

Chapter 16

Duck Plague

Synonyms

Duck virus enteritis, DVE

Cause

Duck plague is caused by a herpesvirus. Infection often results in an acute, contagious, and fatal disease. As with many other herpesviruses, duck plague virus can establish inapparent infections in birds that survive exposure to it, a state referred to as latency. During latency, the virus cannot be detected by standard methods for virus isolation. Studies of domestic species of waterfowl have detected multiple strains of the virus that vary in their ability to cause disease and death. Little is known about the response of wild waterfowl to strain differences.

Duck plague outbreaks are thought to be caused when birds that carry the virus shed it through fecal or oral discharge, thus releasing the virus into food and water with which susceptible birds may have contact. Experimental studies have demonstrated spontaneous virus shedding by duck plague carriers during spring. Changes in the duration of daylight and onset of breeding are thought to be physiological stresses that stimulate virus shedding at this time of year. The carriers are immune to the disease, but the virus shed by them causes infection and disease among susceptible waterfowl. Bird-to-bird contact and contact with virus that has contaminated the environment perpetuate an outbreak. Scavenging and decomposition of carcasses of infected birds also contaminate the environment by releasing viruses from tissues and body fluids. Virus transmission through the egg has been reported, but the role of the egg in the disease cycle remains to be resolved.

Species Affected

Only ducks, geese, and swans are susceptible to duck plague. Other aquatic birds do not become infected, and the absence of mortality of American coot, shorebirds, and other waterbirds that may be present during a waterfowl die-off can be an important indication that duck plague may be involved. Susceptibility varies greatly among waterfowl species (Fig. 16.1). In one study with a highly virulent virus, it took 300,000 times more virus material to infect northern pintail than to infect blue-winged teal.

Distribution

The first reported duck plague outbreak in North America struck the white Pekin duck industry of Long Island, New

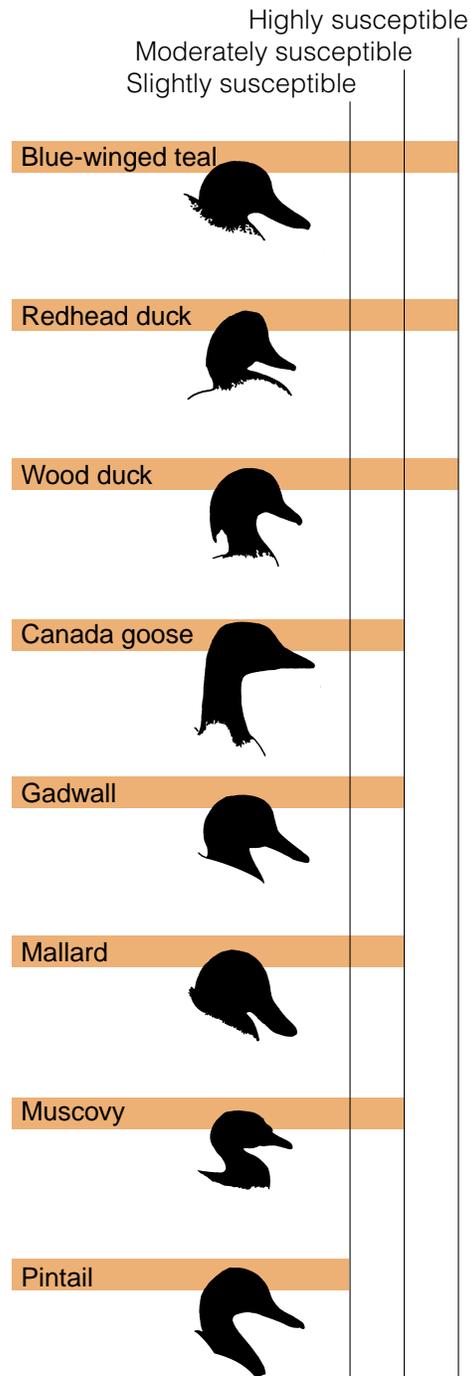


Figure 16.1 Comparative susceptibility of eight waterfowl species to duck plague virus.

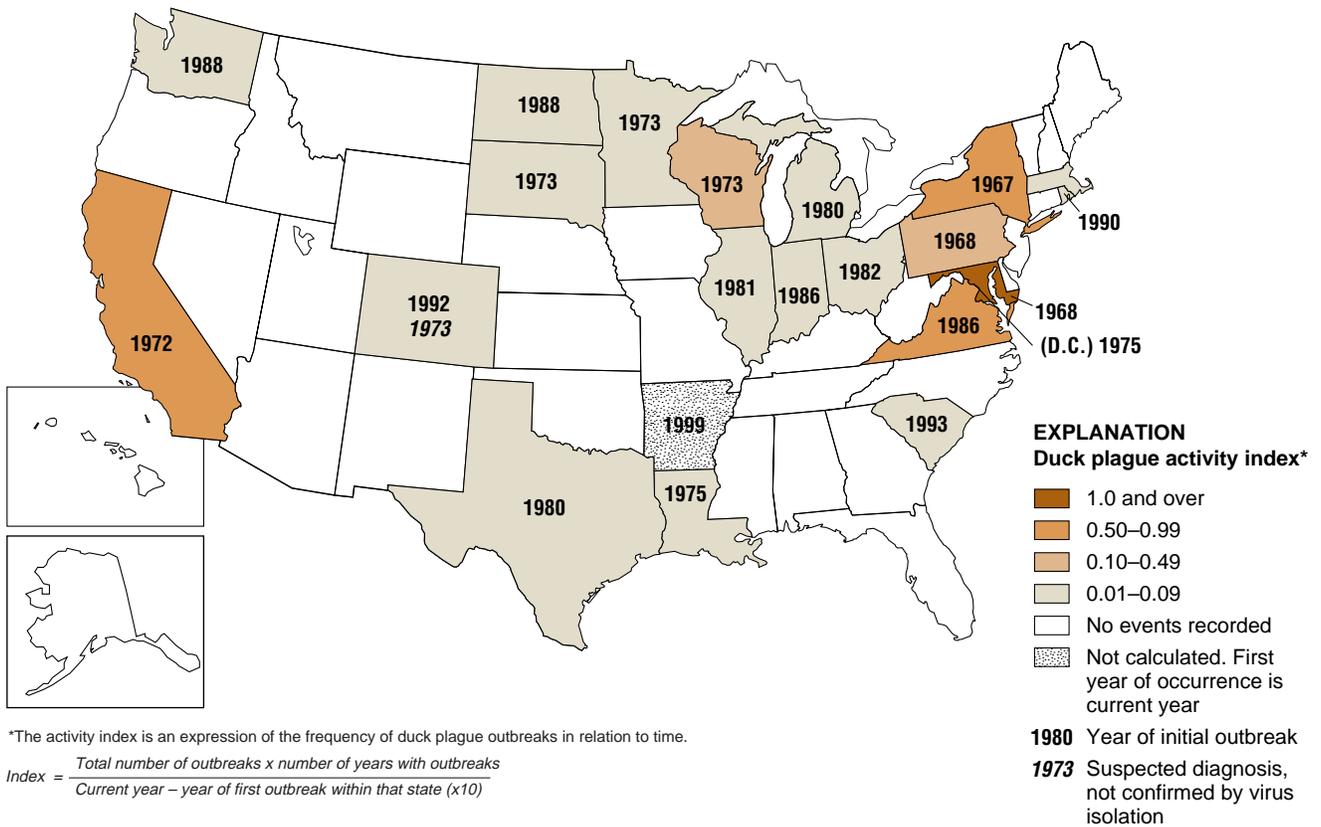


Figure 16.2 Frequency of duck plague since year of first outbreak (1967–1996).

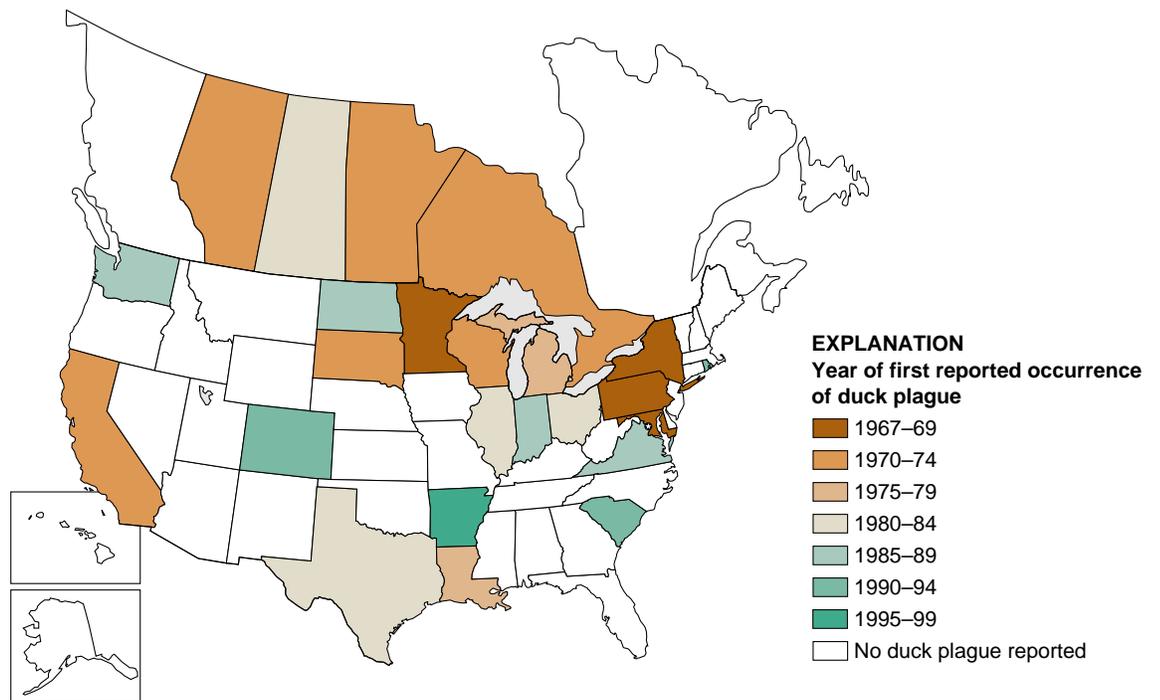


Figure 16.3 Reported North American distribution of duck plague by period of first occurrence.

York in 1967. Since then, duck plague has broken out from coast to coast and from Canada to Texas. The frequency of duck plague outbreaks has varied considerably geographically. The greatest frequency of duck plague activity has been reported in Maryland, followed by California, Virginia, and New York (Fig. 16.2). The disease has also been reported in several Canadian Provinces since it first was observed in the United States (Fig. 16.3). First reported in the Netherlands in 1923, duck plague has also been reported in several other countries in Europe and in Asia since 1958. The frequency of duck plague varies within different types of waterfowl, and failure to respond to these differences complicates disease prevention and control efforts. The different types of waterfowl aggregations involved and the relative frequency of duck plague activity within these different populations are highlighted in Tables 16.1 and 16.2.

Despite the cumulative widespread geographic distribution and frequent occurrence of duck plague in captive and

feral waterfowl in North America, wild waterfowl have been affected only infrequently. The only major outbreaks in migratory waterfowl have happened in South Dakota and New York. In January 1973, more than 40,000 of 100,000 mallards and a smaller number of Canada geese and other species died at Lake Andes National Wildlife Refuge in South Dakota while they were wintering there (Fig. 16.4). The only other duck plague event that caused substantial loss of wild waterfowl occurred during February 1994 in the Finger Lakes region of western New York State. Approximately 1,200 carcasses were recovered, primarily American black duck and mallard, with nearly three times as many black duck as mallard carcasses. The carcasses that were recovered were approximately 24 percent of the black duck and 3 percent of the mallard populations present at the outbreak location. During the initial 1967 outbreak in white Pekin ducks on Long Island, several hundred wild waterfowl carcasses (primarily mallard and American black duck) were recovered

Table 16.1 *Types of waterfowl involved in outbreaks of duck plague in the United States.*

Waterfowl classification	Population composition
Commercial	Birds raised for consumptive markets; for example, white Pekin ducks.
Captive collections	Zoological and other collections of birds for display and research.
Game farm	Birds raised for release for sporting programs; for example, mallard ducks.
Feral	Nonmigratory, nonconfined waterfowl of various species.
Nonmigratory	Resident populations of native wild species; for example, mallard ducks and Canada geese.
Migratory	North American waterfowl that breed in one geographic area and winter in another before returning to their Northern breeding grounds.

Table 16.2 *Relative frequency of duck plague in different types of waterfowl within the United States.*

Waterfowl classification	Occurrence of disease	
	Mortality events	Trends, 1967–1996
Commercial	Rare	Was the primary virus source, but is currently rare
Captive collections	Occasional	None; sporadic outbreaks
Game farm	Occasional	None; sporadic outbreaks
Feral	Common	Increasing outbreaks, and currently prime virus source
Nonmigratory	Occasional	None; sporadic outbreaks
Migratory	Rare	None; rare

from adjacent Flanders Bay, apparently as a result of disease transmission from white Pekin ducks. Those carcasses represented approximately 5 percent of the wild mallard and black duck populations on Flanders Bay during the duck plague outbreak. Mortality in the white Pekin duck flocks was much greater, averaging 45 percent in mature ducks (2-year olds) and 17 percent in immature ducks (younger than 5 months of age). Equally important was the 25–40 percent decrease in egg production by mature breeder ducks that were present during the outbreak. With the exception of the Lake Andes, Finger Lakes, and Flanders Bay outbreaks, duck plague in migratory waterfowl has been limited to a small number of birds. All confirmed outbreaks have also involved commercial, avicultural, captive-raised, or feral waterfowl.

The pattern of duck plague within North America is that of an emerging disease. The number of outbreaks being diagnosed is increasing each decade (Fig. 16.5). The great majority of outbreaks occur within the Atlantic Flyway (Fig. 16.6) and nearly all of those events are within Maryland and Virginia (Fig. 16.7). The factors responsible for the continued emergence and geographic spread of duck plague within North America are unknown, as is the distribution of duck plague among free-living North American waterfowl populations.

Some individuals believe that a large number of surviving wild waterfowl exposed to this disease at Lake Andes became disease carriers, that these disease carriers have perpetuated infections in other wild waterfowl, and that duck plague is now widespread among migratory waterfowl. However, surveys of wild waterfowl conducted by the U.S.

Department of Agriculture in 1967 and by the National Wildlife Health Center (NWHC) from 1978 to 1986 and in 1982–1983 did not detect any evidence of duck plague carriers. In the latter NWHC survey, more than 4,500 waterfowl across the United States were sampled (Fig. 16.8). Sampling sites included major waterfowl concentration areas and areas where duck plague has been a recurrent disease problem in captive and feral waterfowl. Although none of the birds sampled during either NWHC survey were shedding detectable duck plague virus, the previously described problem of inapparent carriers complicates interpretation of these results. New technology that was not yet developed at the time of that survey provides increased ability to detect duck plague carriers and resolve the question of sources for infection.

The absence of duck plague as a cause of mortality in the thousands of wild waterfowl necropsied by the NWHC provides additional evidence that duck plague is not an established disease in wild North American waterfowl. These examinations, performed since 1975, were of waterfowl found dead on National Wildlife Refuges and other major waterfowl concentration areas.

Seasonality

Duck plague outbreaks have been reported during every month except August and September. Approximately 86 percent of these outbreaks occurred from March through June (Fig. 16.9). This pattern of spring outbreaks has also been reported for captive waterfowl collections in England, and it may be associated with the physiological changes referred to above.



Figure 16.4 During the 1973 outbreak of duck plague at Lake Andes National Wildlife Refuge in South Dakota, more than 40,000 mallards died.

Photo by Milton Friend

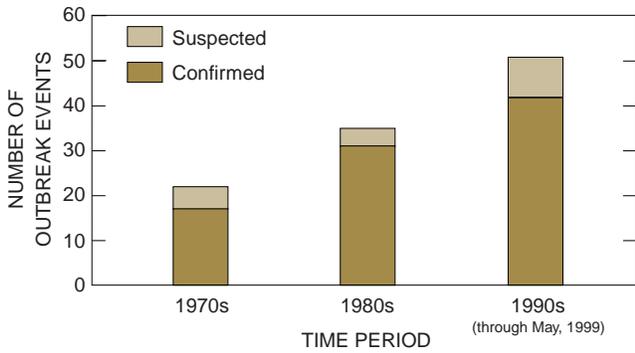


Figure 16.5 Duck plague outbreaks in the United States, 1970s to 1999.

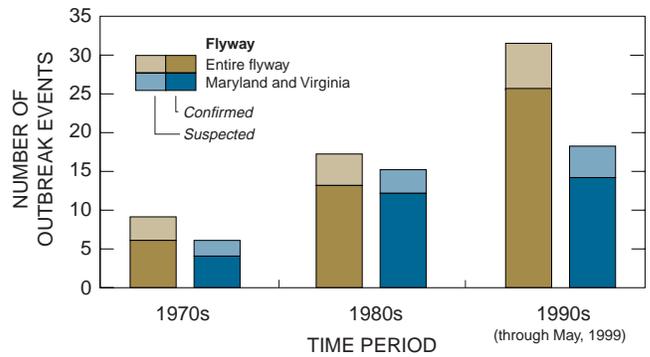


Figure 16.7 Duck plague outbreaks in the Atlantic Flyway, 1970s to 1999.

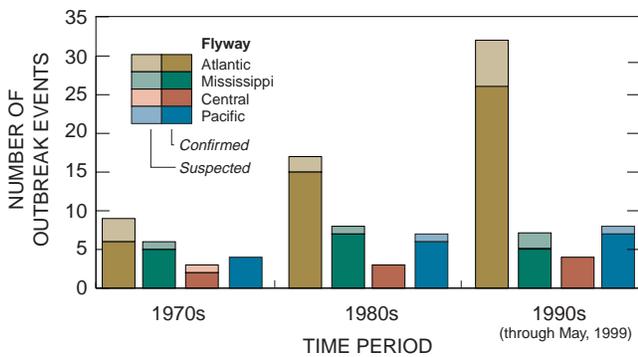


Figure 16.6 Duck plague outbreaks in the United States by flyway, 1970s to 1999.

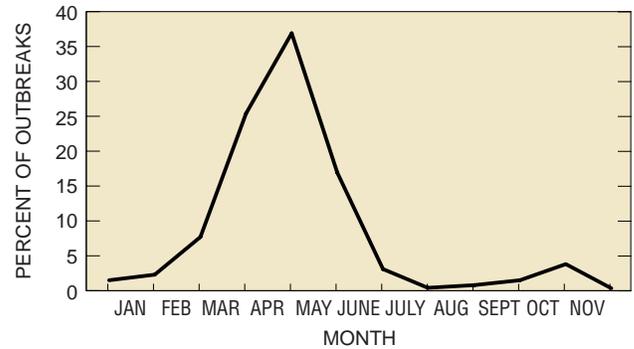


Figure 16.9 Month of onset of duck plague outbreaks, 1967–1996.



Figure 16.8 Sampling locations for 1982–1983 duck plague survey.

Field Signs

There is no prolonged illness associated with duck plague; therefore, sick birds are seldom seen in the field, and birds that are healthy one day may be found dead the next. The incubation period between virus exposure and death is generally 3–7 days in domestic ducks, and experimental studies have found that it is as long as 14 days in wild waterfowl. Wing-clipped mallards released to monitor the Lake Andes duck plague outbreak died 4–11 days after their release.

Sick birds may be hypersensitive to light, causing them to seek dense cover or other darkened areas. They may exhibit extreme thirst, droopiness, and bloody discharge from the vent (Fig. 16.10A) or bill (Fig. 16.10B). The ground may be blood-stained where sick birds have rested (Fig. 16.10C). Therefore, duck plague should be suspected when blood-soiled areas are seen following the flushing of birds, where blood splotches that do not appear to be related to predation or other plausible explanations are seen in the environment, or where bloody discharges are seen where dead birds are lying (Fig. 16.10D). In males, the penis may be prolapsed (Fig. 16.10E).

An ulcerative “cold sore” lesion under the tongue from which virus can be shed has been seen in some infected waterfowl (Fig. 16.11). Routine examination of apparently healthy waterfowl for this lesion during banding operations may be helpful in identifying inapparent carriers. Birds with these lesions should be euthanized (see Chapter 5, Euthanasia) and submitted to a qualified disease diagnostic laboratory for examination.

Death may be preceded by loss of wariness, inability to fly, and finally by a series of convulsions that could be misinterpreted as pesticide poisoning or other diseases such as avian cholera (Fig. 16.12).

Gross Lesions

Duck plague virus attacks the vascular system, and can result in hemorrhaging and free blood throughout the gastrointestinal tract (Fig. 16.13A). At the Lake Andes outbreak, the most prominent lesions were hemorrhagic or necrotic bands circumscribing the intestine in mallards (Figs. 16.13B, C, and D) and disk-shaped ulcers in Canada geese (Figs. 16.13E and F). Sometimes there were “cheesy,” raised plaques along the longitudinal folds of the esophagus and proventriculus (Fig. 16.14A) and on the mucosal surface of the lower intestine (Fig. 16.14B). Areas of tissue death (spots) were also evident in the liver (Fig. 16.14C), as was hemorrhaging on the heart surface of some birds (Fig. 16.14D).

It is important to recognize that the appearance of lesions may differ somewhat from species to species and that not all lesions are present in all birds at all times. Outbreaks of duck plague in captive and nonmigratory waterfowl have often resulted in infected birds with less distinct lesions. Of all the lesions illustrated, those of greatest value in diagnosing duck

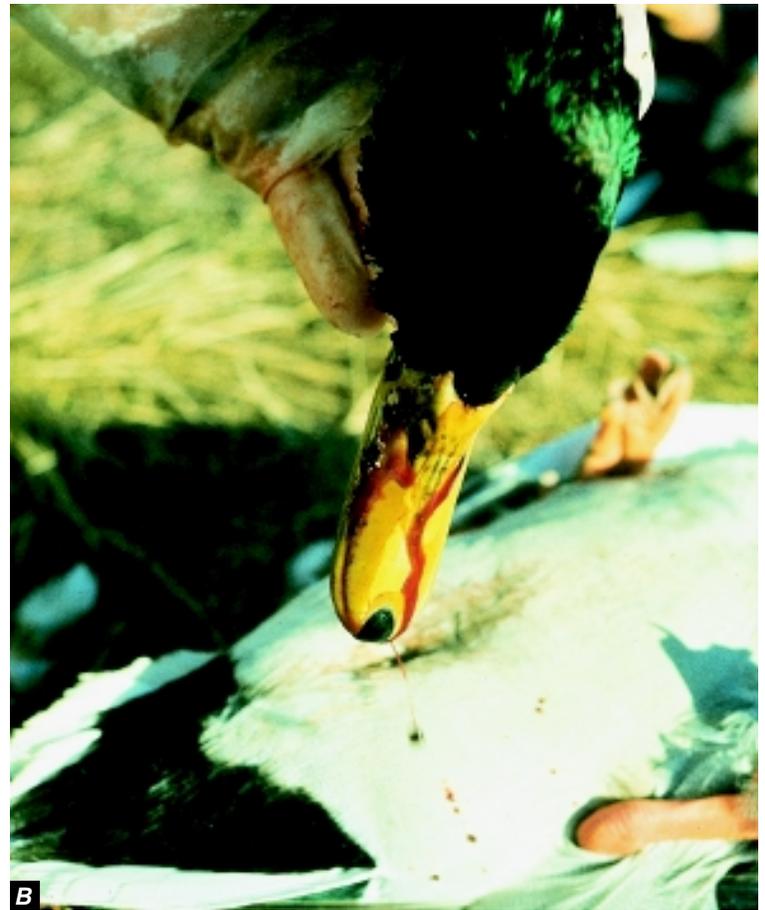


Figure 16.10 Field signs associated with duck plague include: **(A)** blood staining of the vent area; **(B)** blood dripping from the bill or a blood-stained bill; **(C)** blood-stained environment from which a resting mallard has just taken flight; **(D)** blood-stained ice from the nasal discharge of a mallard dying from duck plague; and **(E)** prolapse of the penis.



Photos by Milton Friend



Photo by James Rumminger

Figure 16.11 A “cold sore” under the tongue.



A



B



C



D

Photos by Milton Friend

Figure 16.12 Death sequence observed during terminal stages of duck plague infection at Lake Andes National Wildlife Refuge began with **(A)** the head of the bird dropping forward, wings becoming partially extended from the sides, and tail becoming fanned and rigid. This was followed by **(B)** the bird swimming in a tight circle while rapidly beating the water with its wings and with the head pulled back and twisted to the side. **(C)** At times, birds would fall over on their side, be unable to regain a normal body position, and drown. **(D)** Other birds would simply stop swimming, relax, and quietly die. This entire sequence generally lasted only a few minutes.



Photos by Milton Friend (except photo D)



Photo by James Rummigen



Figure 16.13 Appearance of major lesions of duck plague; (A) hemorrhage and free blood in the lumen of the gastrointestinal tract; (B and C) external appearance of hemorrhagic bands in mallard intestine; and (D) appearance of bands when intestine is opened; (E) external appearance of similar lesions in intestine of a Canada goose; and (F) buttonlike rather than bandlike appearance of lesions when intestine is opened.

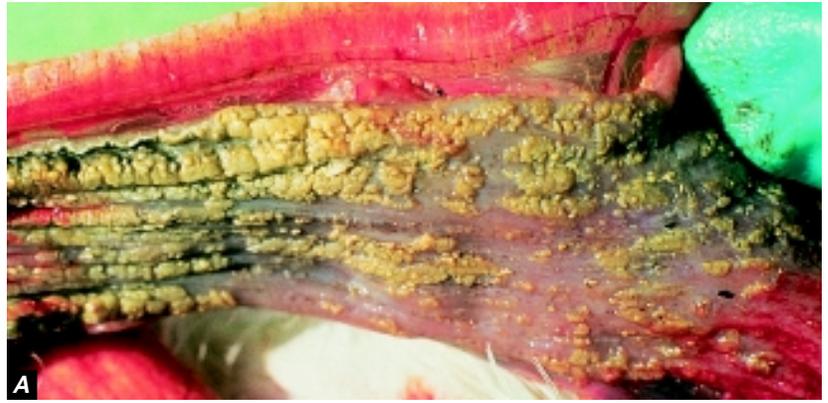


Photo by Steve Schmidt

Figure 16.14 Other internal lesions of duck plague include: **(A)** cheesy, raised plaques along the longitudinal folds of the esophagus, proventriculus, and **(B)** inside (mucosal) surface of the lower intestine. **(C)** Necrotic spots may occur in the liver, and **(D)** varying degrees of hemorrhage on the heart surface.

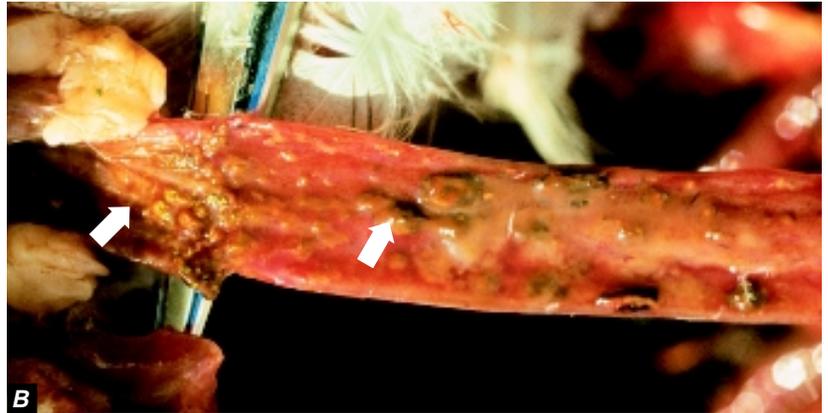


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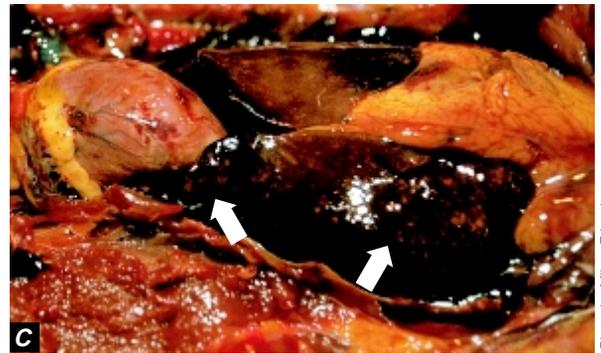


Photo by Milton Friend



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plague are hemorrhagic or necrotic bands or disks within the intestine, large amounts of free blood in the digestive tract, and cheesy plaques in the esophagus and cloaca. Liver and heart lesions of duck plague are grossly similar to those of avian cholera, and they cannot be used to distinguish between these two diseases.

Diagnosis

Although a presumptive diagnosis of duck plague may be made on the basis of characteristic internal lesions, final diagnosis can only be made by virus isolation and identification. Ducks, geese, and swans that have characteristic signs or lesions should be euthanized and shipped to a qualified diagnostic laboratory as quickly as possible. Submit whole birds rather than tissues. When this is not possible, the liver should be removed, wrapped in clean aluminum foil, and then placed in a plastic bag and frozen for shipment. The remainder of the carcass should be incinerated if possible and the area and instruments used to process the carcass disinfected. Take particular care in preserving and packaging specimens to avoid their decomposition during transit and contamination of the shipping containers (see Chapter 2, Specimen Collection and Preservation, and Chapter 3, Specimen Shipment).

Control

The primary objectives for duck plague control activities are to minimize exposure of the population-at-risk at the outbreak site and to minimize the amount of virus present in the environment as a source for potential exposure of waterfowl that may use the site in the near future. Control of duck plague outbreaks requires rapid response and aggressive actions to prevent disease spread and establishment.

Birds with inapparent duck plague infections are probably the major reservoir of this disease and they pose the greatest problem for disease prevention and control. Clinically ill birds actively shed the virus and are recognized as sick birds. However, asymptomatic healthy duck plague carriers can shed the virus periodically, but they are not overtly identifiable. Therefore, destruction of infected flocks, including eggs, is recommended whenever possible because infected birds that survive are likely to become carriers and can initiate subsequent outbreaks. New technology provides promise for determining whether or not there are carriers in a flock. The success of new technology for detecting carriers will allow selective euthanization of those birds and not the remainder of the flock.

Duck plague virus is hardy, and it can remain viable for weeks under certain environmental conditions; for example, the virus could be recovered from Lake Andes water held at 4 °C for 60 days under laboratory conditions. Duck plague virus is instantly inactivated at pH 3 and below and at pH 11 and above. Therefore, rigorous decontamination of infected waters (for example, by chlorination) and grounds (that is,

by raising pH) and burning or decontamination of physical structures, litter, and other materials at outbreak sites should be carried out to the extent practical. Carcass collection should be thorough and incineration used for disposal. Personnel and equipment used at outbreak sites should be decontaminated before leaving the site to prevent mechanical spread of the virus to other waterfowl areas; chlorine bleach and phenol base disinfectants are suitable for this (see Chapter 4, Disease Control Operations).

A low virulence live-virus vaccine has been developed for combating duck plague in the domestic white Pekin, but this vaccine has not been proven entirely reliable in protecting other species of ducks and geese. It should not be considered as a means of controlling or preventing outbreaks in migratory birds.

The close association between duck plague outbreaks and captive waterfowl, especially muscovy and mallard, needs to be considered. Waterfowl release programs should not use birds or eggs from flocks with a history of this disease unless the flock has subsequently been shown by adequate testing and other technical assessments to be free of duck plague. Birds scheduled for release should be confined for at least 2 weeks before release. Birds that die during this period should be submitted to a qualified disease diagnostic laboratory. If duck plague is found to be the cause of death in any of these birds, none of the remaining birds should be released. Also, managers of areas for wild waterfowl should not permit the maintenance of domestic waterfowl, especially muscovy ducks, on the area or waterfowl display flocks that have not been certified free of duck plague.

Human Health Considerations

None.

Milton Friend

(Modified from an earlier chapter by Christopher J. Brand)

Supplementary Reading

- Brand, C.J., and Docherty, D.E., 1984, A survey of North American migratory waterfowl for duck plague (duck virus enteritis) virus: *Journal of Wildlife Disease*, v. 20, p. 261–266.
- Hansen, W.R., Brown, S.E., Nashold, S.W., and Knudson, D.L., 1999, Identification of duck plague virus by polymerase chain reaction: *Avian Diseases* v. 43, p. 106–115.
- Leibovitz, L., 1971, Duck plague, *in* Davis, J. W., and others, eds., *Infectious and parasitic diseases of wild birds*: Ames, Iowa, Iowa State University Press, p. 22–33.
- Wobeser, G.A., 1997, Duck plague, *in* *Diseases of wild waterfowl* (2nd ed): New York, N.Y., Plenum Press, p. 15–27.

