Saskatchewan

Newcastle disease in cormorants

Newcastle disease was diagnosed in double-crested cormorants (*Phalacrocorax auritus*) from central Saskatchewan in summer 1997. This is the 4th year in which this disease has been found in cormorants since it was first reported in 1993 (1).

Disease was first noted on August 2, 1997, when one cormorant with bilateral paralysis of wings and legs was found on Island A, Doré Lake, where about 3000 pairs of cormorants and 500 pairs of American white pelicans (*Pelecanus erythrorhynchos*) breed. On a second visit 3 wk later, there were at least 93 cormorant carcasses and 34 cormorants with neurologic signs, including paralysis of the wings, legs, or both, and torticollis. On August 10, 1997, similar clinical signs were observed in about 25 cormorants on Pelican Island, Lavallée Lake, a colony site with about 3500 pairs of cormorants and 8000 pairs of pelicans; 116 cormorant carcasses were found there 2 mo later. All cormorants involved were fledged young of the year. There was no evidence of neurologic disease or unusual mortality in pelicans or other birds in the area.

Eight affected cormorants were examined by necropsy. All had histopathology consistent with Newcastle disease, including a nonsuppurative encephalomyelitis with neuronal necrosis, perivascular cuffing, and gliosis. Newcastle disease virus was isolated from one bird from each colony following inoculation of pooled tissues into embryonated chicken eggs. The virus isolate from Doré Lake had an intracerebral pathogenicity index (ICPI) of 1.56 and an intravenous pathogenicity index (IVPI) of 1.09; that from Lavallée Lake had an ICPI of 1.72 and an IVPI of 1.62. According to European Community standards (2), to which Canada adheres, both isolates were classified as pathogenic.

Newcastle disease was previously diagnosed in cormorants from Saskatchewan and other parts of Canada in 1990 (1), 1992 (3), and 1995 (4). In 1992, cormorants from the north central states of the USA were also affected (5). The repeated occurrence of Newcastle disease in cormorants and the genotypic stability of the isolates from different years (6) suggest that pathogenic Newcastle disease is circulating within double-crested cormorant populations in North America. This virus, which is a reportable disease agent, is infectious for other birds and transmission from cormorants to a commercial turkey flock in North Dakota has been recorded (6). **Those involved in poultry farms, wildlife rehabilitation centers, and other bird collections should remain aware of the risk of transmission of Newcastle disease from cormorants to the birds in their care.**

References

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Quebec

Distribution of *Streptococcus suis* capsular types in 1997

From January to December 1997, 450 streptococcal isolates were received in our laboratory as *Streptococcus suis* for serotyping. Of these, 358 were from different veterinary diagnostic laboratories in Quebec; the other isolates were received from other Canadian provinces and the United States. Serotyping was carried out using the coagglutination test. The capsular types of the 450 isolates are given in Table 1.

In 1997, capsular type 2 had a prevalence of 18%, which was identical with that reported in 1996 (1). The other frequent serotypes were, in decreasing

order, capsular types 3, ½, 7, 8, 4, and 1. Capsular types 5, 23, and 34 had a prevalence of 2%. Capsular types 6, 12, 19, 20, 24, 26, 32, and 33 were not found in 1997. The distribution of capsular types received from the United States and from Canadian provinces, other than Quebec, was similar to that in Quebec (data not shown).

Table 2 compares the distribution of the 6 most prevalent *S. suis* capsular types between 1990 and 1997. As reported in past years, about 60% of isolates belong to capsular types 2, ½, 3, 4, 7, and 8 (2); if the untypeable isolates are not taken into account, the percentage is more than 70%. The results support a proposal for diagnostic laboratories to perform serotyping with a limited number of antisera, and refer their untypeable isolates to a reference laboratory. The number of untypeable isolates is always important, but some of them are not capsulated upon arrival at our laboratory or show autoagglutination. Also, since we do not always carry out a complete biochemical identification on isolates from other laboratories, it is possible that some of them do not belong to the *S. suis* species.

References

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Table 1. Numerical distribution of capsular types of *Streptococcus suis* in 450 isolates recovered from diseased pigs in 1997

Capsular type	Number of isolates	%	Capsular type	Number of isolates	%
1	11	3	18	2	<1
2	83	18	19	0	0
½	49	11	20	0	0
3	49	11	21	1	<1
4	24	5	22	3	<1
5	9	2	23	9	2
6	0	0	24	0	0
7	30	7	25	1	<1
8	30	7	26	0	0
9	6	1	27	4	1
10	5	1	28	2	<1
11	1	<1	29	1	<1
12	0	0	30	3	<1
13	1	<1	31	1	<1
14	1	<1	32	0	0
15	1	<1	33	0	0
16	5	1	34	10	2
17	1	<1	NT	107	24

NT = Untypeable isolates

Table 2. Distribution in percentages of the 6 most prevalent *Streptococcus suis* capsular types between 1990 and 1997

Capsular type	1990	1991	1992	1993	1994	1995	1996	1997
2	32	21	23	19	24	18	18	18
½	9	12	13	8	9	14	8	11
3	14	12	13	10	10	12	14	11
4	4	4	5	3	5	8	5	5
7	3	7	7	7	6	8	10	7
8	7	6	7	8	7	7	6	7

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