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REVENGE OF THE TREES: ENVIRONMENTAL DETERMINANTS AND POPULATION EFFECTS OF INFECTIOUS DISEASE OUTBREAKS ON A BREEDING COLONY OF DOUBLE-CRESTED CORMORANTS (*PHALACROCORAX AURITUS*) OVER A PERIOD OF 21 YEARS

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ABSTRACT: During 16 of 21 consecutive annual breeding seasons, two diseases, Newcastle disease and avian cholera, killed approximately 50% of juvenile Double-crested Cormorants (*Phalacrocorax auritus*) in a large nesting colony in Canada. From 1994 to 2014, we recorded data annually on disease occurrence, causal pathogens, species and age classes affected, total number of breeding pairs of cormorants on the colony site, and other biological parameters. A mathematical model of pathogen transmission was constructed to assess the potential importance of transmission parameters and to test a hypothesis regarding the potential effect of the observed progressive loss of nest trees and the consequent shift from tree-nesting to ground-nesting behavior. The model indicated that juveniles from ground nests were 14 times more likely to die from epidemic disease (50.14% mortality) than were juveniles from nests in trees (3.57% mortality). Additive disease-related mortality of juvenile cormorants in the observed range of 40–60% would reduce a closed cormorant population over time. There was no directional change in the colony population during the study period, suggesting that immigration had compensated for disease-related mortality. Our results highlight the preeminent influence of environmental factors on pathogen transmission and the value of long-term data sets.

Key words: Avian cholera, Double-crested Cormorant, environmental determinant, Newcastle disease, *Phalacrocorax auritus*, population.

INTRODUCTION

One outcome of the past three decades of research and responses to new and newly important (emerging) infectious diseases has been a renewed recognition of the preeminence of environmental factors in determining the location, timing, scale, and outcome of epidemics in people and animals (Eisenberg et al. 2007). Short-term studies of such complex relationships can easily lead to false conclusions because both biological and environmental factors of major importance may fluctuate over time frames longer than the study periods (Pelton and van Manen 1996). In this article, we report the results of a continuous, multicomponent study of infectious disease epidemics on a large breeding colony of Double-crested Cormorants (*Phalacrocorax auritus*; hereafter, cormorants) in the southern boreal forest of Canada carried out from 1994 to 2014. We offer a hypothesis derived from these long-term data regarding environmental determinants of the epidemics observed, and explore the potential impacts of these epidemics on the colony population.

The Double-crested Cormorant is abundant and breeds in colonies distributed across the interior of Canada and the US. Over most of its range, this cormorant migrates north to breed in summer and south in winter. The continental population is between 1 and 2 million birds, including about 350,000 breeding pairs (Dorr et al. 2014). It is likely that the current population is similar in size to the population before European settlement of North America, but the species now is widely perceived to be overabundant (Wires 2014).

In 1990, an epidemic of Newcastle disease (ND), defined as a disease in any bird species caused by infection with strains of avian paramyxovirus-1 (APMV-1), which are highly pathogenic in chickens, was discovered in cormorants in western Canada, and at additional locations in Canada and the US in subsequent years (Wobeser et al. 1993; Kuiken 1999). The disease was not, and is not, present in commercial poultry in Canada or the US, and its discovery in cormorants posed an apparent new risk to the continental agricultural economy (Cattoli et al. 2011).

In 1994, research on ND and other causes of mortality in cormorants was initiated on Island A, a provincial wildlife sanctuary on Doré Lake (Saskatchewan, Canada), which is the site of a large nesting colony. Intensive study for 3 yr (Kuiken et al. 1998b) was followed by 18 additional years of monitoring the occurrence and causes of diseases producing large-scale mortality (epidemics) in cormorants during the breeding season; during that period, epidemic avian cholera (AC; infection with the bacterium *Pasteurella multocida*) was also discovered.

This article reports the results of these 21 yr of observation. In addition to the occurrence of epidemic diseases, data were collected on the pathogens causing epidemics, the range of species and age classes affected, the presence in freshly laid eggs of antibodies against APMV-1, and the total number of breeding pairs of cormorants. The potential impacts of the observed epidemics on the breeding population of cormorants on Island A were estimated. A model of APMV-1 transmission dynamics on Island A was constructed and was used to identify the parameters potentially most important in determining the occurrence and scale of epidemics and to examine whether changes over time in the proportion of nests in trees versus on the ground might influence disease occurrence, as was observed by Cleary (1977) and proposed by Kuiken et al. (1998b).

MATERIALS AND METHODS

Study site

Island A is a small island (approximately 100×300 m) located in Doré Lake, Saskatchewan, Canada (54°46′N, 107°17′W). Colony-nesting waterbirds, particularly cormorants and American White Pelicans (*Pelecanus erythrorhynchos*), began nesting there in 1983 when the island was forested (Hanbidge 1989). Large-scale mortality of cormorants was reported for the first time on Island A by provincial conservation officers in 1992, at which time all trees on the island were dead. During the 21 yr of this study, in addition to cormorants, approximately 1,500 nesting pairs of pelicans, fewer than 10 pairs of Herring Gulls (*Larus argentatus*), one pair of Bald Eagles (*Haliaeetus leucocephalus*), but no other bird species, were found nesting on Island A in some years.

Detection of epidemics

During 1994–96, the colony was monitored intensively (Kuiken 2016; Kuiken et al. 1998b). From 1997 to 2014, the colony was visited by two to four people one to three times each summer in late July and early August, the period during which hatchling cormorants have left their nests and are most vulnerable to bird-to-bird transmission of contagious pathogens (Kuiken et al. 1998b). At each visit, Island A was searched systematically and completely on foot for sick or dead birds. Dead birds were identified to species based on field characteristics; were categorized as nestling (down-covered and present in a nest, approximately 0–4 wk old), juvenile (postnestling subadult that has left the nest but is fed by parents off the nest or has just achieved flight and independence [fledged], approximately 4–8 wk old), or adult, based on location, size, and feathering; and were counted. When dead birds were discovered, necropsies to determine all potential causes of death were conducted on up to 10 freshly dead cormorants either on Island A or at the Western College of Veterinary Medicine in Saskatoon. When found, specimens of other species were also necropsied. Basic sanitary procedures were used to prevent unintended transport of pathogens from Island A to other locations.

Detection of pathogens

In all cases, grossly evident and microscopic lesions were assessed by qualified veterinary pathologists. Samples of liver, spleen, kidney, heart, and brain were frozen and subsequently used for detection of APMV-1 by PCR or virus isolation (Wobeser et al. 1993; Kuiken et al. 1998b, Wise et al. 2004). Immunohistochemistry was also used to identify APMV-1 in formalin-fixed, histologic sections (Kuiken et al. 1999b). If a specimen was found to be infected with APMV-1, samples were sent to the National Centre for

TABLE 1. Freshly voided excrement of Double-crested Cormorants (*Phalacrocorax auritus*) analyzed for the presence of avian paramyxovirus-1 by PCR on Island A (Doré Lake, Saskatchewan, Canada).

Year	Collection date	No. samples	No. PCR positive	July–August epidemic
2004	2 June	300	0	None
2006	22 May	300	0	Avian cholera
2008	25 August	98	3[a]	Newcastle disease
2009	25 May	500	1[b]	Avian cholera
2010	2 June	550	0	Avian cholera

[a] Cycle threshold (Ct) values of 39, 40, and 41. One cloacal sample from a live, moribund bird also was positive (Ct of 34).
[b] Ct value of 37.

Foreign Animal Diseases, Canadian Food Inspection Agency, to identify the virus and to assess its pathogenicity to chickens by gene sequencing or by experimental infections (Clavijo et al. 1998).

Samples of liver and spleen were cultured for pathogenic bacteria at the veterinary diagnostic laboratory of the Western College of Veterinary Medicine. Samples were inoculated on blood and MacConkey agar plates (Oxoid Canada, Nepean, ON, Canada), and isolates were identified by standard laboratory procedures (Quinn et al. 2011).

Detection of antibodies to APMV-1

The concentration of antibodies in the yolk of freshly laid eggs was used to assess the level of humoral immunity to APMV-1 in breeding female cormorants returning to the colony site in spring. From 1994 to 2003, 100 freshly laid eggs were collected randomly (one freshly laid egg from the nest nearest a randomly selected intersection point on a 1-m grid superimposed on a map of the island) across the colony in late May each year. Antibodies to AMPV-1 were measured in 1:10 or 1:5 dilutions of the yolk in phosphate-buffered saline by hemagglutination inhibition (Kuiken et al. 1998b).

Detection of APMV-1 in excrement

In 4 different yr, freshly voided excrement from cormorants was collected on Island A in late May or early June to determine whether adult cormorants returning to the colony site were shedding AMPV-1, and in August 2008, samples of freshly voided excrement from cormorants of unknown age and one sample from the cloaca of a moribund juvenile cormorant were collected during an epidemic of ND (Table 1). Samples were held on ice for 1–3 d and then frozen at –80 C until analyzed. The PCR for AMPV-1 was performed at the Department of Viroscience, Erasmus University Medical Centre (Rotterdam, the Netherlands) by published methods (Wise et al. 2004).

Estimate of breeding population

The total number of active cormorant nests on Island A was counted each year from 1996 to 2014 using photographs taken directly above the colony by low-flying aircraft (Fig. 1). Photographs were taken in late May or early June to coincide with the peak of incubation activity. Each active nest thus counted was assumed to represent a breeding pair of cormorants (Somers et al. 2010).

Impact of diseases on the population

Stage-specific mortality rates were used to estimate the capacity of the Island A breeding colony to sustain its breeding population of cormorants under various magnitudes of additive mortality from epidemic diseases, based on

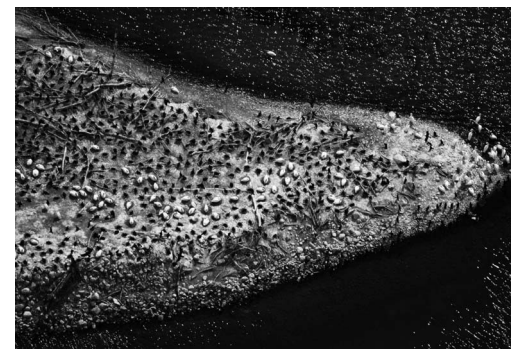

FIGURE 1. Aerial photograph of a portion of Island A (Doré Lake, Saskatchewan, Canada) taken on 18 May 2010 showing Double-crested Cormorants (*Phalacrocorax auritus*) on their nests during the incubation period. The number of breeding pairs on the colony was estimated each year by counting the black incubating birds in such photographs. The even spacing of nests in the colony facilitated visual recognition. Larger white birds are American White Pelicans (*Pelecanus erythrorhynchos*); long linear objects are fallen dead trees. Photograph by D. Guedo.

TABLE 2. Parameters used to estimate population effects of mortality from epidemic diseases in Double-crested Cormorants (*Phalacrocorax auritus*) on Island A, Doré Lake, Saskatchewan, Canada.

Parameter	Value	ID
Hatchlings fledged per nest	1.4	A
No. of breeding birds (nests×2)	13,698	B
Year 1 annual mortality rate	0.52	C
Year 2 annual mortality rate	0.26	D
Adult annual mortality rate	0.15	E

published annual mortality data (Dorr et al. 2014) and on data of reproductive success measured on Island A from 1994 to 1996 (Kuiken et al. 1999a). These calculations assumed that immigration and emigration of breeding cormorants on Island A did not occur and that mortality of juveniles from epidemics of ND and AC occurred in addition to published annual mortality rates for cormorants in their first year. Parameter values and calculations for these estimates are in Tables 2 and 3. The average number of cormorants fledged per nest (breeding pair), 1.4, is the rounded mean estimate for this colony in 1994–96 (1.38; Kuiken et al. 1999a). The average number of breeding pairs on Island A is from this study. Annual survival rates for different age classes of cormorants are from Dorr et al. (2014).

Outbreak dynamics during the nesting season

Outbreak dynamics for ND in juvenile cormorants were modeled using a compartmental epidemiologic model with four classes: susceptible individuals (S), which have not yet been exposed to APMV-1; exposed individuals (E), which have been infected and are incubating the virus but are not yet infectious; infectious individuals (I), which are actively shedding the virus; and individuals that have recovered (R) from infection and are no longer infectious. We assumed that recovered individuals acquire complete immunity and cannot become reinfected during the nesting season. We used the following first-order differential equations to represent the infection dynamics of ND in juvenile cormorants:

$$S = \frac{dS}{dt} = -\beta \times (S \times I) \quad (1)$$

$$E = \frac{dE}{dt} = \beta \times (S \times I) - E \quad (2)$$

$$I = \frac{dI}{dt} = E \times k - I \quad (3)$$

$$R = \frac{dR}{dt} = I \times \alpha \quad (4)$$

Model parameters: Model parameter values and initial conditions used for simulation are listed in Table 4. The rate at which exposed juvenile cormorants become infectious (k) is the

TABLE 3. Derived values used to estimate population effects of mortality from epidemic diseases in Double-crested Cormorants (*Phalacrocorax auritus*) on Island A (Doré Lake, Saskatchewan, Canada).[a]

Estimate	Value	Calculation	% Required annual recruitment
Annual			
Annual adult mortality (number of breeding birds to be replaced)	2,055	B×E	
Number of fledglings surviving to age 2 yr (first breeding)			
With no additional mortality from disease, ID F	2,593	A×B×C×D	126
With 40% additional fledgling mortality from disease	1,556	(A×B×0.6)C×D	76
With 50% additional fledgling mortality from disease, ID G	1,296	(A×B×0.5)C×D	63
With 60% additional fledgling mortality from disease	1,037	(A×B×0.4)C×D	50
Five years			
Annual adult mortality (birds)	10,274	5(B×E)	
Number of fledglings surviving to age 2 yr (first breeding)			
With no additional mortality from disease	12,964	5(A×B×C×D)	126
With 50% additional fledgling mortality from disease in three of 5 yr	9,852	(2×F)+(3×G)	88
With 50% additional fledgling mortality from disease in five of 5 yr	7,778	5×G	63

[a] ID abbreviations and those used in calculations relate those given in Table 2.

TABLE 4. Parameters used to model the transmission dynamics of avian paramyxovirus-1 in this study of Double-crested Cormorants (*Phalacrocorax auritus*) on Island A (Doré Lake, Saskatchewan, Canada).

Parameter	Definition	Value	References
β	Disease transmission rate	1.33×10^{-4}	
k	Rate at which exposed cormorants become infectious (1/incubation period)	1/3.27	Kuiken et al. 1998a
α	Recovery rate (1/shedding period)	1/13.9	Kuiken et al. 1998a
m	Disease-induced mortality rate (1/survival time of infectious birds)	1/3	Saif and Fadley 2008

inverse of the virus incubation period, which ranged from 2 to 4 d (mean=3.27 d) in experimental infection trials (Kuiken et al. 1998a). Recovery rate (α) is the inverse of the number of days cormorants remain infectious (shedding period), which was calculated as the mean shedding period for experimentally infected juvenile cormorants (Kuiken et al. 1998a). Disease-induced mortality rate (m) for infected birds is the inverse of survival time, which is short in juvenile cormorants infected with APMV-1 but for which no precise data are available. Postinfection survival time in infected young chickens is often 3 d or less (Saif and Fadly 2008); 3 d was used here as the initial parameter value. For simplicity, we assumed that mortality from sources other than disease during the juvenile period between leaving the nest and fledging was constant and negligible, as reported by Kuiken et al. (1999a). Finally, in the absence of empirical estimates of transmission rate of APMV-1 between infected and susceptible juvenile cormorants, we estimated the transmission rate (β) by fixing all other parameters to their literature-derived values and then calculating the value of β required to produce the estimated 50% average juvenile mortality by the end of the nesting season observed in this study (see Results).

Initial conditions: The total number of juvenile cormorants (N) was calculated as the average total number of nests counted from 1996 to 2014 (6,849) multiplied by the mean number of young produced per nest observed on Island A from 1994 to 1996 (1.38), yielding 9,452 juvenile birds. We assumed that all nestling cormorants were susceptible to infection at hatching but that only some individuals acquired infection from their parents before leaving the nest. The initial number of infected juveniles was estimated based on our detection of virus in only one of 1,650 fresh fecal samples collected during the period of egg incubation, an apparent prevalence of infection in incubating breeding adults of 0.00061. The total number of infected returning adults was estimated as that apparent prevalence multiplied by the total number of adult breeding birds (13,698, twice the average nest count), yielding 8.4 infected returning adults. Assuming that each infected adult is in a different nest and that all offspring in that nest are infected when they leave the nest, the number of infected juveniles initially in the colony is 11.6 (1.38 per nest×8.4 infected adults). The initial number of juvenile cormorants in the exposed and recovered classes was set at zero.

Sensitivity analysis: In this model, we were most interested in one outcome: the total mortality of juvenile cormorants during the 30-d prefledging period. The sensitivity of the model to variation in model parameters was estimated by carrying out a global sensitivity analysis using the Latin hyper cube (LHC) method with partial rank-correlation coefficient index (Blower and Dowlattabadi 1994; Marino et al. 2008). This method tests multiple combinations of model parameters drawn from a distribution to evaluate the relative influence of each parameter on model outcomes of interest. The LHC consisted of 1,000 simulations of different parameter combinations, with all model parameters varied by $\pm20\%$ and parameter values sampled from a uniform distribution within this range. We also varied the number of infected juveniles at the start of the simulation by $\pm20\%$ within the LHC framework to investigate the sensitivity of model outcomes to that initial condition.

Nesting behaviour scenarios: To determine the effect of the timing of juveniles leaving the nest on ND outbreak dynamics, we used the model to compare two nesting scenarios: (1) nestling cormorants leaving the nest at 6 wk old (corresponding to tree-nesting behavior), and (2) nestlings leaving the nest at 4 wk old (corresponding to ground-nesting behavior; Lewis 1929).

All analyses were carried out with packages *deSolve* and *lhs* in R, version 3.0.2 (R Core Team 2017).

TABLE 5. Occurrence of epidemic diseases among Double-crested Cormorants (*Phalacrocorax auritus*) on Island A (Doré Lake, Saskatchewan, Canada) from 1994 to 2014. Mortality estimates for 1998–2012 are actual carcass counts made during single visits to the island and are minimum estimates. Nests with incubating adults were counted from aerial photographs. Antibodies to avian paramyxovirus-1 (APMV-1) were measured in yolks of freshly laid eggs.

Year	Epidemic occurrence	Disease	Total mortality estimate	Active nest count	% APMV-1 antibody prevalence
1994	No	—	—	No data	54[b]
1995	Yes	Newcastle disease	32–64%[b]	No data	51[b]
1996	No	—	—	7,070	66[b]
1997	Yes	Newcastle disease	No estimate	7,874	71
1998	Yes	Avian cholera	>1,523	11,094	97
1999	Yes	Newcastle disease	>831	4,171	100
2000	Yes	Avian cholera	No estimate	9,585	92
2001	No	(Newcastle disease)[a]	—	6,520	100
2002	Yes	Avian cholera	>1,081	5,766	95
2003	Yes	Newcastle disease	>500	8,422	43
2004	No	—	—	7,378	—[c]
2005	Yes	Avian cholera	>2,350	4,740	—
2006	Yes	Avian cholera	>3,000	8,129	—
2007	Yes	Avian cholera	>1,039	7,165	—
2008	Yes	Newcastle disease	>500	7,909	—
2009	Yes	Avian cholera	>1,070	6,911	—
2010	Yes	Avian cholera	>1,308	6,960	—
2011	No	—	—	7,561	—
2012	Yes	Newcastle disease	>274	6,654	—
2013	Yes	Avian cholera	No estimate	2,912	—
2014	Yes	Avian cholera	No estimate	3,305	—

[a] Avian paramyxovirus-1 was detected in two Double-crested Cormorant nestlings found moribund together on one nest, but no epidemic was detected.

[b] Data from Kuiken et al. 1998b.

[c] This component of the study was discontinued after 2003.

RESULTS

Disease epidemics

Outbreaks of two diseases, ND and AC, resulted in large-scale mortality on Island A in 16 of the 21 nesting seasons monitored in this study (Table 5). No other causes of epidemic disease were detected during this study by the diagnostic methods employed: necropsy, virus culture, PCR for APMV-1, histopathology, and screening cultures for bacterial pathogens. In four of the five seasons without outbreaks, neither pathogen was detected in the few birds found sick or dead on Island A; in 2001, infection with APMV-1 was detected in two moribund nestlings found in one nest, but large-scale mortality from that or other causes did not occur during the 2001 nesting season.

Nearly all cormorants dying in these epidemics were 6–8 wk old, fully feathered, and approaching adult size and independence. Adult cormorants were never found among the sick or dead in outbreaks of ND, and they were rare but present among dead birds observed during AC epidemics. Although ND affected only the cormorants, AC also affected a few of the other bird species (Table 6).

In the intensively studied epidemic of ND in 1995, the apparent mortality rate among juvenile cormorants was 32% (Kuiken et al. 1998b). This was considered a minimum estimate because detection of dead birds on

TABLE 6. Species of birds found dead in small numbers and to be infected with *Pasteurella multocida* during epidemics of avian cholera in Double-crested Cormorants on Island A (Doré Lake, Saskatchewan, Canada) in 1994–2014.

Common name	Scientific name
American White Pelican	*Pelecanus erythrorhynchos*
Canada Goose	*Branta canadensis*
Western Sandpiper	*Calidris mauri*
Semi-palmated Sandpiper	*Calidris pusilla*
Least Sandpiper	*Calidris minutilla*
Herring Gull	*Larus argentatus*

the colony site was less than 100% and because cormorants dead from ND were also found on a large adjacent island without nests. In assessments of subsequent epidemics, mortality data were limited to counts of dead cormorants made during single, brief visits to the colony site during the epidemics; thus, all are minimum mortality counts (Table 5). The largest number of dead birds counted during an epidemic was 3,000, counted at what was likely the peak of mortality in 2006. That is 27% of the 11,218 juveniles estimated to be in the Island A population for that year and is close to the minimum mortality estimate of 32% of juvenile birds on the colony site made in 1995. As reasoned by Kuiken et al. (1998b), because some affected birds die off the colony site, carcasses can quickly be eliminated from the site by scavenging and by wave action at the shoreline, and counts of dead birds on the colony site are imperfect, we estimated that ND and AC killed approximately 50% of juvenile birds in each of the 16 yr when epidemics occurred.

Breeding population

From 1996 to 2014, the average number of cormorant nests counted from aerial photographs was 6,849 (range, 2,912–11,094; Table 5). Thus, on average, 13,698 breeding cormorants nested on the colony each year. There was no evident relationship between nest counts and occurrence of epidemic diseases (Table 5).

Antibodies to APMV

From 1994 to 2003, the percentage of antibody prevalence to APMV-1 in the yolk of freshly laid cormorant eggs varied from 43% to 100% (Table 5). Yolks with antibody titers of 1:20 or greater were considered positive (Kuiken et al. 1998b). There was no evident relationship among antibody prevalence, breeding population size, or occurrence of ND. Epidemics of ND occurred in cormorants hatched in years with the lowest and with the highest recorded antibody prevalence in the eggs sampled (Table 5).

Detection of AMPV-1 in cormorant excrement

In the 1,650 fresh samples of excrement collected from adult cormorants in spring, RNA attributable to APMV-1 was found by PCR in only one sample, with a cycle threshold value of 37 (Table 1). We detected RNA attributable to APMV-1 in three of 98 freshly voided samples collected from full-grown cormorants of unknown age in August during an epidemic of ND and in a sample collected from the cloaca of one juvenile cormorant, moribund from ND, during the same epidemic.

Impact of diseases on the population

The annual adult mortality rate of 0.15 (Dorr et al. 2014) implies that 15% of the breeding bird population of Island A must be replaced each year if the breeding population is to be maintained over time. Cormorants breed for the first time at 2 yr old (Dorr et al. 2014). In years with no additional mortality of juveniles from disease, the number of juveniles fledged per nest, combined with the expected annual mortality of 52% and 26% of these birds in their first and second years, respectively (Dorr et al. 2014), yields a surplus of available new birds able to begin breeding in year three: 126% of the number required for replacement. In years with additional mortality among juveniles of 40, 50, or 60% (the range considered in this study), only 76, 63, and 50%, respectively, of the number of new breeders required to maintain the

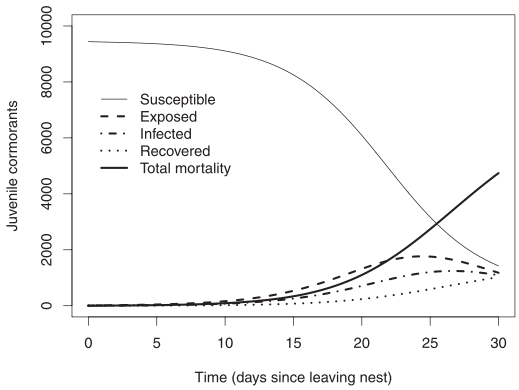

FIGURE 2. Model output showing change in infection status of juvenile cormorants over the course of the breeding season during an outbreak of Newcastle disease (ND). Outbreak dynamics were modeled using a compartmental epidemiologic model with four classes: susceptible individuals, which have not yet been exposed to the ND virus; exposed individuals, which have been infected and are incubating the virus but are not yet infectious; infectious individuals, which are actively shedding the virus; and recovered individuals, which are no longer infectious.

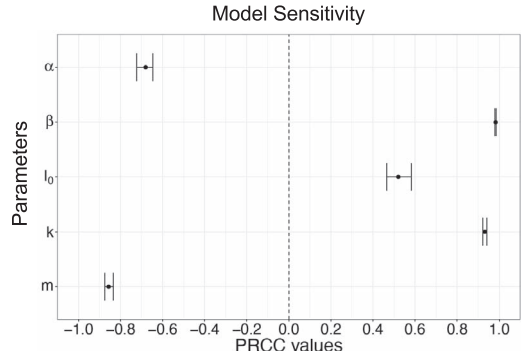

FIGURE 3. Sensitivity of the model of Newcastle disease dynamics in juvenile cormorants to variation in: recovery rate (α), transmission rate (β), the number of infected juvenile cormorants at the start of the simulation (I_0), the rate at which exposed juvenile cormorants become infectious (k), and disease-induced mortality rate (m). Model sensitivity was estimated by varying each parameter by ±20% using the Latin hyper cube method and partial rank-correlation coefficients (PRCC). The PRCC values vary between −1 and 1 and measure how strongly variation in input parameters influenced total mortality of juvenile cormorants 30 days after leaving the nest. Error bars show 95% confidence intervals from 1,000 bootstrap replicates.

average breeding bird population size would be available 2 yr later.

As shown in Table 5, during the 21 yr of this study, 5-yr periods with the fewest epidemic years consisted of three epidemic years and two disease-free years. The most-affected 5-yr periods consisted of 5 consecutive yr of epidemic disease. As shown in Tables 2 and 3, the overall estimated effect on the breeding bird population of Island A from 50% additional mortality of juveniles caused by disease occurring in three or in five breeding seasons during a 5-yr period is production of 88% or of 63%, respectively, of the required replacement breeders.

Outbreak dynamics during the nesting season

Infection dynamics: The transmission model developed for APMV-1 suggested that virus transmission and resulting mortality are limited during the first 10 d after juvenile cormorants leave the nest but increase rapidly thereafter until juveniles fledge, progressively gain independence, and begin to disperse from the colony site (Fig. 2). Infection dynamics were most sensitive to changes in transmission rate (β), disease-induced mortality rate (m), and the rate at which exposed juvenile cormorants become infectious (k; Fig. 3). Increases in transmission rate (β) and in the rate at which exposed birds become infectious (k) caused an increase in total mortality of juveniles at the end of the nesting season, whereas more rapid mortality of infected birds (m) slowed down transmission, resulting in lower total mortality. Infection dynamics were moderately sensitive to the rate at which infected individuals recovered from infection (α), with more rapid recovery slowing down transmission and reducing total mortality. An increase in the number of infected juveniles at the time of their departure from the nest (I_0) accelerated transmission, resulting in greater total mortality; however, infection dynamics were least sensitive to variation in that parameter.

Nesting behavior scenarios: Simulations of ground-nesting vs. tree-nesting scenarios suggested that the earlier departure from the nest observed in ground-nesting cormorants has a

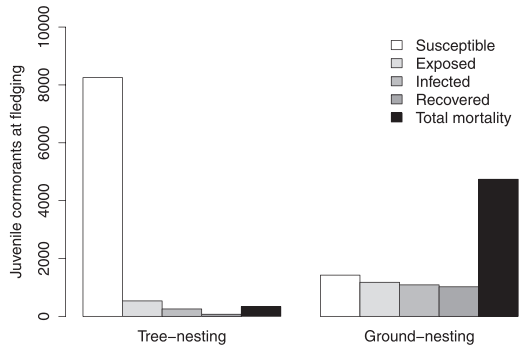

FIGURE 4. Model-estimated impact of the loss of tree-nesting habitat on Newcastle disease dynamics in juvenile cormorants. When trees are present, adults preferentially nest in trees, and juveniles leave the nest at 6 wk of age (tree-nesting scenario). In the absence of trees, adults nest on the ground, allowing juveniles to leave the nest and transmit infection 2 wk earlier at 4 wk of age (ground-nesting scenario). The bars show the estimated number of juvenile cormorants in each infection class, as well as total mortality, at the end of the nesting season.

significant effect on the occurrence of major disease outbreaks (Fig. 4). Juveniles from nests on the ground that left the nest at 4 wk old suffered 14 times greater disease-induced mortality by the end of the nesting season (50.32% mortality) than did nestlings in tree-nests that left the nest 2 wk later (3.56% mortality).

DISCUSSION

In 16 of 21 consecutive breeding seasons, the cormorants using Island A as a breeding site experienced approximately 50% mortality of hatch-year offspring because of epidemic infectious diseases during their juvenile period. From these data, it was possible to estimate the substantial potential impact of disease-associated mortality on the colony population (Tables 2, 3) and to document that such negative effects could not be detected in the actual population data. The rapid growth of the cormorant population from the 1970s to the present (Dorr et al. 2014; Wires 2014) implies that mortality factors, including diseases, have had no important impact on the population of the species on a continental scale. Immigration of breeding birds probably compensated completely for the loss of replacement breeders because of epidemics on Island A.

A plausible mathematical model of contagious pathogen transmission dynamics in this colony indicated that juveniles from nests on the ground were 14 times more likely to die of infectious disease than were juveniles from nests in trees, the critical difference being the 4-wk (ground nests) versus 2-wk (tree nests) period during which pathogen transmission could occur (Kuiken et al. 1998b).

Island A was first colonized by cormorants in 1983 when it was a forested island. Initially, cormorants nested primarily in trees but also on the ground (Hanbidge 1989). Cormorant excrement is toxic to vascular plants (Ishida 1997), and all trees were dead by 1992. As trees died and fell over, an ever-larger proportion of the cormorants had to nest on the ground; by 1994, nearly all nests were on the ground. In his seminal study of the Double-crested Cormorant, Lewis (1929) noted that nestlings from nests on the ground left the nest and began to live in groups of postnestling juvenile birds at about 4 wk old and 2 wk earlier and younger than nestlings from nests built in trees. Kuiken et al. (1998b) noted that nestlings left ground nests at 4 wk old and that they began dispersing from Island A starting at about 8 wk old. Kuiken et al. (1998b) also noted that contact among nestlings from different nests did not occur until nestlings left the nest, but, thereafter, physical interactions among postnestling juveniles conducive to transmission of contagious pathogens was intensive. Juveniles from nests on the ground were, thus, at risk of pathogen transmission among postnestling juveniles for 2 wk longer than were juveniles from tree nests.

As trees died and an increasing proportion of cormorants on Island A came to nest on the ground, the average duration of the period of potential pathogen transmission among juvenile cormorants would have increased from approximately 2 wk toward 4 wk, simultaneously increasing the probability that a contagious pathogen present on the colony would cause a large-scale epidemic.

This revenge-of-the-trees hypothesis, in which deforestation by cormorants results in epidemics, may apply principally along the northern tier of the breeding range of the Double-crested Cormorant, where trees for nesting are often abundant at new colony sites, and where breeding phenology is highly synchronous and tightly compressed into the short boreal summer. Our model is highly supportive of this hypothesis in this setting. The duration of the contact period among juveniles had an enormous effect on total mortality (Fig. 4).

Our model of transmission dynamics was developed for AMPV-1 and not for *Pasteurella multocida*. Like the virulent strains of AMPV-1 affecting the Island A colony, virulent *Pasteurella multocida* is highly contagious and rapidly fatal (Samuel et al. 2005) and affected juvenile cormorants almost exclusively on Island A. Although several parameters required for our model were not available for *P. multocida*, it is probable that the transmission dynamics of this bacterium in cormorants on Island A was similar to that of APMV-1.

It is not known how the pathogenic strains of APMV-1 and *Pasteurella multocida* arrived on the Island A colony in epidemic years. Both pathogens are known to infect adult birds asymptomatically in other species and APMV-1 has produced asymptomatic infections in cormorants (Kuiken et al. 1998a; Samuel et al. 2005). Shedding of these pathogens by asymptomatic carrier birds seems the most plausible source among cormorants on Island A. In this study, APMV-1 was detected in only one of 1,650 samples of fresh excrement collected from adult birds between late May and early June. These data imply that shedding of this virus is not common, but can occur, among adult cormorants during the period of egg incubation. According to our model, as few as eight to nine infectious adults in a breeding population of 13,700 birds is sufficient to initiate an epidemic.

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