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THE 1992 EPIZOOTIC OF NEWCASTLE DISEASE IN DOUBLE-CRESTED CORMORANTS IN NORTH AMERICA

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ABSTRACT: In the summer of 1992, morbidity and mortality in juvenile double-crested cormorants (*Phalacrocorax auritus*; DCC) attributable to Newcastle disease virus (NDV) was observed for the first time in seven northern USA states and one Canadian province, and recurred in three western Canadian provinces. Based on clinical signs and laboratory diagnostic findings, DCC mortality from NDV occurred in 59 of the 63 nesting colonies and two of three non-colony sites investigated. An estimate of in excess of 20,000 DCC died, with mortality rates ranging from <1 to 37% in Great Lakes colonies to 20 to 92% in Minnesota (USA) and North and South Dakota (USA) colonies. Sick juvenile white pelicans (*Pelecanus erythrorhynchos*) exhibiting signs similar to sick cormorants, and dead pelicans were observed in Minnesota and North Dakota. Mortality rates in pelican colonies were as high as in the adjacent cormorant colonies, but no cause for the mortality of an estimated 5,000 pelicans was determined. No evidence of NDV was found in other species nesting in proximity to affected cormorants. Although the source of the NDV infection is unknown in cormorants, the simultaneous onset of the epizootics in juvenile birds over a wide geographic area implies that the virus was acquired by adults prior to migration and was carried back to nest sites, exposing susceptible nestlings. The possible transmission of this virus from free-ranging wild birds to domestic poultry is a concern. Based on repeated epizootics in cormorants since 1990, NDV seems to be established in DCC.

Key words: Double-crested cormorant, epizootiology, mortality, Newcastle disease, Newcastle disease virus, *Phalacrocorax auritus.*

INTRODUCTION

Newcastle disease (ND), caused by strains of avian paramyxovirus-1 virulent for poultry, was first recognized in domestic fowl in the Dutch East Indies (Indonesia) and in Newcastle-upon-Tyne, England in 1926 (Alexander, 1997). Newcastle Disease Virus (NDV) is capable of infecting a wide variety of birds (Alexander, 1986) and certain NDV strains have traversed the globe in panzootic style manifesting new forms of the disease. Most virulent strains of avian paramyxovirus-1 infect captive parrots and domestic poultry but NDV also has been isolated from wild birds in several parts of the world (Alexander, 1997; Ritchie, 1995). Epizootics of ND were documented in shags (*Phalacrocorax aristotelis*) in Great Britain in 1949 (Blaxland, 1951) and in double-crested cormorants (*Phalacrocorax auritus*; DCC) in Canada in 1975 and 1990 (Wobeser et al., 1993). Prior to the 1992 outbreak, NDV had not been reported as a cause of mortality in native wild birds in the USA.

In the summer of 1992, sick and dead juvenile DCC were observed in nesting colonies over a wide geographic area in seven northern states of the USA and four Canadian provinces. Clinical signs in sick birds suggested neurological disease and NDV was isolated from affected cormorants and characterized, and associated lesions were described (Heckert, 1993; Banerjee et al, 1994; Heckert et al., 1996; Meteyer et al., 1997). The 1992 event is the first recorded outbreak of neurotropic

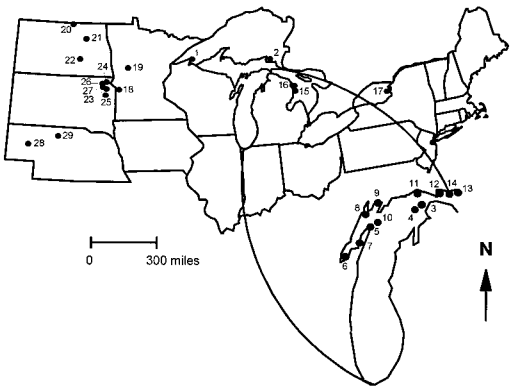

FIGURE 1. Locations in the USA investigated in 1992 for evidence of Newcastle disease in double-crested cormorants (see Table 1 for name and latitude/longitude of each numbered site).

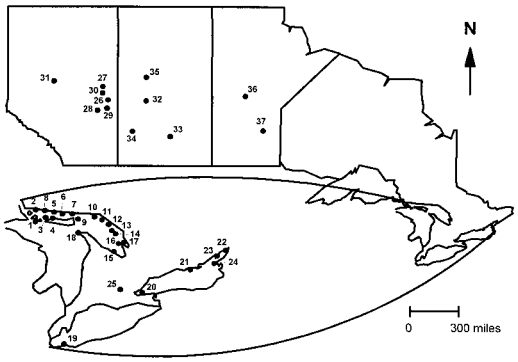

FIGURE 2. Locations in Canada investigated in 1992 for evidence of Newcastle disease in double-crested cormorants (see Table 2 for name and latitude/longitude of each numbered site).

NDV in free-living DCC in the USA. We summarize information on cormorant mortality in the USA and Canada and on morbidity and mortality in other colonial waterbird species in close proximity to affected cormorant colonies.

MATERIALS AND METHODS

Following initial reports of mortality in DCC in Minnesota (USA), Michigan (USA), and Ontario (Canada) in late June and early July 1992 disease alerts were distributed in the USA by the National Wildlife Health Center (NWHC) and in Canada by wildlife disease workers in several provinces. The reports described signs of ND in affected cormorants and provided general information on NDV, including the concern that humans can spread the disease to other birds. Descriptions of abnormal behavior and morbidity/mortality in juvenile DCC and other species of colonially nesting waterbirds were solicited from wildlife personnel by the same agencies that distributed the disease alerts. In addition to colonies of DCC visited annually in the course of population or other research, some normally unobserved DCC colonies were investigated to determine the extent and estimate the impact of the disease.

Sixty-three primarily ground-nesting colonies of DCC were investigated for the presence of colonial waterbird morbidity and mortality. Data from three additional non-colony sites in Canada are incorporated here also (Figs. 1, 2). The time spent at each colony site was limited to minimize human impact on nesting birds. USA nesting colonies were visited prior to the time that offspring normally fledge, at approximately 6 wk of age. In some colonies, logistical reasons (colony size, disturbance of birds, density of vegetation) dictated that only a portion of the colony was observed. Nests in trees were observed from the ground only. Sick and dead birds were counted. In colonies that could not be examined entirely, estimates of the number of sick and dead birds and the juvenile population size were based on counting parts of the colony. To estimate the juvenile population, the number of active nests was multiplied by two, assuming two fledged young per nest (Weseloh et al., 1995). The parts of the colony that were counted were then multiplied by an appropriate factor based on an estimate of the proportion of the entire colony which had been examined. In Canadian Great Lakes colonies, some of which were visited after fledging, methods were similar, but affected birds were counted both on the colony and in near shore water. Field investigations in western Canada were less systematic, and only anecdotal retrospective information was available in some cases.

Samples were collected for further diagnostic investigation from 50 of these sites. In the USA, freshly dead carcasses or euthanized birds were either necropsied outside the nesting area at the site or shipped to a diagnostic laboratory for necropsy. Tissue samples for virus isolation taken from carcasses necropsied in the field were stored in cryovials in a liquid nitrogen dry shipper and tissues for histopathology were fixed in 10% neutral buffered formalin. All necropsies were performed in the laboratory in Canada.

In the USA, necropsies and diagnostic evaluations of 114 DCC from 24 colonies were performed: NWHC (Madison, Wisconsin, USA; 93 DCC); Animal Health Diagnostic Laboratory

(East Lansing, Michigan, USA; nine DCC; Banerjee et al., 1994); Northeastern Research Center for Wildlife Diseases, (Storrs, Connecticut, USA; two DCC); and USDA National Veterinary Services Laboratory (Ames, Iowa, USA; 10 DCC; Meteyer et al., 1997). In addition, necropsies were performed at the NWHC in the same manner as the DCC with the same laboratory procedures on 42 birds of five other species; 24 white pelicans (*Pelecanus erythrorhynchos*), four unidentified gulls, two Franklin's gulls (*Larus pipixcan*), and five ring-billed gulls (*Larus delawarensis*) from midwest locations, and three herring gulls (*Larus argentatus*), two ring-billed gulls and two black-crowned night-herons (*Nycticorax nycticorax*) from Great Lakes locations.

In Canada, 67 DCC from 19 locations were necropsied by five laboratories: Alberta Agriculture (Edmonton, Alberta, Canada; 13 DCC); Western College of Veterinary Medicine (Saskatoon, Saskatchewan, Canada; 21 DCC); Manitoba Department of Agriculture (Winnipeg, Manitoba, Canada; five DCC); Ontario Veterinary College (Guelph, Ontario, Canada; 25 DCC); Ontario Ministry of Agriculture, Food and Rural Affairs (Brighton, Ontario, Canada; three DCC), generally as described above. Virus isolation was carried out by inoculation of embryonated eggs via the allantoic sac (Senne, 1989). All viral isolates were sent to Agriculture and Agrifood Canada (Hull, Quebec, Canada) for confirmation of diagnosis and evaluation of virulence in domestic poultry (Heckert, 1993). Necropsies and subsequent laboratory investigations also were performed on two white pelicans and two unspeciated gulls from sites other than the Great Lakes in Ontario, and two Caspian terns (*Sterna caspia*), one herring gull, four ring-billed gulls, and two black-crowned night-herons from the Great Lakes region in Ontario in the same manner as on DCC.

For each DCC colony, we tabulated estimated mortality (number of dead juveniles/estimated juvenile population) and presence/absence of clinical signs compatible with ND, microscopic lesions characteristic of ND in DCC (Wobeser et al., 1993; Banerjee et al., 1994; Meteyer et al., 1997), seropositive birds, and virus positive birds (Tables 1, 2).

One hundred forty-six clinically affected DCC from nine colonies in the USA and 152 normal or clinically affected DCC from 13 colonies in Canada were examined serologically (Tables 1, 2). In the USA, blood was collected from the jugular vein of affected cormorants, allowed to clot, centrifuged and the serum was decanted and stored in a plastic vial chilled or frozen for transport to the laboratory. In Canada, blood was collected from the brachial vein, placed on ice or refrigerated as whole blood, and transported to the laboratory within 2 days of collection. Serum was tested for antibodies to NDV by hemagglutination inhibition (HI) (Canada—Allan and Gogh, 1974; USA—Meteyer et al., 1997). A serum was considered "positive" if it reacted with an HI titer to NDV of $\geq$1:8 (Canada) or $\geq$1:10 (USA).

RESULTS

In cormorants, neurological signs compatible with ND were recognized only in young of the year, usually in >3-wk-old birds. The signs included paresis or paralysis of one or both legs, sometimes with feet clenched; paresis or paralysis of one or both wings; inability to hold affected wings tucked against the body; drooping of the head; torticollis; deviation of the tail from midline; tremors; and inability or reluctance to move. Some younger birds were found sick or dead in nests, sometimes with apparently normal nestmates. Older fledged birds with wing or leg paresis, often unilateral, were observed attempting to walk, swim, dive, or fly.

Estimated mortality of juvenile cormorants in the affected midwest USA colonies ranged from 4 to 92% and estimated mortality in the Great Lakes (USA and Canada) colonies ranged from <1 to 37%. Mortality was not calculated for some Canadian colonies. The total number of dead DCC was estimated conservatively at 5,000 birds in the USA and in excess of 15,000 in Canada (Tables 1, 2).

In the USA, sick and dead juvenile white pelicans, Franklin's gulls, ring-billed gulls, and herring gulls were observed in nesting colonies, most of which were in close proximity to affected cormorant colonies. Sick pelicans exhibited behavior similar to sick cormorants, including unilateral or bilateral wing and or leg paralysis/paresis, drooping neck, and an inability or reluctance to move. Abnormal behavior seen in gulls was limited to unilateral or bilateral wing and leg paralysis/paresis. Approximately 5,000 juvenile pelicans died, with the majority of birds found at the

TABLE 1. Locations in the USA investigated in 1992 for evidence of Newcastle disease in double-crested cormorants.

Site	Lat/Long	Dates	Juv Mort[a]	% Mort[b]	Signs[c]	Histol[d]	Serol[e]	Virol[f]
Lake Superior								
1. [g] Gull Island	46°54′N 90°26′W	7/20, 8/19	256	22%	+	+	−	−
2. Taquamenon Island	46°31′N 84°56′W	6/22, 7/14	125	18%	+	+	+	+
Lake Michigan								
3. Hat Island	45°48′N 85°18′W	6/29, 7/10, 16	53	7%	+	+		+
4. Grape Island	45°46′N 85°25′W	6/29, 7/11, 16	87	9%	+	+		+
5. Hat Island	45°06′N 87°19′W	7/14, 21	4	<1%	+	+	+	+
6. Cat Island	44°33′N 88°00′W	7/28	6		+	−		−
7. Spider Island	45°12′N 86°58′W	wkly. 6/30–9/16	225	4%	+	+	+	+
8. Fisherman's Island		7/9	79	13%	+			
9. Snake Island	45°44′N 86°39′W	6/26	7	<1%	+	+[i]		+[h]
10. Little Gull Island	45°29′N 86°42′W	6/27, 7/9, 29	136	9%	+	+	+	+
11. Naubinway Island	46°04′N 85°26′W	6/28, 8/19	30	6%	−			
Lake Huron								
12. St. Martin's Shoal	45°26′N 86°46′W	6/23, 7/13, 8/2	115	7%	+	+	+	−
13. West Saddlebag Reef	45°57′N 84°02′W	7/14	13	4%	+	−	+	+
14. Goose Island	45°55′N 84°25′W	7/13	none					
15. Scarecrow Island	44°54′N 83°19′W	7/29	6	1%		+	+	−
16. Bird Island	44°53′N 83°19′W	7/29	5	1%		+		+
Lake Ontario								
17. Little Galloo Island	43°53′N 76°23′W	7/?, 8/17	496	5%	+			+[h]
Minnesota								
18. Marsh Lake	45°09′N 96°10′W	6/20–7/29, 8/10	620	78%	+	+	+	+
19. Chautauqua Lake	46°14′N 96°00′W	7/13, 20, 27, 9/1	734	92%	+	+		+
North Dakota								
20. Willow Lake	48°55′N 100°08′W	7/23, 27, 8/5	330	44%	+	+		−
21. Devil's Lake	47°55′N 98°48′W	8/8, 13, 19, 28, 9/1	1024	41%	+	+		+[h]
22. Chase Lake	47°00′N 99°26′W	7/21, 8/03	81	46%	+	+		−

TABLE 1. Continued.

Site	Lat/Long	Dates	Juv Mort[a]	% Mort[b]	Signs[c]	Histol[d]	Serol[e]	Virol[f]
South Dakota								
23. Bitter Lake	45°16′N 97°18′W	7/10, 21, 8/1, 17	36	36%	+	−		−
24. Drywood Lake	45°35′N 97°10′W	7/10–8/15	20	20%	+			
25. Grass Lake	45°03′N 97°22′W	7/16, 8/1	145	48%	+	−		−
26. Piyas Lake	45°35′N 97°20′W	7/10–8/15	140	47%	+			
27. South Waubay Unit	45°23′N 97°27′W	7/9, 10, 13, 21, 8/1	400	67%	+	+		+
Nebraska								
28. Crescent Lake	41°43′N 102°29′W	7/21, 8/3, 2, 9/1	66	15%	+	+		+
29. Valentine NWR	42°18′N 101°00′W	7/16, 8/10	80	4%	+	−		−

[a] Juv Mort—juvenile double-crested cormorant (DCC) mortality.
[b] % Mort—mortality represented as a percentage of the estimated juvenile DCC population.
[c] "Signs"—clinical signs characteristic of confirmed cases of Newcastle Disease (ND) were observed in live DCC (+), not observed in live DCC (−), or no live DCC were observed ( ) at these sites.
[d] "Histol"—characteristic microscopic lesions of ND were recognized (+), not recognized (−) or not looked for ( ) in DCC at these sites.
[e] "Serol"—an HI titer of ≥1:10 to Newcastle disease virus (NDV) was found (+), was not found (−), or antibody titer was not evaluated ( ) from DCC at these sites.
[f] "Virol"—NDV was isolated (+), was not isolated (−) or isolation was not attempted ( ) from DCC at these sites.
[g] Location numbers correspond to numbered locations in Figure 1.
[h] Virus isolation at the USDA National Veterinary Services Laboratory in Ames, Iowa.
[i] Diagnostic evaluation at the Animal Health Diagnostic Laboratory, Michigan State University, East Lansing, Michigan (Banerjee et al., 1994).

TABLE 2. Locations in Canada investigated in 1992 for evidence of Newcastle disease in double-crested cormorants.

Site	Lat/Long	Dates	Juv Mort[a]	% Mort[b]	Signs[c]	Histol[d]	Serol[e]	Virol[f]
Lake Huron								
1. [g] Wheeler Reef	45°54′N 83°31′W	6/23–7/24	343	37%	+			
2. Middle Grant Island	46°08′N 83°20′W	7/7, 17, 24, 8/1	152	18%	+		+	
3. West Rock	46°06′N 82°12′W	7/18, 8/1	347	15%	+			
4. Cousins Island	46°05′N 82°49′W	7/12–7/24	125	23%	+		+	
5. Doucet Rock	46°08′N 82°51′W	7/6	68	14%	+	+	+	+
6. Gull Rock	46°06′N 82°19′W	7/19	39	10%	+			
7. Elm Island	46°01′N 82°08′W	7/4, 11, 19	93	19%	+		−	
8. Kalulah Rock	46°16′N 83°36′W	7/8	4	17%	+	+	−	−
9. West Rock	45°50′N 81°28′W	7/8, 22	494	19%	+		+	
10. Gull Island	45°51′N 81°16′W	7/8, 22	172	22%	+		+	
11. SW Gull Rock	45°12′N 80°13′W	7/21	54	8%	+			
12. Wallis Rock	45°24′N 80°33′W	7/23	50	24%	+	+		+
13. Little McCoy's	45°27′N 80°29′W	7/23	94		+			
14. S. Limestone Is.	45°23′N 80°32′W	7/23	none					
15. Nottawasaga Island	44°32′N 80°16′W	8/5	none					
16. North Watcher	44°58′N 80°04′W	8/6	43					
17. South Watcher	44°57′N 80°04′W	8/6	86		+			
18. Tobermory	45°15′N 81°40′W	10/2	2			−		−
Lake Erie								
19. East Sister Island	41°49′N 82°52′W	7/29	6		+		+	
Lake Ontario								
20. Hamilton Harbor	43°15′N 79°51′W	7/29–8/21	no est.		+	+	+	−
21. Gull I.–Presqu'le	43°59′N 77°44′W	7/29–8/17	227	15%	+	+	+	−
22. Pigeon Island	44°04′N 76°33′W	7/22	94	6%	+		+	
23. Salmon Island	44°12′N 76°36′W	8/16	none					
24. Snake Island	44°11′N 76°33′W	8/7	34	6%	+	+		−
25. Eramosa Township	43°40′N 80°15′W	8/10	1		+	+		+

TABLE 2. Continued.

Site	Lat/Long	Dates	Juv Mort[a]	% Mort[b]	Signs[c]	Histol[d]	Serol[e]	Virol[f]
Alberta								
26. Muriel Lake	54°10′N 110°40′W	7/30	290	12%	+	+		+[h]
27. Portage Lake	55°58′N 112°04′W	8/5	700	50%	+	+		−
28. Lower Therien Lake	53°55′N 111°20′W	8/5	260	65%	+	+		−
29. Canard Lake	53°52′N 111°21′W	8/6	300	51%	+	+		−
30. Lac la Biche	54°46′N 112°00′W	8/4	350	22%	+	+		−
31. Utikima Lake	55°50′N 115°20′W	9/4	no est.		+	+		+[h]
Saskatchewan								
32. Redberry Lake	52°40′N 107°10′W	7/21	20+	10%	+	+	+	+
33. Last Mountain Lake	51°10′N 105°15′W	7/21	no est.		+	+	+	+
34. Kindersley	51°28′N 109°10′W	8/25	1		+	+		+
35. Doré Lake	54°45′N 107°17′W	9/10	1–3K		+	−		−
Manitoba								
36. L. Winnipegosis	52°30′N 100°00′W	8/6	10–20K		+	+		+
37. Lake Winnipeg	53°N 98°W	unknown	no est.		+			

[a] Juv Mort—juvenile double-crested cormorant (DCC) mortality.
[b] % Mort—mortality represented as a percentage of the estimated juvenile DCC population.
[c] "Signs"—clinical signs characteristic of confirmed cases of Newcastle Disease (ND) were observed in live DCC (+), not observed in live DCC (−), or no live DCC were observed ( ) at these sites.
[d] "Histol"—characteristic microscopic lesions of ND were recognized (+), not recognized (−) or not looked for ( ) in DCC at these sites.
[e] "Serol"—an HI titer of ≥1:8 to Newcastle disease virus (NDV) was found (+), was not found (−), or antibody titer was not evaluated ( ) from DCC at these sites.
[f] "Virol"—NDV was isolated (+), was not isolated (−) or isolation was not attempted ( ) from DCC at these sites.
[g] Location numbers correspond to numbered locations in Figure 2.
[h] A hemagglutinating agent compatible with NDV was isolated (+) from this site.

Marsh Lake (Minnesota) and Chase Lake (North Dakota) colony sites where mortality rates were estimated at ≥90%. Gulls were submitted from North Dakota, Lake Michigan, and Lake Huron. The highest mortality in gulls was recorded from Chase Lake and Devil's Lake in North Dakota, and High Island in Lake Michigan. The estimated loss was about 3,100 birds.

In Canada, greater-than-expected numbers of dead pelicans and gulls were reported from Redberry Lake and Doré Lake (Saskatchewan) but not from other locations. Although two dead pelicans were submitted from Lake of the Woods in northwestern Ontario, greater-than-expected mortality in pelicans could not be confirmed at this locality. Observation of colonies of Caspian terns, and ring-billed and herring gulls on Lakes Erie, Huron and Ontario usually did not reveal abnormal mortalities, even when these species shared ground with cormorants. Signs in 14 herring gulls observed on Nottawasaga Island in Georgian Bay (Lake Huron) were considered compatible with botulism by the observers, but these birds were not submitted to the laboratory. Two sick and three dead black-crowned night-herons were observed on Snake Island (Lake Ontario).

Double-crested cormorant mortality attributable to ND was present at 27 USA colony sites, 30 Canadian colony sites, and at two non-colony sites in Canada (Tables 1, 2). Affected colonies were scattered over a wide geographic area, from northern Alberta (Canada), through the upper midwest of the USA, and involved all of the Great Lakes as far as the east end of Lake Ontario (Figs. 1, 2). Clinical signs in breeding colonies were reported from the third week in June to the end of August in the USA, and from the fourth week of June to mid-August in Canada (Tables 1, 2). Individual flightless cormorants with clinical signs consistent with ND, particularly unilateral wing paralysis, were reported on the lower Great Lakes into late autumn.

Necropsy findings have previously been reported in a subset of American cormorants from this epizootic (Banerjee et al., 1994; Meteyer et al., 1997) and no consistent significant gross lesions were found in the additional DCC reported here, from the USA or Canada. Newcastle disease virus was isolated from birds from 15 USA colony sites in Minnesota, Nebraska, North Dakota and South Dakota, Lake Superior, Lake Michigan, Lake Huron and Lake Ontario (Table 1). Newcastle disease virus was isolated from five Canadian colony sites and two non-colony sites in Saskatchewan, Manitoba, Lake Huron and Lake Ontario; a hemagglutinating agent compatible with NDV was isolated from two colony sites in Alberta (Table 2). Virus from the Marsh Lake (Minnesota) site was characterized as neurotropic velogenic NDV for domestic poultry (Meteyer et al., 1997) as was a virus isolated from Michigan (Banerjee et al., 1994). NDV isolates from DCC in Canada were variously characterized as mesogenic ($n = 3$), or velogenic neurotropic ($n = 4$), by Agriculture Canada (Heckert, 1993); isolates from Alberta could not be recovered for characterization. Four USA and eight Canadian colony sites had DCC with clinical signs and microscopic lesions consistent with ND, but from which the virus could not be isolated (Tables 1, 2). Virus was not isolated from DCC collected in the USA after 1 August 1992. The DCC collected in Canada after 10 August 1992 did not have microscopic lesions characteristic of ND nor was NDV isolated from tissues. NDV was not isolated from birds which did not have microscopic lesions characteristic of ND. Forty-four of 146 DCC sampled in eight of nine colonies in the USA had antibody to NDV at a titer of ≥1:10 (Table 1). In Canada, 37/152 DCC, involving 11 of 13 colonies sampled (Table 2), had antibody to NDV at a titer of ≥1:8.

Diagnostic evaluation in the USA of 21 of 24 white pelicans, 10 of 16 gulls, and both black-crowned night-herons found them to be in poor to emaciated body con-

dition with an undetermined etiology. Six of 24 pelicans had moderate to severe gastric parasitism; one of six gulls in good body condition had ingested a foreign body, one had trauma, and four died of undetermined cause. In Canada, all gulls and both pelicans examined were in poor to emaciated condition with an undetermined etiology. One Caspian tern had septic arthritis of the shoulder; the other had no lesions identified. Both black-crowned night-herons were emaciated, with heavy gastric parasitism. Virus was not isolated from any of the birds examined and microscopic lesions compatible with ND (non-suppurative encephalitis) were seen only in one of two sick black-crowned night-herons examined from Snake Island (Lake Ontario). Of 149 gulls and terns from Ontario examined serologically, only one ring-billed gull from Gull Island (Lake Ontario) had a weak positive titer to NDV (1:32).

DISCUSSION

The clinical signs and microscopic lesions in the DCC described in this paper were similar to those attributed to neurotropic paramyxovirus-1 infections in domestic and wild birds, including DCC (Heckert, 1993; Wobeser et al., 1993; Banerjee et al., 1994; Alexander, 1997). Spasticity, tremors and frequent asymmetry of paralysis serve to differentiate clinical ND from other paralytic diseases that might affect colonially nesting birds, including botulism (Wobeser, 1981), and domoic acid intoxication in marine environments (Fritz et al., 1992). NDV isolation rates may have underestimated prevalence of infection, possibly due to interference of antibody with virus isolation in some cases, or reduction in virus titer below detectable levels in chronically affected survivors (Wobeser et al., 1993).

Breeding populations of DCC have increased dramatically in the last 20 yr. This is attributed to decreased organochlorine pesticide contamination, increased protection (Ludwig, 1984; Price and Weseloh, 1986), and an increased food supply, primarily alewife (*Alosa pseudoharengus*) (Weseloh et al., 1995). Populations in colonies on the Great Lakes where ND occurred in 1992 were reported to be at an all time high in 1991 (Weseloh et al., 1995). A large DCC population, high nesting densities, heavy contamination with excreta, and older juveniles traveling in groups in and around the colonies, were favorable for the transmission of an infectious agent.

This outbreak involved six of the 10 DCC colonies known to have been affected by ND in western Canada during 1990 (Wobeser et al., 1993). However, the geographic distribution in 1992 was apparently much greater, extending south and east through the Dakotas, Nebraska, and Minnesota to involve the entire Great Lakes basin. The isolated nature of cormorant colonies and lack of observation undoubtedly resulted in some affected colonies being missed during both outbreaks. Many Great Lakes colonies were observed at least annually during the nesting season (Weseloh et al., 1995). Unless colonies were visited prior to an outbreak, it is probable that some evidence of the disease, in the form of abnormally large numbers of sick and dead birds, or survivors with residual signs of paralysis, would be noted. Therefore, the more widespread detection of ND in cormorants in 1992 was probably real and not solely due to increased surveillance. Despite the fact that ND was not observed in DCC on the Great Lakes prior to 1992, antibody to NDV was present in eggs from Great Lakes colonies in 1991 (Wobeser et al., 1993), evidence of previous infection with NDV or a cross-reacting paramyxovirus in that population (Alexander, 1997). Whether that exposure occurred on the wintering or breeding grounds is unknown.

The circumstances under which estimates of mortality were made dictate that they can be considered only approximate. At some localities in the midwestern USA the impact of ND on juvenile cormorants

was high (>50% mortality), while on the Great Lakes, generally <20% of offspring were killed. Since observations at a given colony were often conducted only once, apparent geographic variations in morbidity and mortality among colonies may simply reflect the fact that populations were observed at different points on the epidemic curve. Variations in the number of susceptible birds also may have occurred, due to differences in prevalence and concentration of maternal antibody reflecting the infection experience of the mother, and/or the time of introduction of virus in relation to the decline of maternal antibody concentration in the chicks.

In a rapidly expanding DCC population, such as that on the Great Lakes, recruitment rates may be as high as 50–70% (Price and Weseloh, 1986). In such a circumstance, 20% fledgling mortality in one year would cause only a minor transient decline in the rate of population growth. In a more stable population, with a much lower annual rate of recruitment (Ludwig, 1974), even a 90% mortality rate in 1 yr will cause only a minor decline in absolute numbers of birds recruited to the population. While comparisons with the impact of the 1990 outbreak in western Canada are difficult, in absolute terms DCC mortality in that region during 1992 seems to have been similar.

Failure to diagnose ND in other colonial nesting birds, especially pelicans, gulls, and terns, at many of these same locations is a conundrum. Neither microscopic lesions compatible with ND, nor NDV, were demonstrated in affected birds from colonies in the midwest of the USA where mortalities were high, despite the fact that both had been demonstrated in pelicans and ring-billed gulls in the 1990 Canadian outbreak (Wobeser et al., 1993). In Canada, there was little field or laboratory evidence of ND in species other than DCC, with the exception of a single black-crowned night-heron, and a single ring-billed gull seropositive at low titer.

The source of the NDV which caused this outbreak is unknown. Since the disease erupted simultaneously over a wide geographic area, in similarly-aged juvenile birds, the virus was most likely carried to nesting sites by adults infected prior to the northward spring migration (Meteyer et al., 1997). Migrating DCC that nest over a wide geographic range congregate on wintering areas (Dolbeer, 1991) that extend from the Gulf coast of Mexico and Texas to Florida, and include the Mississippi River delta in northwestern Mississippi. A wintering site could be a source of exposure to a virus maintained within a subpopulation of cormorants, or in other wild or domestic avian species. However, no mortality has been reported in DCC on wintering grounds, nor have epizootics of velogenic NDV in poultry been reported from the southern USA. Velogenic NDV epizootics in domestic poultry occur in Mexico (P. Fernandez, pers. comm.) but information on outbreaks where cormorants potentially may have been exposed is not available.

The fact that several epizootics of ND have been recognized in DCC in North America implies that NDV may be maintained in this species. This is supported by antibody titers in DCC eggs indicating that NDV or an antigenically related virus was circulating in Great Lakes colonies prior to the epizootics in 1992 (Wobeser et al., 1993). In 1993, eggs with positive antibody titers to NDV (>1:20) were found in 19 of 20 DCC colonies located in coastal British Columbia, Alberta, Saskatchewan, Manitoba, the Great Lakes (Ontario), the St. Lawrence River (Quebec), and coastal Prince Edward Island. The single negative colony was in Saskatchewan. The prevalence and magnitude of titers appeared to be greater in western Canada than in the Great Lakes and eastern Canada (T. Bollinger, unpubl. data). Despite surveillance of most USA DCC nesting colonies in the summers of 1993 and 1994, no mortality attributable to ND was detected (L. C. Glaser, unpubl. data). In Canada in 1995 and 1996, ND was diagnosed in low num-

bers (<5 cases/yr) in DCC and a Caspian tern on Lakes Erie and Ontario and environs, on Prince Edward Island (Canada), and in New Brunswick (Canada), but local mortality was <1% (G. D. Campbell, P.-Y. Daoust, and J. Shapiro, pers. comm.). However, in 1995, an estimated 32 to 64% of juvenile DCC at Doré Lake (Saskatchewan) died of ND (T. Kuiken, pers. comm.). In 1997, morbidity and mortality attributable to NDV occurred in two Saskatchewan colonies (Kuiken et al., 1998), and in three DCC colonies in three different states west of the Rocky Mountains in the USA (L. C. Glaser, unpubl. data), where ND had not been observed previously.

The epidemiological factors that dictate recurrent local or extensive epizootics of ND in DCC are unclear. The age class affected (mainly unfledged juvenile DCC over 2 to 3 wk of age) suggests that maternal antibody in eggs (Wobeser et al., 1993) may protect hatchlings against infection. However, the likelihood that the virus is exchanged on the wintering grounds and introduced into the breeding colonies by adult birds in which clinical signs of ND have not been recognized suggests that older birds, while susceptible to infection, are resistant to disease. The transformation of a low-virulence virus to a velogenic form of NDV in DCC might explain the sporadic nature of epizootics but this phenomenon would be difficult to document.

While the viruses isolated were described as variably mesogenic or velogenic based on pathogenicity indices in North America, they are considered an unequivocal threat to domestic poultry (Heckert et al., 1996). Although DCC are not typically in contact with domestic poultry operations, other species, such as gulls, frequent both cormorant colonies and poultry operations. An outbreak of velogenic ND occurred in a range flock of turkeys in North Dakota in August 1992 about 5.5 km from a DCC colony with ND mortality (Mixon and Pearson, 1992). Newcastle disease virus indistinguishable from the 1992 cormorant virus was isolated from several infected turkeys in this range flock (Heckert et al., 1996), which was eradicated. Diagnostic evaluation of other species, such as gulls, that nested in close proximity to affected cormorants, and in some localities were showing clinical signs similar to cormorants with confirmed ND, was pursued based on the concern that poultry more likely would be exposed to these species. Although no concrete evidence of NDV etiology was found, the possibility of infection in these species closely associated with infected DCC cannot be ruled out, since NDV identical to that in DCC was isolated from a pelican and a ring-billed gull during the 1990 DCC outbreak in Canada (Heckert et al., 1996).

Based on outbreaks at intervals since 1990, NDV seems to be established in the DCC population. Continued surveillance of free-flying colonial waterbird species is needed to detect and document further the epidemiology and impact of NDV on their populations, and on domestic poultry.

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