: 1097–1101). Affected salmon, that may have hematocrits of <5% instead of the usual ca. 40%, can succumb to environmental stressors such as low pO_2, or to bacterial infections (Evelyn and Traxler, 1978, op. cit.; MacMillan et al., 1980, Can. J. Fish. Aquat. Sci. 37: 799–804).

Ceratomyxosis, the disease caused by *Ceratomyxa shasta*, is a severe illness affecting many species of cultured and wild salmonids in certain Pacific watersheds (Johnson et al., 1979, U.S. Dep. Inter. Fish. Wildl. Serv. Fish Dis. Leafl. No. 58, 11

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