



Mallard with duck plague exhibiting prolapsed penis



Duck plague: Intestinal tract with hemorrhagic annular bands



Digestive tract from duck with duck plague: mucosal hemorrhages & necrosis



Esophageal mucosa with viral inclusions (arrows)

EMERGING AND EPIDEMIC DISEASES OF WILD BIRDS

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Slide 1: Title, authors & institutions

Introduction: While most of the American Association of Avian Pathologists study sets are devoted to descriptions of disease conditions in domestic poultry, this study set will describe diseases of wild birds. Diseases of wild birds are important not only in their relation to the health of wild bird populations, but also because they represent important endemic infectious agents which pose threats to both poultry and in some cases human health. While there are many diseases of wild birds, we chose these particular ones because: they have wide geographic distributions; several have the potential to produce high mortality outbreaks; they are important pathogens which could produce disease in either poultry or humans; and several are relatively new emergent diseases in North American wild bird populations. In addition, unless there is something relatively unique about the disease in wild birds or not widely known, we have attempted to avoid duplication with previous AAAP study sets. Therefore, we have included neurotropic Newcastle disease endemic in wild birds, since the previous AAAP study set covered only velogenic viscerotropic Newcastle disease as an exotic disease. We have also chosen to include *Mycoplasma gallisepticum*, which has its own previous study set since this disease has recently emerged and spread across North America in wild birds. Cholera has been previously well described in the AAAP Fowl Cholera study set and is not

included here. Other diseases could have been included, such as salmonellosis, psittacosis, or histomoniasis, but were not due to constraints of the author's familiarity with the diseases, access to suitable images, and simply uncertainty in some cases whether or not these truly represented emerging diseases.

Diseases Included:

Viral:

Duck Plague/Duck Viral Enteritis

West Nile Virus

Newcastle Disease

Bacterial:

Avian Tuberculosis

Finch Conjunctivitis

Undetermined etiology:

Avian Vacuolar Myelinopathy

Slide 2: Outline

Duck Plague/Duck Viral Enteritis

Disease Description: Duck plague, also known as duck viral enteritis (DVE), is an acute, highly contagious disease which causes sporadic outbreaks throughout various regions of North America including the Midwest and eastern United States, California, and Texas. It affects ducks, geese, and swans with high mortality both in free-ranging and captive situations. Initially, DVE was diagnosed in captive Peking ducks and wild waterfowl in adjacent wetlands on Long Island, New York, in 1967. The largest outbreak in wild waterfowl occurred at Lake Andes National Wildlife Refuge, South Dakota, in Jan.-Feb.,

1973. Mortality in the Lake Andes outbreak exceeded 40,000 birds. Since then, multiple outbreaks in free-ranging waterfowl have occurred in scattered sites from coast to coast in the United States and several Canadian provinces, frequently in city park and zoo settings where domestic and feral waterfowl intermingle. Relative susceptibility to infection and mortality rates vary markedly between waterfowl species. Blue-winged teal, wood ducks, and redheads are highly susceptible; mallards, wood ducks and gadwall are moderately susceptible; pintail ducks are least susceptible. The majority of free-ranging bird outbreaks occur during March through June, although mortalities have been reported in every month except August and September. This is an acute hemorrhagic disease with marked pathology in the lymphoid and digestive tissues.

Slide 3: Pile of duck carcasses from die-off due to duck plague at Lake Andes NWR in 1973.

Etiology: The causative agent is a herpesvirus, subfamily *Alphaherpesvirinae*, which is nonhemagglutinating and nonhemadsorbing.

Symptoms/Clinical Signs: Wild waterfowl infected with duck plague generally have a very short clinical disease course. Since disease course is short, generally affected birds are in good body condition. Signs include loss of normal wariness, hypersensitivity to light, thirst, weakness, ruffled feathers, reluctance to fly, watery diarrhea. More specific signs include bloody discharge from the vent or bill, and males may exhibit prolapse of the penis. Other birds may exhibit no clinical signs, and survivors may develop a chronic carrier state. Mortality varies widely from 5-100%.

Slide 4: Bloody discharge draining from the bill of a mallard naturally infected with duck plague.

Slide 5: Prolapsed penis evident on this naturally infected mallard.

Gross Lesions: Some birds infected with DVE will have body cavities and the intestinal lumen filled with large amounts of free blood. The mucosal surfaces of the oral cavity, esophagus, small intestine, ceca and cloaca frequently develop adherent crusted plaques. The spleen is generally small, dark and mottled. The liver may be mottled, and covered with disseminated hemorrhages and pale necrotic foci. The annular bands of lymphoid tissue found in ducks and distributed along the intestine are occasionally hemorrhagic and necrotic. In geese, the intestinal associated lymphoid patches appear as circular or oval areas of hemorrhage and necrosis.

Slide 6: Intestinal tract removed from a naturally infected duck. Note the hemorrhagic annular bands visible along the length of the intestinal tract.

Slide 7: Caudal digestive tract from a naturally infected duck. Note the mucosal hemorrhages and mucosal fibrino-necrotic plaques present in the cloaca, rectum and ceca.

Histopathology: Vessels throughout many tissues exhibit endothelial disruption, with perivascular hemorrhage and edema. Lymphoid tissues, including spleen, thymus, bursa, and intestinal lymphoid patches, have widespread lymphoid depletion and lymphoid necrosis, and may develop intranuclear inclusions within tissue macrophages (reticulum cells). Portions of the gastrointestinal tract, including esophagus and small intestine, have superficial mucosal necrosis with adherent fibrino-necrotic pseudomembranes. Several parenchymatous organs, primarily liver, and also including pancreas and kidney, have widespread hemorrhages and necrotic foci. Within and adjacent to necrotic foci hepatocytes are swollen, with prominent pale intranuclear inclusions which marginate the nucleoli and chromatin material. Similar intranuclear inclusions are commonly found in