DUCK PLAGUE EPIZOOTICS IN THE UNITED STATES, 1967–1995

Authors: Converse, Kathryn A., and Kidd, Gregory A.

Source: Journal of Wildlife Diseases, 37(2) : 347-357

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-37.2.347

Journal of Wildlife Diseases, 37(2), 2001, pp. 347–357

PERSPECTIVES. . .

DUCK PLAGUE EPIZOOTICS IN THE UNITED STATES, 1967–1995

Kathryn A. Converse[1] **and Gregory A. Kidd**[2]

[1]U.S. Geological Survey, Biological Research Division, National Wildlife Health Center, 6006 Schroeder Road, Madison, Wisconsin, 53711, USA
[2]U.S. Fish and Wildlife Service, 4511 Helgesen Drive, Madison, Wisconsin, 53718, USA
[3]Corresponding author

ABSTRACT: In 1967, the first confirmed diagnosis of duck plague (DP) in the USA was made from pekin ducks (*Anas platyrhynchos domesticus*) on commercial duck farms on Long Island, New York. Within 10 mo, DP was confirmed as the cause of death in migratory waterfowl on a Long Island bay. This paper reviews 120 DP epizootics reported from 1967 to 1995 that involved waterfowl species native to North America or were reported in areas with free-flying waterfowl at risk. Duck plague epizootics occurred in 21 states with the greatest number reported in Maryland (29), New York (18), California (16), and Pennsylvania (13). The greatest frequency of epizootics (86%) was detected during the months of March to June. At least 40 waterfowl species were affected with the highest frequency of epizootics reported in captive or captive-reared ducks including muscovy ducks (*Cairina moschata*) (68%), mallard ducks (*A. platyrhynchos*) (18%) and black ducks (*A. rubripes*) (14%). The greatest number of waterfowl died in three epizootics that involved primarily migratory birds in 1967 and 1994 in New York (USA) and 1973 in South Dakota (USA). The greatest number of DP epizootics reported since 1967 appear to have involved flocks of non-migratory rather than migratory waterfowl; therefore, in our opinion it remains unknown if DP is enzootic in either non-migratory or migratory waterfowl.

Key words: Anseriformes, duck plague, duck virus enteritis, enzootic, epizootic, migratory waterfowl, virus.

INTRODUCTION

Duck plague (DP; also called duck virus enteritis) is a contagious viral disease of Anseriformes that has caused mortality and reduction in egg production in commercial waterfowl (Walker et al., 1969) and variable mortality in wild waterfowl (Walker et al., 1969; Wobeser, 1997). There are a variety of strains of DP virus, some which are more pathogenic than others, and susceptibility to the virus vary by host species (Jansen, 1968; Dardiri and Butterfield, 1969; Spieker, 1996). Waterfowl have survived natural and experimental infections of DP. Pearson and Cassidy (1997) reported DP virus was not isolated from 345 surviving mallard ducks collected during a DP epizootic in South Dakota (USA) but 13% of the ducks were seropositive at dilutions ≥ 1:16. Dardiri and Butterfield (1969) recovered DP virus 17 days after infection from the cloacae of clinically healthy waterfowl. Mallard ducks (*Anas platyrhynchos*) experimentally exposed in a study by Burgess and Yuill (1983) also appeared healthy, shed large amounts of virus, and did not seroconvert. A survey of urban and confined waterfowl surviving nine DP epizootics in six states detected virus in combined oral and cloacal swabs of birds at three sites and DP neutralizing antibody was found in birds from all nine sites (Brand and Docherty, 1988). Exposure of mallard, muscovy (*Cairina moschata*), and pekin (*A. platyrhynchos domesticus*) ducks to DP virus resulted in no hatching of eggs laid by carrier muscovy ducks, significantly reduced hatchability of eggs laid by carrier mallards and no reduction in hatchability of eggs laid by carrier pekin ducks (compared to control pekin ducks which only had 10% hatchability) (Burgess and Yuill, 1981). Burgess and Yuill (1981) also documented vertical transmission to ducklings, that in turn shed small amounts of DP virus. If vertical transmission of DP virus has occurred in resident and migratory waterfowl

populations, the effect of any decrease in egg hatchability and virus shedding by ducklings is unknown.

Duck plague was first diagnosed in the USA in January 1967 in pekin ducks on commercial duck farms on Long Island (New York, USA; Leibovitz and Hwang, 1968). Within 1 mo, DP was confirmed in a free-ranging mute swan (*Cygnus olor*) using a lagoon associated with these farms (Walker et al., 1969). In November 1967, DP was confirmed in migratory waterfowl using nearby Flanders Bay (New York, USA); species included black ducks (*A. rubripes*), mallard ducks, a Canada goose (*Branta canadensis*), and a bufflehead duck (*Bucephala albeola*) (Leibovitz, 1968).

Prior to the first reported epizootic in the USA, DP was reported by Jansen (1968) to occur in Europe and Asia. Jansen (1968) discussed the possibility that DP may have occurred as early as 1923 in the Netherlands and was reported as fowl plague. A contagious viral disease that caused gross lesions similar to DP was diagnosed in muscovy ducks in the Republic of South Africa by Kaschula (1948).

The source for DP introduction in the USA remains unknown. Authors have speculated that this disease was introduced either directly with imported domestic and exotic waterfowl, or indirectly by contact with people or equipment contaminated by the virus through their association with the foreign duck industry, or even by wild Anseriformes (Leibovitz and Hwang, 1968; Walker et al., 1969). Newcomb (1968) considered introduction of DP into the USA by migratory waterfowl improbable and transmission on fomites unlikely, and supported the theory of importation via exotic ducks and geese. Following the 1967 outbreak of DP in New York, the virus was thought to have been transmitted among farms by introduction of infected ornamental and domestic waterfowl, exposure to wild waterfowl, contact with common sources of contaminated water on farms, or traffic in infected material associated with tourists, equipment, or supplies (Leibovitz and Hwang, 1968).

The possibility that a disease similar to DP occurred in the northeastern USA prior to 1967 was discussed by Leibovitz and Hwang (1968). There was serological evidence of exposure to DP virus at three other commercial duck farms on Long Island prior to the 1967 epizootic (Newcomb, 1968), but DP virus was not isolated. Furthermore, a 1969 letter from the Pennsylvania Department of Agriculture (Harrisburg, Pennsylvania, USA), discussed a "muscovy disease" periodically reported by duck fanciers that characteristically killed all their muscovy ducks and (*Alopochen aegyptiacus*) geese.

Between 1967 and 1973, DP was considered by the USA Department of Agriculture (USDA, Washington, D.C.) as a reportable disease in the USA, and disease control efforts during DP epizootics were conducted by the Animal Health Division, (Animal and Plant Health Inspection Service, USDA, Washington, D.C.; Walker et al., 1969). Because convalescent ducks were known to be silent carriers of this herpesvirus, control methods for DP included quarantine, eradication of affected flocks, and decontamination of the environment. Vaccination against duck plague was recommended for use "only in flocks not known to have been affected with the disease" (Walker et al., 1969). In 1973, the largest reported epizootic of DP killed an estimated 43,000 birds, primarily mallard ducks, from an estimated population of 163,500 migratory waterfowl at the Lake Andes National Wildlife Refuge (South Dakota, USA; Pearson and Cassidy, 1997). Discussions between the U.S. Fish and Wildlife Service (USFWS; Washington, D.C.) and USDA during this epizootic led the USDA to decide later in 1973 to revoke the classification of DP as an exotic disease for the commercial duck industry (L. N. Locke, pers. comm.). Although the USDA remained informed about duck plague epizootics and often assisted with disease control efforts, primary documen-

tation and management of DP in free-flying and migratory birds was transferred to the USFWS and state conservation agencies. The U.S. Geological Survey National Wildlife Health Center (NWHC; Madison, Wisconsin, USA) with the cooperation of many federal, state, and private agencies, has maintained a national database of wildlife epizootics that includes records of DP epizootics in which migratory waterfowl were either involved or considered at risk because free-flying waterfowl had access to the mortality site.

The potential for DP to cause both mortality and a carrier state in resident and migratory waterfowl has fueled a continuous debate regarding the sources of the virus and approaches for management of this disease. Much of this controversy is caused by a lack of information about DP and uncertainty about responsibilities of private individuals and various agencies for specific waterfowl populations. We summarize the reported occurrences of DP in the USA to define the seasonal, annual, and geographic distribution of DP epizootics and the prevalence of this disease by species, to aid in development of research programs and disease management decisions.

MATERIALS AND METHODS

Data were collected through review of published literature, annual reports of the United States Livestock Sanitary Association (Washington, D.C.), and the Animal Health Association (USAHA; formerly the United States Livestock Sanitary Association), and unpublished reports prepared by various governmental and nongovernmental agencies and diagnostic facilities. Data after 1973, following the shift in reporting from USDA to USFWS and use of vaccines in duck production facilities, primarily represent epizootics where free-flying or migratory waterfowl died or were reported at risk from contact with sick waterfowl or a contaminated environment. Epizootics that occurred within commercial or private waterfowl populations were seldom reported unless free-flying waterfowl had access to the site and few are included in this review. Locations of DP epizootics were identified and mapped by states within decades. We summarized the reported techniques used for disease control in epizootics.

In this paper, we categorized waterfowl as migratory, resident, free-flying, or exotic. North American waterfowl that traveled long distances annually to satisfy needs for food and reproduction were considered migratory while waterfowl that remained in the same geographic area throughout the year were considered residents. Waterfowl that did not follow the known seasonal migratory patterns or captive-reared waterfowl that were not confined or pinioned were considered free-flying. Exotic waterfowl were species that are not native to the USA.

Since 1967, diagnosticians have confirmed DP by many factors or combinations of factors including the case history, presence of gross and microscopic lesions consistent with DP, mortality following animal subinoculation, and isolation and identification of the virus. Prior to the late 1970's, presence of DP virus was usually confirmed by: inoculation of suspected tissue into ducklings that subsequently died with characteristic gross and microscopic lesions, serum neutralization tests, plaque assay (Dardiri and Hess, 1967), or fluorescent antibody cell culture (Erickson et al., 1974). Since the late 1970's, isolation of virus in primary duck embryo fibroblasts and serum neutralization have been the more commonly selected methods of culture and identification (Docherty and Slota, 1988).

We developed nine general classifications to describe sites where DP was reported including: (1) private and commercial collections - areas with waterfowl housed and cared for by private individuals; (2) farms - waterfowl present with domestic animals in an agricultural setting; (3) municipal - urban parks and recreation areas, golf courses, and other more open areas in an urban setting; (4) residential - urban or suburban creeks, ponds or small lakes surrounded by residences; (5) zoos - public facilities with lifetime captive birds and open waterfowl displays; (6) research - research facilities with captive waterfowl; (7) lake/reservoir - larger bodies of water usually with minimal development of shorelines; (8) bay/shore/creek - shorelines of fresh and tidal moving waters; and (9) unknown.

RESULTS

Duck plague was diagnosed as the cause of waterfowl mortality in 120 epizootics reported between 1967 and 1995. DP was reported every year except in 1974. Total annual mortality was <350 waterfowl in 26 of the 29 yr, despite eight or more epizo-

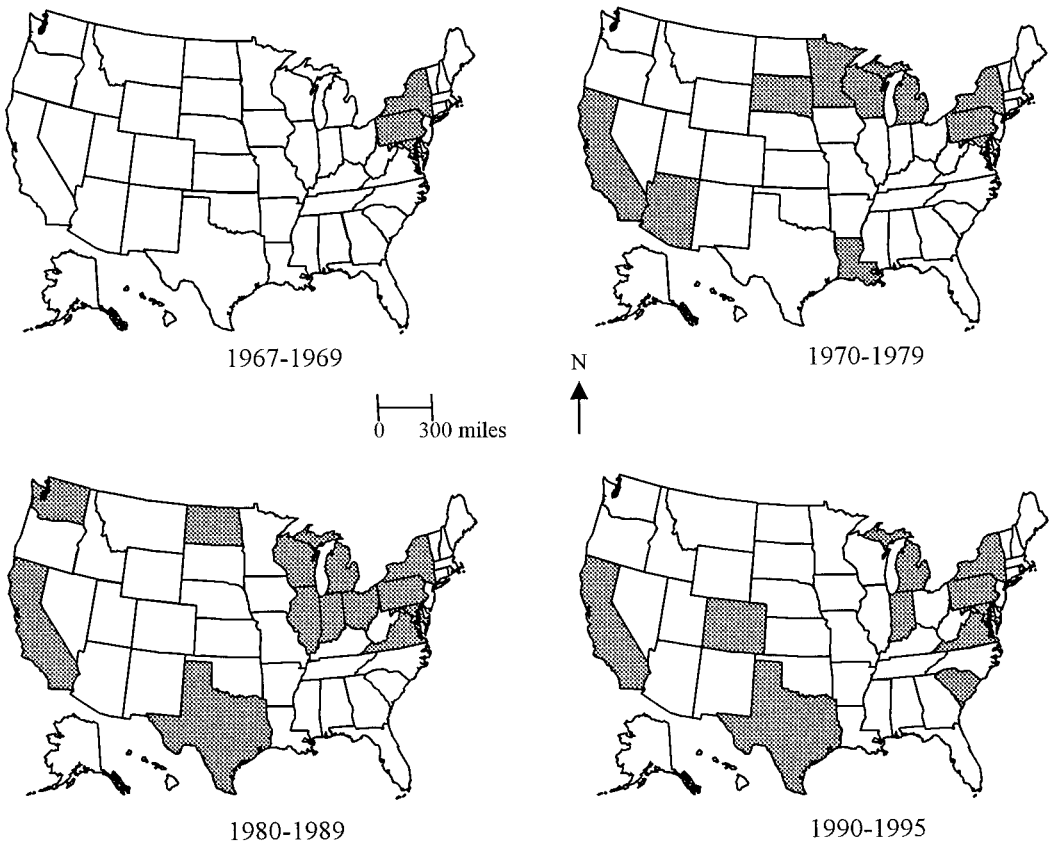

FIGURE 1. Annual duck plague epizootics and total mortality reported in the USA, 1967–1995.

otics in some years (Fig. 1). Annual DP mortality exceeded 1,200 in 1967, 1973, and 1994, due to occurrence of major epizootics involving migratory waterfowl.

Duck plague was confirmed in 98 (82%) of the 120 epizootics by virus isolation, mortality following animal sub-inoculation, serum neutralization, microscopic confirmation of viral intranuclear inclusion bodies in tissue cells, or identification of a herpesvirus using electron microscopy. In the remaining 22 (18%) epizootics, the diagnosis of DP was based on history and gross lesions in 21 epizootics and one diagnosis was based solely on the history. In many epizootics, virus isolation was either not attempted or it was unsuccessful due to tissue autolysis.

Caseous plaques along the longitudinal folds of the esophagus, proventriculus, and mucosal surface of the lower intestine as described by Leibovitz (1971) and Synder et al. (1973) were the gross lesions most often described in epizootics. Waterfowl examined during some epizootics had areas of hemorrhage and free blood throughout the intestinal tract, hemorrhagic or necrotic bands circumscribing the intestine, disk-shaped ulcers in the intestines, necrotic foci in the liver, or hemorrhage on the heart surface. Microscopic lesions included intranuclear and intracytoplasmic inclusions, focal liver necrosis, necrosis of esophageal and intestinal mucosa, and capillary hemorrhage (Montgomery et al., 1981; Barr et al., 1992; Davidson et al., 1993). Leibovitz (1969) found that characteristic lesions of DP in domestic waterfowl were absent in some migratory Anseriformes. Our review of diagnostic descriptions of duck plague lesions showed variation in the presence or sever-

TABLE 1. Native North American waterfowl species reported dead in duck plague epizootics in the USA, 1967–1995, listed in descending order by the number of epizootics.

Species	Number of epizootics	Estimated mortality
Muscovy Duck (*Cairina moschata*)[a]	82	1,568[c]
Mallard (*Anas platyrhynchos*)[b]	21	29,200[c]
Black Duck (*Anas rubripes*)[b]	17	1,522
Canada Goose (*Branta canadensis*)[b]	10	658
Mute Swan (*Cygnus olor*)[a]	6	17
Wood Duck (*Aix sponsa*)[b]	4	7[c]
American Wigeon (*Anas American*)	3	3[3]
Common Merganser (*Mergus merganser*)[b]	3	4[c]
Gadwall (*Anas strepera*)	2	2
Bufflehead (*Bucephala albeola*)[b]	2	3[c]
Redhead Duck (*Aythya Americana*)[b]	2	2[c]
Goldeneye (*Bucephala* sp.)[b]	2	2[c]
Canvasback Duck (*Aythya valisineria*)[b]	2	1[c]
King Eider (*Somateria spectabilis*)	1	4
Ruddy Duck (*Oxyura jamaicensis*)	1	1
Cinnamon Teal (*Anas cyanoptera*)	1	1
Green-winged Teal (*Anas crecca*)	1	1
Blue-winged Teal (*Anas discors*)	1	1
Hooded Merganser (*Lophodytes cucullat*)	1	1
Greater Scaup (*Aythya marila*)[b]	1	1[c]
Lesser Scaup (*Aythya affinis*)	1	1
Mottled Duck (*Anas fulvigula*)	1	1
Black-bellied Whistling Duck (*Dendrocygna autumnalis*)	1	1

[a] Reported as migratory species in epizootics, status as a North American species is in question.
[b] Includes migratory waterfowl present in five epizootics.
[c] Number dead not stated in at least one epizootic.

ity of reported lesions by species and epizootics.

There were 40 individual species reported dead during DP epizootics, 23 were native North American species (Table 1), while 17 were exotic species. The remaining waterfowl were either hybrids or unidentified species. Only four of the 40 species were found dead in 10 or more epizootics; muscovy ducks died in 82 epizootics, mallard ducks in 21, black ducks in 17, and Canada geese in 10. Wood ducks (*Aix sponsa*), mute swans and five hybrid groups were collected in four to six epizootics, and 34 species and 10 unidentified species were collected in three or fewer epizootics. Species recorded as migratory waterfowl in DP epizootics included Canada geese, and mallard, black, bufflehead, American wigeon (*A. americana*), redhead (*Aythya americana*), canvasback (*A. valisineria*), greater scaup (*A. marila*), common merganser (*Mergus merganser*), goldeneye (*Bucephala* sp.), and wood ducks. Mute swans were listed as migratory birds in the 1967 epizootic.

Free-flying waterfowl were reported to be at risk in 91 (76%) of the epizootics. Despite the potential for exposure between free-flying waterfowl and sick birds in 23 (19%) epizootics, confirmation of this exposure was not recorded or unknown by the person reporting the epizootics. In six epizootics (5%), written accounts specifically stated that "wild" waterfowl were not in contact with the affected waterfowl; two were reported on farms, two in captive birds in research facilities, and two in penned birds in private collections.

During the past 29 yr, DP was reported in 21 states with great variability in annual incidence and location (Fig. 2). From 1967 through 1969, 10 DP epizootics were reported in three northeastern states includ-

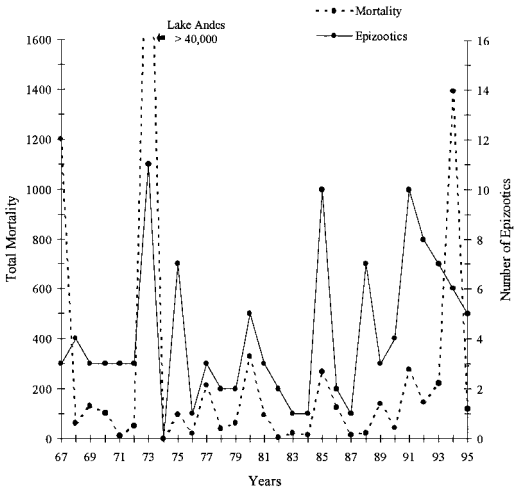

FIGURE 2. Reported Duck plague epizootics in the USA, 1967 to 1995.

ing New York (6), Pennsylvania (3), and Maryland (1) (Fig. 2). During the 1970's, there were 35 epizootics in 11 states with recurrence in the same three northeastern states of New York (10), Pennsylvania (4), Maryland (5) and first time reports in eight states scattered throughout the USA including California (4), Wisconsin (3), South Dakota (2), Minnesota (2), Louisiana (2), Arizona (1), District of Columbia (1), and Michigan (1) (Fig. 2). During the 1980's, 35 epizootics were reported in 13 states with recurrence in the six states of Maryland (13), California (5), Wisconsin (3), Pennsylvania (1), New York (1), and Michigan (1); and first time reports in Texas (2), Ohio (2), Virginia (2), Washington (2), North Dakota (1), Illinois (1), and Indiana (1) (Fig. 2). From 1990–1995, 40 epizootics were reported in 11 states with recurrence in the eight states of Maryland (10), Virginia (7), California (7), Pennsylvania (5), Texas (3), Michigan (2), Indiana (2), and New York (1); and first time reports in Rhode Island (1), Colorado (1), and South Carolina (1) (Fig. 2). Overall, the greatest number of epizootics was reported in Maryland (29), New York (18), California (16), Pennsylvania (13), Virginia (9), and Wisconsin (6). The remaining 15 states reporting DP had five or fewer epizootics. Throughout the four decades, DP may have occurred more than once at 10 of the 120 unique locations although exact descriptions of some locations within townships was variable.

Several states had long intervals between reported DP epizootics. For example, Pennsylvania reported DP in 1968–1972, 1980, and 1991–1994. Texas had two epizootics in 1980 with a 10 yr interval until the next epizootic in 1990. New York had annual epizootics from 1967 to 1973; DP was reported again in 1975, 1989, and 1994. The first reported DP epizootics in Virginia were in 1986 and 1988 along the Maryland border. Since 1991, DP has been reported annually in Virginia.

Since 1967, the incidence of reported DP epizootics has varied annually from a high of 11 in 1973 to none in 1974, with an average of four epizootics per year (Fig. 1). The cumulative reported DP mortality for the 29 yr was <50,000 waterfowl and with the exception of two events, represented epizootics involving small numbers of waterfowl. There was an average annual mortality of 39 (1–277) birds during the 115 DP epizootics without reported mortality in migratory waterfowl (Fig. 1). Migratory waterfowl were involved in the three DP epizootics with the greatest mortality. In November 1967, an estimated 100 waterfowl (primarily black and mallard ducks) died at Flanders Bay (Long Island, New York) in association with losses of approximately 1,100 commercial ducks; in January 1973, 43,000 waterfowl (primarily mallard ducks) died at Lake Andes National Wildlife Refuge (South Dakota) and in February 1994, 1,150 ducks (primarily black ducks) died at Keuka Lake (New York). The remaining two epizootics in migrating waterfowl reported in this paper, are two events with isolation of DP in a solitary adult waterfowl that was found dead during the nesting season in the USA; a black duck collected in 1985 in Maryland, and a mallard duck collected in 1988 during an intensive study of nesting

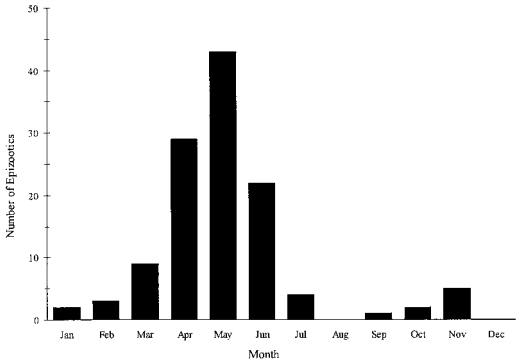

FIGURE 3. Month of onset of 120 duck plague epizootics in the USA, 1967–1995.

TABLE 2. Frequency of reported duck plague epizootics in the USA, 1967–1995, by site classification.

Site classification	Number of epizootics	Frequency of epizootics %
Private collection	34	28
Farm	28	23
Municipal	15	13
Residential	13	11
Zoo	9	8
Research	6	5
Shore/creek	6	5
Lake/reservoir	5	4
Unknown	4	3
Total	120	100.0

waterfowl in North Dakota (C. J. Brand, pers. comm.).

The greatest frequency of duck plague epizootics (86%) were reported from March through June (Fig. 3). Burgess and Yuill (1983) found that cloacal shedding of DP virus by experimental mallards peaked during May and oral shedding of virus peaked in June. In the 1967 epizootic in commercial waterfowl, mortality was higher in flocks in egg production than in immature breeders (Walker et al., 1969). In contrast, three epizootics involving migratory waterfowl were reported during January and February; two of the events, Lake Andes and New York Finger lakes, occurred when flocks were concentrated during severe weather conditions. The seasonal distributions of the remaining eight epizootics were: one in February in captive waterfowl in a zoo, two in October and one in November in free-flying mute swans in the New York's Finger Lakes region, and four epizootics were reported in November 1980 in private collections in Texas, Pennsylvania, and Maryland that received ducks from a single commercial producer. No epizootics were reported to start in August or December.

DP was reported with the greatest frequency (75%) in free-flying, resident or exotic waterfowl associated with site classifications of private collections, farms, and municipal and residential areas (Table 2). The five DP epizootics involving migratory waterfowl occurred at sites within the classifications of lake/reservoir and bay/shore/creek.

Historically, disease control efforts following confirmation of DP have varied and include (1) no intervention with or without presence of surviving waterfowl assumed to be exposed; (2) decontamination of areas after all the waterfowl present had died; (3) placement of a lifetime quarantine on waterfowl remaining after an epizootic; and (4) quarantine, eradication of remaining resident and free-flying waterfowl and decontamination of the affected area. During epizootics, it was often reported as impossible to identify or locate free-flying waterfowl that may have been exposed to the virus. Because of the low number of DP epizootics in migratory waterfowl, many wildlife managers considered DP to be an exotic disease of wild waterfowl and, when possible, followed the conservative recommendations of USDA (quarantine, eradication of exposed waterfowl, and site decontamination) (Walker et al., 1969), and guidelines for management of DP epizootics as presented by Nettles and Thorne (1988).

DISCUSSION

We assume that DP was introduced into the USA in 1967 but there is evidence that DP could have been present in waterfowl in northeastern USA prior to that time. Despite the fact that DP was reported in

28 of 29 yr after the first epizootic in New York, it was only reported in 21 states. The greatest number of epizootics has occurred only intermittently in the original three states reporting DP. Duck plague has been reported in other states with a more unpredictable pattern of distribution. In the first progress report on DP surveillance in American waterfowl, Leibovitz (1968) stated, "if this disease is an emerging exotic infection in wild Anseriformes, then it is probable that there will be a future geographical extension from the known loci of infection. This extension would then conform to the migratory patterns of susceptible Anseriformes." In the Atlantic Migratory Bird Flyway, DP continued to occur in the three states reporting DP in the 1960's (New York, Maryland, and Pennsylvania) but each state had intervals of 3 to 14 yr with no epizootics. There could have been an extension of DP from Maryland to waterfowl species in neighboring Virginia, but DP epizootics were not reported to occur in other states throughout the Atlantic Flyway. If the 29 occurrences of DP in mallard ducks and black ducks in the greater Chesapeake Bay shorelines of Maryland and Virginia had included migratory waterfowl, we expect that DP would occur throughout their breeding grounds in Manitoba and Saskatchewan. There is only one report of a mallard duck dying of DP in Saskatchewan in 1984 (Wobeser and Docherty, 1987). Similarly, in the Mississippi, Central, and Pacific Migratory Bird Flyways, DP was reported in <50% of the states and mortality involving only one migratory mallard was reported on the breeding grounds in North Dakota (C. J. Brand, pers. comm.). In Canada, as in the USA, DP has been reported in resident and captive waterfowl but there have been no epizootics reported in migratory waterfowl (Wobeser, 1997). In Canada, as in the USA, lack of surveillance for DP, particularly on waterfowl nesting areas, may account for fewer reports of DP mortality.

By comparison, avian cholera, a contagious bacterial disease of migratory birds that also can also produce a carrier state (Samuel, et al., 1997), has been reported in 51 waterfowl species (NWHC, unpubl. data) along migratory pathways in the USA and Canada since the first reported epizootics in waterfowl in 1944 in California and Texas (Brand, 1984; Botzler, 1991). Avian cholera has been reported in snow geese on northern breeding grounds and geese with serum antibody levels indicative of exposure to *Pasteurella multocida* were found at Wrangel Island (Russia) and Banks Island (Northwest Territories, Canada) (Samuel et al., 1999).

We conclude that it remains unknown whether or not DP is enzootic in either resident or migratory waterfowl. In this review we document DP mortality in 23 species native to North America. Although most of the birds of these species were captive reared, their death supports the susceptibility of many migratory waterfowl species as discussed by Spieker (1996) and Wobeser (1997). Reports of only three epizootics in migratory waterfowl, involving more than one bird, and occurrence during winter suggest carrier birds are either not present in migratory populations, there are differences in seasonal shedding of DP virus by carrier birds, or the existence of other factors are responsible for initiating an epizootic in these populations. Pearson and Cassidy (1997) state that the absence of DP epizootics in migratory waterfowl for 20 yr after the Lake Andes epizootic is evidence that DP carriers can exist in a population and not shed virus to initiate major epizootics or that a DP carrier state does not develop or persist in "free-flying wild waterfowl." If the later is true, then migratory waterfowl may not be the DP carriers causing epizootics in nonmigratory waterfowl. Epizootics involving small numbers of migratory birds may have gone undetected, as demonstrated by confirmation of DP in three solitary waterfowl, assumed to be migratory, that were collected during their breeding season in the USA and Canada. During the

29 yr covered by this review, the ability to detect small mortality events throughout the USA was evidenced by NWHC records of 1,111 epizootics involving ≤100 waterfowl; more than 940 of those epizootics involved ≤50 waterfowl and 616 involved ≤25 waterfowl (NWHC, unpubl. data).

Several waterfowl surveys were conducted to determine either the presence of DP virus or serum antibodies to DP in resident and migratory waterfowl populations. The first DP survey, conducted in 1968, determined DP antibodies were not present in more than 3,000 migratory and commercial waterfowl collected at 14 sites in 12 states (Walker et al., 1969). During 1968, the USDA investigated all reported epizootics in migratory waterfowl in Delaware, Florida, Maryland, Massachusetts, Pennsylvania, and Virginia and concluded DP was not present (Walker et al., 1969). A second survey of more than 4,700 migratory mallard, pintail and black ducks throughout the USA, conducted in 1982 and 1983, yielded no DP virus (Brand and Docherty, 1984). Lin et al. (1984) found antibodies in 17% of 421 "non-domestic" waterfowl collected in California during a survey presumed to occur after an April epizootic. In a series of surveys conducted by Brand and Docherty (1988), from 1978 through 1986, cloacal and oral pharyngeal swabs and blood samples were taken from waterfowl surviving eight DP epizootics. Samples from three of eight sites were positive for DP virus including one wild green-winged teal (*A. crecca*), but neutralizing antibody to DP virus was found in waterfowl at seven of eight sites. Brand and Docherty (1988) also sampled free-flying waterfowl within 8–52 km of four DP epizootics. Duck plague virus was not detected in the cloacal and tracheal swabs at four sites but neutralizing antibody to DP was detected in five urban mallards, one pekin duck and a wild blue-winged teal (*A. discors*) in the vicinity of one site. Similarly in Britain, Asplin (1970) conducted a survey of "wildfowl" collected during a winter banding effort and found 3/510 waterfowl had DP antibodies; the site was 80 miles from a Dutch DP site. Wobeser (1997) and Burgess and Yuill (1983) address the possibility that virus was present but not detected in surveys or research studies. If migratory waterfowl are shedding virus at a different time as Pearson and Cassidy (1997) suggest, then carriers may be detected less frequently during a particular window of sampling. Field studies have been completed in the USA and Britain to test a recently developed polymerase chain reaction (PCR) assay for detecting waterfowl that are shedding duck plague virus (Hansen et al., 1999). This new tool will be used to continue evaluation of enzootic DP infections in migrating and non-migrating waterfowl populations.

The high frequency of reported epizootics involving muscovy ducks and feral or captive-reared mallard ducks and black ducks suggest to us either greater susceptibility of these hosts to virus transmitted by migratory waterfowl or presence of a reservoir for maintenance and transmission of this virus within these domestic and resident populations. We feel that the higher frequency of DP epizootics in private collections, farms, and residential and municipal flocks during late spring when migratory flocks are not present supports the presence of a reservoir of DP virus in these populations or possibly greater transmission of the virus in crowded conditions. In addition, the ability to detect mortality in populations using these sites is assumed to be higher than detection in migratory populations.

Although mortality was low (1 to 277) in DP epizootics in non-migratory waterfowl, losses of 100 to 43,000 in three epizootics involving migratory waterfowl are evidence of the potential of this disease to kill large numbers of susceptible migratory waterfowl. We believe that efforts to manage DP may help reduce the potential for development of DP carriers and subsequently a reservoir for DP virus in resident and migratory waterfowl populations. We

urge all states, even those that have never reported DP epizootics, to make an effort to initiate or maintain surveillance of waterfowl flocks to detect early signs of disease.

ACKNOWLEDGMENTS

We gratefully acknowledge the efforts of field personnel who collected sick waterfowl and the diagnostic facilities whose staff examined birds and diagnosed DP over the 29 years of this review. We also thank NWHC personnel who updated and maintained files of these events. Particular thanks are due to C. J. Brand, M. Friend, L. N. Locke, V. Nettles, and G. Stein who reviewed earlier drafts of this manuscript and provided insights into the history of this disease.

LITERATURE CITED

ASPLIN, F. D. 1970. Examination of sera from wildfowl for antibodies against the viruses of duck plague, duck hepatitis and duck influenza. Veterinary Record 87: 182–183.

BARR, B. C., D. A. JESSUP, D. E. DOCHERTY, AND L. J. LOWENSTINE. 1992. Epithelial intracytoplasmic herpes viral inclusions associated with an outbreak of duck viral enteritis. Avian Diseases 36: 164–168.

BOTZLER, R. G. 1991. Epizootiology of avian cholera in wildfowl. Journal of Wildlife Diseases 27: 367–395.

BRAND, C. J. 1984. Avian cholera in the Central and Mississippi Flyways during 1979–80. The Journal of Wildlife Management 48: 399–406.

———, AND D. E. DOCHERTY. 1984. A survey of North American migratory waterfowl for duck plague (duck virus enteritis) virus. Journal of Wildlife Diseases 20: 261–266.

———, AND ———. 1988. Post-epizootic surveys of waterfowl for duck plague (duck virus enteritis). Avian Diseases 32: 722–730.

BURGESS, E. C., AND T. M. YUILL. 1981. Vertical transmission of duck plague virus (DPV) by apparently healthy DPV carrier waterfowl. Avian Diseases 25: 795–800.

———, AND ———. 1983. The influence of seven environmental and physiological factors on duck plague virus shedding by carrier mallards. Journal of Wildlife Diseases 19: 77–81.

DARDIRI, A. H., AND W. R. HESS. 1967. The incidence of neutralizing antibodies to duck plague virus in serums from domestic ducks and wild waterfowl in the United States of America. Proceedings of the United States Livestock Sanitary Association 1967: 225–237.

———, AND W. K. BUTTERFIELD. 1969. The susceptibility and response of wild waterfowl to duck plague virus. Acta Zoologica et Pathologica Antverpiensia 48: 373–383.

DAVIDSON, S., K. A. CONVERSE, A. N. HAMIR, AND R. J. ECKROADE. 1993. Duck viral enteritis in domestic muscovy ducks in Pennsylvania. Avian Diseases 37: 1142–1146.

DOCHERTY, D. E., AND P. G. SLOTA. 1988. Use of muscovy duck embryo fibroblasts for the isolation of viruses from wild birds. Journal of Tissue Culture Methods 11: 165–170.

ERICKSON, G. A., S. J. PROCTOR, J. E. PEARSON, AND G. A. GUSTAFSON. 1974. Diagnosis of duck virus enteritis. Proceedings of the American Association of Veterinary Laboratory Diagnosticians 1974: 85–90.

HANSEN, W. R., S. E. BROWN, S. W. NASHOLD, AND D. L. KNUDSON. 1999. Identification of duck plague virus by polymerase chain reaction. Avian Diseases 43: 106–115.

JANSEN, J. H. 1968. Duck plague. Journal of the American Veterinary Medical Association 152: 1009–1016.

KASCHULA, V. R. 1948. A new viral disease of the muscovy duck [(Cairina moschata (Linn)] present in Natal. Journals of the 5th African Veterinary Medical Association 17: 18–26.

LEIBOVITZ, L. 1968. Progress report: duck plague surveillance of American Anseriformes. Bulletin of the Wildlife Disease Association 4: 87–91.

———. 1969. The comparative pathology of duck plague in wild Anseriformes. The Journal of Wildlife Management 33: 294–303.

———. 1971. Gross and histopathologic changes of duck plague (duck virus enteritis). American Journal Veterinary Research 32: 275–290.

———, AND J. HWANG. 1968. Duck plague on the American continent. Avian Diseases 12: 361–378.

LIN, W., K. M. LAM, AND W. E. CLARK. 1984. Isolation of an apathogenic immunogenic strain of duck virus enteritis from waterfowl in California. Avian Diseases 28: 641–650.

MONTGOMERY, R. D., G. STEIN, JR., M. N. NOVILLA, S. S. HURLEY, AND R. J. FINK. 1981. An outbreak of duck virus enteritis (duck plague) in a captive flock of mixed waterfowl. Avian Diseases 25: 207–213.

NETTLES, V. F., AND E. T. THORNE. 1988. Guidelines for responding to duck plague outbreaks in captive/semi-captive flocks. Proceedings of the United States Animal Health Association 1998: 507–513.

NEWCOMB, S. S. 1968. Duck virus enteritis (duck plague) epizootiology and related investigations. Journal of the American Veterinary Medical Association 153: 1897–1902.

PEARSON, G. L., AND D. R. CASSIDY. 1997. Perspectives on the diagnosis, epizootiology, and control of the 1973 duck plague epizootic in wild waterfowl at Lake Andes, South Dakota. Journal of Wildlife Diseases 33: 681–705.

SAMUEL, M. D., D. R. GOLDBERG, D. J. SHADDUCK, J. I. PRICE, AND E. G. COOCH. 1997. Pasteurella multicoda Serotype 1 isolated from a lesser snow goose: Evidence of a carrier state. Journal of Wildlife Diseases 33: 332–335.

———, M. D., D. J. SHADDUCK, D. R. GOLDBERG, V. BARANYUK, L. SILEO, AND J. I. PRICE. 1999. Antibodies against Pasteurella multocida in snow geese in the western arctic. Journal of Wildlife Diseases 35: 440–449.

SPIEKER, J. O., T. M. YUILL, AND E. BURGESS. 1996. Virulence of six strains of duck plague virus in eight waterfowl species. Journal of Wildlife Diseases 32: 453–460.

SYNDER, S. B., J. G. FOX, L. H. CAMPBELL, K. F. TAM, AND O. A. SOAVE. 1973. An epornitic of duck virus enteritis (duck plague) in California. Journal of the American Veterinary Medical Association 163: 647–652.

WALKER, J. W., C. J. PFOW, S. S. NEWCOMB, W. D. URBAN, H. E. NADLER, AND L. N. LOCKE. 1969. Status of duck virus enteritis (duck plague) in the USA. Proceedings of the United States Animal Health Association 1969: 254–279.

WOBESER, G. A., AND D. E. DOCHERTY. 1987. A solitary case of duck plague in a wild mallard. Journal of Wildlife Diseases 23: 479–482.

———. 1997. Diseases of wild waterfowl. 2nd Edition. Plenum Press, New York, New York. 324 pp.

Received for publication 22 April 1998.