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SHORT COMMUNICATIONS

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A Solitary Case of Duck Plague in a Wild Mallard

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ABSTRACT: Duck plague was diagnosed on the basis of pathology and virus isolation in a wild female mallard *Anas platyrhynchos* found dead near Saskatoon, Saskatchewan. Day-old Pekin ducklings and one of two adult mallards died with lesions typical of duck plague following inoculation of tissue from the wild bird. This is believed to be the only reported case of duck plague in a wild bird since a major outbreak occurred in South Dakota in 1973, and the fourth such report in North America.

Key words: Duck plague, mallard, pathology, *Anas platychynchos*, case history.

Much has been learned about the pathogenesis of duck plague (DP) through experimental infections (Proctor and Matthews, 1976; Spieker, 1978; Burgess et al., 1979; Burgess and Yuill, 1983). However, the prevalence and epizootiology of the disease in wild waterfowl remain speculative. Since the first diagnosis in North America in 1967 (Leibovitz and Hwang, 1968), there have been only three published reports of DP in free-flying birds. DP virus was isolated from individuals of several free-living species found dead on Long Island, New York at about the time of the original epizootic in domestic ducks in the area (Leibovitz, 1968) and from black ducks (*Anas rubripes*) in Maryland the following summer (Dardiri and Butterfield, 1969). In 1973 a major outbreak at Lake Andes, South Dakota resulted in the death of about 40,000 mallards (*Anas platyrhynchos*) and 270 Canada geese (*Branta canadensis*) (Friend and Pearson, 1973). Since 1973 there have been outbreaks of DP among captive waterfowl in many areas of North America including California (Snyder et al., 1973), Pennsylvania (Hwang et al., 1975), Quebec (Bernier and Filion, 1975), Alberta (Hanson and Willis, 1976), Washington, D.C. (Montali et al., 1976) and Maryland (Montgomery et al., 1981). Wild waterfowl were suggested as the likely source of infection in several of these instances, but Brand and Docherty (1984) did not find DP virus in any of 4,792 wild waterfowl examined, and we are not aware of any report of DP in free-flying waterfowl since the Lake Andes die-off.

Similarly, in Great Britain there have been several outbreaks among captive birds each year since the disease was first recognized in 1972. However, there has been no evidence of disease in free-flying waterfowl (Gough, 1984).

On 24 May 1984 an adult female mallard found dead on a pond at a zoological park in Saskatoon was submitted to the Western College of Veterinary Medicine for necropsy. The bird was free-flying and had been observed on the pond with a mate for several days prior to her death. It was assumed the bird was a recently arrived migrant and that the pair were establishing a nest in the area. No captive waterfowl were on this pond, but pinioned birds of both native and exotic species of swans, geese and ducks were present on another pond approximately 100 m distant.

When submitted, abundant bloody fluid ran from the bill as the bird was handled.

The bird was in excellent body condition with a large ovary containing many developing ova. The liver was swollen, congested and had tiny white foci, as well as a few ecchymotic hemorrhages. The lymphoid annular bands of the intestine were visible as hemorrhagic areas through the serosa. The esophagus, proventriculus, gizzard and intestine contained thin bloody fluid with focal accumulations of fibrin and debris adherent to the mucosa of the intestine, particularly over the annular bands. The mucosa of the cloaca was covered totally by an adherent necrotic membrane.

Microscopically, there was degeneration and necrosis of hepatocytes with eosinophilic intranuclear inclusion bodies in some cells. There was necrosis of epithelial cells in the intestinal mucosa, particularly over annular bands. Virions, resembling those of an herpesvirus, were present within intranuclear inclusion bodies in hepatocytes examined by transmission electron microscopy.

Ten-day-old Pekin ducklings obtained from a local poultry hatchery were exposed to material from the mallard. Four were given 0.1 ml of a 10% suspension of liver in phosphate buffered saline with penicillin and streptomycin by intramuscular (IM) injection. Four other birds were given 0.1 ml of a similar suspension of splenic tissue by the same route. The other two birds were placed in a common pen with the inoculated birds. All inoculated ducklings became depressed and inactive between 3 and 7 days postinoculation (PI), with swelling of the periorbital tissues, ocular discharge, and pasting of the eyelids. The birds could stand if stimulated, but two had head tilt, one had head tremors and two had mild ataxia. One had sparse red-brown nasal discharge. One of the uninoculated contact birds developed similar clinical signs 6 days after exposure to the inoculated birds. The other contact duckling remained healthy until killed 15 days postexposure.

Ducklings were killed for necropsy when clinical signs were severe. All were dehydrated with severe atrophy of lymphoid organs. In some it was difficult to identify thymic lobules, the spleen was 3–4 mm diameter and very dark, and the bursa of Fabricius was never larger than 8 × 2 mm. The liver was pale and friable with petechial hemorrhages and/or white foci in five birds. The intestines were empty, with hyperemic annular bands in four ducklings. The cloaca was hyperemic in five birds, with adherent necrotic material in one. Microscopically, there was hepatic and intestinal mucosal necrosis with intranuclear inclusion bodies in cells at both sites. The two ducklings killed on days 7 and 8 PI had superimposed *Escherichia coli* septicemia.

Two adult mallards (male and female) reared in captivity from eggs collected in the wild in 1983 each were given 0.5 ml of pooled liver and spleen suspension by IM injection. On day 7 PI the female became weak and unable to stand and was killed when moribund. The only gross lesions seen were petechia on the pericardium and epicardium and adhesion of fecal material to the cloacal mucosa. Histologically there was ballooning degeneration with necrosis of superficial epithelial cells in the lower esophagus, cecum and cloaca, and occasional cells contained intranuclear inclusion bodies. There was focal hepatic necrosis. The male remained normal to day 12 PI, and no gross or histologic lesions were found in this bird.

Portions of liver held frozen at −70 C from the wild mallard, Pekin ducklings and inoculated mallard were sent on dry ice under United States Department of Agriculture permit to the National Wildlife Health Center, Madison, Wisconsin for virus isolation. The livers were diluted 1:10 in Hanks' balanced salt solution, ground in a Waring blender, centrifuged at 200 *g* for 30 min and passed through a 0.22 μm filter. The filtrates were checked for ste-

rility by inoculation of thioglycollate broth. Subsamples of the tissue filtrates were inoculated in 1 ml amounts onto monolayers of muscovy duck (*Cairinia moschata*) embryo fibroblasts prepared by standard methods in 25 cm^2 bottles (Rovozzo and Burke, 1973) and incubated at 40 C. Viral cytopathic effect (CPE) was noted in all cell cultures within 4 days PI. The CPE consisted of rounding of cells followed by degeneration resulting in plaques. Each of the isolates was identified as duck plague virus using a serum neutralization test (Tokumaru, 1969).

The source of infection in this case is unknown. Birds dying in the collection of the park have been submitted for necropsy examination for more than 10 yr with no evidence of DP before or since this case. It seems unlikely that the wild mallard acquired the disease from the captive birds. Alternatively, the infection could have been acquired from other wild birds, or may have been the result of recrudescence of a latent infection.

Mortality among wild waterfowl attracts attention in direct proportion to the number of birds dying. Individual birds found dead, such as in this case, are rarely submitted to a diagnostic laboratory, so that unrecognized low level mortality to DP could occur in nature. The gross lesions in this bird were characteristic of DP, so that experimental infection and virus isolation were done to confirm the diagnosis. However, lesions of DP are probably "maximal" in mallards and Pekin ducks (Leibovitz, 1969) and may be much less prominent (or undescribed) in other species. Thus, individual cases of DP might go unrecognized, even among specimens submitted to a laboratory.

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BOOK REVIEW . . .

George Henry Falkiner Nuttall and the Nuttall Tick Catalogue, James E. Kierans. United States Department of Agriculture, Agriculture Research Service, Miscellaneous Publication No. 1438, Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, USA. 1985. 1785 pp. $61.00 U.S.

This huge volume will not be of great value to many *Journal of Wildlife Diseases* readers, but will be very useful to systematists in acarology. It represents a complete revision and updating of Nuttall's large tick collection housed in the British Museum. Probably the only larger collections are those of Harry Hoogstraal, soon to be housed at the Smithsonian Institute and the Rocky Mountain Laboratories, Hamilton, Montana. Kierans spent a year studying the collection (3,972 entries) and notes of Nuttall, and has produced a useful, easily usable document. Included is a biography of Nuttall, his tick collection in the original and updated form (presented side-by-side for easy comparison), and four appendices listing type specimens ($n = 160$) of tick species, species of ticks, hosts, and geographic locality.

This is a free volume (limited quantities) to those requesting it from USDA, ARS. It can be purchased from the U.S. Government Printing Office, "but the price is subject to change. Call (202) 783-3238 to verify availability and price."

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