MULTI-SPECIES PATTERNS OF AVIAN CHOLERA MORTALITY IN NEBRASKA'S RAINWATER BASIN

Authors: Blanchong, Julie A., Samuel, Michael D., and Mack, Gene

Source: Journal of Wildlife Diseases, 42(1) : 81-91

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-42.1.81

Journal of Wildlife Diseases, 42(1), 2006, pp. 81–91

MULTI-SPECIES PATTERNS OF AVIAN CHOLERA MORTALITY IN NEBRASKA'S RAINWATER BASIN

Julie A. Blanchong,[1,5] Michael D. Samuel,[2,3] and Gene Mack[4]

[1] Department of Wildlife Ecology, 1630 Linden Drive, University of Wisconsin, Madison, Wisconsin 53706, USA
[2] US Geological Survey, National Wildlife Health Center, 6006 Schroeder Road, Madison, Wisconsin 53711, USA
[3] Current address: US Geological Survey, Wisconsin Cooperative Wildlife Research Unit, 204 Russell Labs, University of Wisconsin, Madison, Wisconsin 53706 USA
[4] US Fish and Wildlife Service, Rainwater Basin Wetlands Management District, PO Box 1686, Kearney, Nebraska 68847, USA
[5] Corresponding author (email:jablanchong@wisc.edu)

ABSTRACT: Nebraska's Rainwater Basin (RWB) is a key spring migration area for millions of waterfowl and other avian species. Avian cholera has been endemic in the RWB since the 1970s and in some years tens of thousands of waterfowl have died from the disease. We evaluated patterns of avian cholera mortality in waterfowl species using the RWB during the last quarter of the 20th century. Mortality patterns changed between the years before (1976–1988) and coincident with (1989–1999) the dramatic increases in lesser snow goose abundance and mortality. Lesser snow geese (*Chen caerulescens caerulescens*) have commonly been associated with mortality events in the RWB and are known to carry virulent strains of *Pasteurella multocida*, the agent causing avian cholera. Lesser snow geese appeared to be the species most affected by avian cholera during 1989–1999; however, mortality in several other waterfowl species was positively correlated with lesser snow goose mortality. Coincident with increased lesser snow goose mortality, spring avian cholera outbreaks were detected earlier and ended earlier compared to 1976–1988. Dense concentrations of lesser snow geese may facilitate intraspecific disease transmission through bird-to-bird contact and wetland contamination. Rates of interspecific avian cholera transmission within the waterfowl community, however, are difficult to determine.

Key words: Avian cholera, *Chen caerulescens caerulescens*, epizootiology, lesser snow geese, Nebraska, *Pasteurella multocida*, Rainwater Basin, waterfowl.

INTRODUCTION

Of the diseases affecting waterfowl in North America, avian cholera, caused by the bacterium *Pasteurella multocida*, is one of the most important infectious diseases affecting wild geese (Friend, 1987; Wobeser, 1997). In addition, this highly contagious disease has the potential to substantially impact other avian species. Transmission of *P. multocida* among waterfowl likely occurs by direct bird-to-bird contact and by either ingestion from contaminated wetlands or by inhalation of contaminated water droplets in aerosols when birds take flight (Botzler, 1991; Wobeser, 1992). Thus, wetlands with high densities of gregarious waterfowl, such as lesser snow geese (*Chen caerulescens caerulescens*), may be at increased risk for disease transmission and severe disease outbreaks. Because the bacterium affects >100 species of waterbirds (Botzler, 1991) factors such as bird density, disease transmission, and species mortality rates may depend on the community of waterfowl hosts using an area.

Although there is considerable uncertainty about the ecology of avian cholera, recent evidence indicates that lesser snow geese, Ross's geese (*Anser rossii*), and possibly other waterbirds, are likely carriers of *P. multocida* and serve as reservoirs for this disease (Samuel et al., 1999a, 2005). In addition, factors such as severe weather, crowding, stress, and other conditions can increase the risk or severity of disease outbreaks (Smith et al., 1990; Botzler, 1991; Windingstad et al., 1998; Samuel et al., 1999b). Mortality rates during disease outbreaks have been difficult to document, but estimates indicate 5–10% of an entire nesting lesser snow goose population may succumb during breeding ground outbreaks, with higher mortality rates occurring in nesting areas with higher bird density (Samuel et al., 1999c). Although avian cholera has killed >100,000 birds during a single outbreak (National Wildlife Health Center [NWHC], unpubl. data) the disease also appears to be transmitted year-round (Samuel et al., 2005) and causes ongoing, low-level mortality within waterfowl populations (Botzler, 1991; Wobeser, 1992; Samuel et al. 1999c).

The Rainwater Basin (RWB) in central Nebraska is a key focal point in the spring migration of millions of ducks, geese, shorebirds, and cranes. These birds stop for an extended period to feed, rest, initiate pairing activities, and acquire nutrient reserves critical for the northern migration and subsequent reproductive success. Large-scale habitat changes in the RWB, however, have produced at least two notable effects on migratory waterfowl. Roughly 90% of the original waterfowl habitat in the RWB has been destroyed (Tiner, 1984). As a result, birds have become more concentrated on remaining basin wetlands that are maintained by natural rainwater or by pumping ground water. This dramatic reduction and shift in habitat has produced extremely high concentrations of birds (>500,000–1,000,000) roosting on many of the remaining wetlands. These crowded conditions can enhance the risk of transmission and spread of infectious diseases (Friend, 1992). Since the 1970s, avian cholera has been a recurrent disease problem in the RWB (Windingstad et al., 1984, 1988), where mortality occurs almost annually, and in some years (e.g., 1998) estimated losses have exceeded 100,000 birds. Risk and severity of diseases like avian cholera may have been further exacerbated by concurrent reductions in wetland quality (Friend, 1981) or increases in eutrophic wetland nutrients (Blanchong et al., 2006). Coincident with habitat changes in the RWB, the midcontinent population of lesser snow geese has grown exponentially (5% annually) in the past 20–25 years (Abraham and Jefferies, 1997). Starting in the late 1980s, the skyrocketing population of midcontinent lesser snow geese also shifted its principal spring migration corridor from Iowa, Missouri, and eastern Nebraska to central Nebraska. These birds were apparently attracted to the abundance of nutrient and energy subsidies provided to waterfowl feeding on waste agricultural crops in the RWB. The ability of lesser snow geese to exploit such food resources throughout the midcontinent has apparently played an important role in creating an overabundance of lesser snow geese in this population (Abraham and Jefferies, 1997).

Although increased harvest has been initiated to reduce the midcontinent population of lesser snow geese, the continued high abundance of these geese may increase the risk of large-scale avian cholera outbreaks in the RWB during spring migration. The purpose of this paper is to review the history of avian cholera outbreaks in the RWB, to characterize the mortality patterns among waterfowl species using the RWB, and to evaluate the relationship between lesser snow goose mortality and mortality patterns in other species. We analyzed carcass collection records for lesser snow geese and other waterfowl species from avian cholera outbreaks over the last quarter of the 20th century (1976–1999). We compared levels of avian cholera mortality (based on carcass collection) in common waterfowl species using the RWB both before and during periods of increasing lesser snow goose abundance, and we evaluated the correlation between lesser snow goose mortality and mortality in these species. In addition, we evaluated species-specific associations between the timing of spring outbreaks before (1976–1988) and coincident with (1989–1999) increased lesser snow goose abundance and mortality.

MATERIALS AND METHODS

The RWB encompasses >10,000 km^2 in south-central Nebraska (Fig. 1). The basin is composed of depressional wetlands primarily formed by wind and fluvial processes and

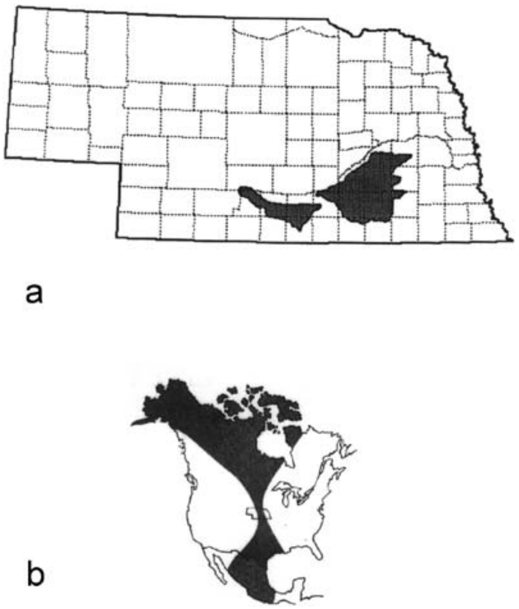

FIGURE 1. Location of Rainwater Basin in Central Nebraska (a) and generalized shape of Central Flyway spring waterfowl migration corridor (b).

dependent upon surface sources of water and/or irrigation–ground water connections. The hydrology of the entire RWB has been significantly altered by drainage pits, intensive watershed modification, water diversion, and intensive agricultural and irrigation practices (Farrar, 1982). This wetland system is also one of the most endangered in North America with ≤10% of the presettlement wetland basins remaining (Farrar, 1982; Smith and Higgins, 1990) within an intensive agricultural environment. The RWB is also recognized as the focal passageway for 7–9 million ducks and 5–7 million geese from three different flyways migrating from their wintering grounds in the southern United States and Mexico to their breeding grounds in prairie and arctic Canada (Gersib et al., 1992).

Avian cholera was first reported in the RWB in 1975 when an estimated 25,000 birds died (Zinkl et al., 1977). Since that time, avian cholera occurred almost annually from 1975–1999 with most losses occurring during spring migration, although fall outbreaks were occasionally reported. Estimated losses have varied each year with peak mortality reported in 1980 (Brand, 1984) and again in 1998 (Rainwater Basin Wetlands Management District, unpubl. data; NWHC, unpubl. data).

For each year of our study period from 1975 to 1999, when bird migration began, wetlands were checked by wetland biologists for dead birds. After dead birds began to be detected (2–5), a sample of fresh carcasses was sent to the NWHC to verify the cause of mortality (Friend, 1987). Each year, approximately 80% of the carcasses tested were diagnosed as avian cholera or suspect avian cholera mortality, indicating that avian cholera outbreaks were occurring and constituted the primary cause of epizootic mortality.

When the number of dead birds on any one wetland reached 20 carcasses, wetland staff systematically searched for and collected dead birds of all species. Other wetlands with high waterfowl use were also checked for dead birds. Bird carcasses in open water were easily detected and retrieved; however, the majority of dead birds were found along the vegetated shoreline and portions of the wetlands with surface water. The number of birds collected was recorded by sex (ducks only) and by species. All species of birds were searched for and collected; however, larger-bodied geese were typically easier to detect than smaller-bodied ducks. Carcasses were usually exposed in shallow water, but sank low in water that was more than 15 cm deep, reducing detection. The carcass search methods did not change substantially over the duration of our study period. Even in years when mortality was very low, biologists checked wetlands for dead birds to make certain that isolated incidents of morality were not overlooked.

We used data on carcasses collected by US Fish and Wildlife Service and Nebraska Game and Parks Commission staff during spring avian cholera mortality events in the RWB from 1976–1999 (1976, 1980–1992, 1995–1999) to evaluate species composition of mortality. The five species with the highest occurrence in carcass collection were lesser snow geese, white-fronted geese (*Anser albifrons*), Canada geese (*Branta canadensis*), mallard ducks (*Anas platyrhynchos*), and northern pintail ducks (*Anas acuta*). Carcasses collected for remaining waterfowl species were pooled and considered collectively as "other waterfowl" mortality.

We used linear regression to evaluate trends in the number of carcasses collected annually during spring outbreaks for each species and to test for an association with an index of species abundance, except "other waterfowl," for which there was no abundance estimate. Unfortunately, annual estimates of local abundance at Rainwater Basin wetlands do not exist for any of the species. Instead, we used annual winter counts of midcontinent populations of lesser snow, white-fronted, and Canada geese and subsequent North American breeding populations for ducks (Wilkins and Cooch,

1999) as a general index representing annual changes in local population abundance. Because our index of abundance for each species was not at a local scale, proportional mortality rates could not be reliably calculated or compared among species. Instead, we evaluated the relationship between lesser snow goose mortality and mortality in other species by comparing numbers of dead birds among species. Specifically, we used linear regression to evaluate the relationship between the number of lesser snow goose carcasses and the number of carcasses of each of the other species collected during the same outbreaks. We used one-tailed t-tests to compare average yearly mortality for each species before (1976–1988) and coincident with (1989–1999) increased lesser snow goose mortality.

We used daily mortality records to construct cumulative annual mortality curves for each species. We compared cumulative mortality curves between the two time periods (1976–1988 and 1989–1999) to investigate the relationship between increasing lesser snow goose mortality and avian cholera mortality in other waterfowl species. We also used these annual cumulative mortality curves to investigate the relative timing of avian cholera outbreaks and to identify species associated with initiation of outbreaks. We defined the beginning of an outbreak to be a cumulative detection of at least 50 bird carcasses of a given species. The end of an outbreak was defined as the last date on which bird carcasses of any species were collected. We used one-tailed t-tests to compare the average date of onset (calculated as number of days since 1 February) and termination of spring avian cholera outbreaks between the time before and during dramatic increases in lesser snow goose mortality. We also used t-tests to compare the timing of outbreak detection in each species between the two times.

RESULTS

From 1976 to 1999, >85,000 carcasses of known species composition were collected during spring avian cholera outbreaks and the number of carcasses varied annually from <500 birds in 1984–1985 to >26,000 in 1998 (Table 1). The number of carcasses collected, although typically much smaller than estimated total mortality (Humberg et al., 1983; Stuzenbaker et al., 1983), generally reflected the severity of avian cholera losses. Lesser snow goose mortality increased dramatically over the last quarter of the 20th century in the RWB (Table 1). Despite high annual variation in the number of carcasses collected, we found an increase in lesser snow goose mortality, but no significant trend for any other species (Table 2). Over time, lesser snow goose carcasses accounted for an increasing proportion of the total avian cholera mortality experienced by waterfowl in the RWB (Fig. 2). Annual mortality data indicated that beginning in 1991, and continuing through 1999, lesser snow goose mortality exceeded mortality in all other species combined (Table 1). Before 1989, avian cholera mortality was distributed among northern pintails (26.3%), mallards (24.7%), and white-fronted geese (23.4%), followed in frequency by Canada geese (11.4%), other waterfowl species (9.4%), and finally lesser snow geese (4.9%). From 1989 to 1999, lesser snow geese comprised the majority of carcasses collected (74.9%), followed by northern pintails (8.8%), white-fronted geese (5.6%), mallards (3.9%), other waterfowl species (3.6%), and Canada geese (3.2%). Annual avian cholera mortality in lesser snow geese was considerably higher during 1989–1999 than during 1976–1988, despite high variation in mortality from year to year (Table 3). In contrast to the strong trend for increased lesser snow goose mortality observed during 1989–1999, annual mortality for each of the other waterfowl species decreased relative to mortality levels during 1976–1988, though differences were not significant for individual species (Table 3). Our inability to detect a temporal trend in avian cholera mortality in other waterfowl species may result from the high annual variation in mortality, or possibly because of a detection bias for larger, more visible lesser snow goose carcasses that resulted in underestimation of mortality for other species.

We found no evidence for a strong relationship between midcontinent population abundance and species-specific

TABLE 1. Number of waterfowl carcasses collected, outbreak onset date, and first species collected during spring avian cholera outbreaks in Nebraska's Rainwater Basin from 1976–1999.[a]

Year	LSGO	WFGO	CAGO	MALL	NOPI	OTHER	Total	Onset date	Onset species
1976	10	55	35	186	427	79	792	16 March	NOPI
1980	160	4251	1443	6385	5930	2055	20224	12 March	WFGO
1981	18	216	127	224	1027	43	1655	3 March	NOPI
1982	60	1918	1018	1204	852	432	5484	5 March	CAGO
1983	50	302	188	109	82	47	778	2 March	CAGO
1984	38	92	107	85	75	68	465	14 March	MALL
1985	69	82	127	59	67	56	460	12 March	CAGO
1986	266	173	123	42	170	79	853	7 March	NOPI
1987	544	271	155	47	124	240	1381	No data	No data
1988	440	635	551	95	218	110	2049	5 March	LSGO, WFGO, OTHER
1989	862	232	35	21	84	57	1291	9 March	LSGO
1990	274	147	116	37	55	67	696	1 March	LSGO
1991	2708	623	134	82	384	126	4057	27 February	LSGO
1992	3610	670	154	107	147	40	4728	3 February	LSGO
1995	1306	78	21	37	79	21	1542	27 February	LSGO
1996	5223	505	92	321	1013	454	7608	12 March	LSGO
1997	2771	257	104	60	92	143	3427	15 March	LSGO
1998	20290	239	962	1267	2575	866	26199	20 February	LSGO
1999	1153	81	14	69	60	83	1460	24 February	LSGO
Total	39852	10827	5506	10437	13461	5066	85149		

[a] LSGO = lesser snow goose; WFGO = white-fronted goose; CAGO = Canada goose; MALL = mallard; NOPI = northern pintail; OTHER = other waterfowl.

avian cholera mortality in either time period. During 1976–1988, mallards were the only species for which we found a significant positive relationship between the number of carcasses collected and the species' breeding abundance (Table 4). There were no significant relationships between abundance and the number of carcasses collected for any species during 1989–1999 (Table 4). These results, however, should be interpreted conservatively because midcontinent abundance is only a general index of local abundance.

Prior to dramatic increases in lesser snow goose mortality (1976–1988), there were no significant relationships between lesser snow goose mortality and mortality in any of the other species (Table 5). During 1989–1999, however, we found significant positive relationships between the number of lesser snow goose carcasses found annually in the RWB and the number of carcasses of Canada geese, mallards, northern pintails, and other waterfowl collected (Table 5). The relationship between the number of lesser

TABLE 2. Annual trend (slope) in avian cholera mortality in Nebraska's Rainwater Basin for several waterfowl species from 1976 to 1999.

Species	Correlation	Slope	t	df	P
Lesser snow goose	0.53	439.30	2.59	17	0.02
White-fronted goose	−0.37	−65.49	−1.66	17	0.12
Canada goose	−0.26	−18.45	−1.09	17	0.29
Mallard	−0.33	−85.41	−1.44	17	0.17
Northern pintail	−0.24	−61.00	−1.04	17	0.31
Other waterfowl	−0.18	−15.58	−0.77	17	0.45

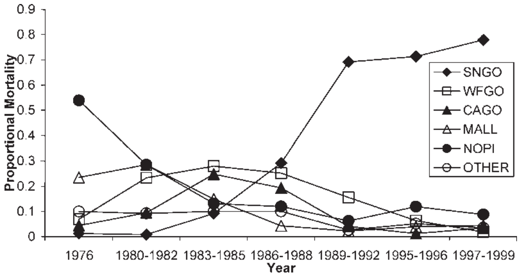

FIGURE 2. Proportion of total mortality contributed by lesser snow geese (SNGO), white-fronted geese (WFGO), Canada geese (CAGO), mallard ducks (MALL), northern pintail ducks (NOPI), and other waterfowl (OTHER) for years from 1976 to 1999.

snow goose and white-fronted goose carcasses collected was also positive, but not statistically significant. Residual statistics and regression influence diagnostics indicated that the high mortality experienced by all species in 1998 may have influenced our analyses. To assess the potential impact of 1998 data on our results, we conducted a second analysis after removing these data. Although correlation coefficients were reduced without the 1998 data, the relationships between lesser snow goose mortality and mortality in northern pintail, mallard, and other waterfowl species remained significant (Table 6). In addition, with removal of 1998 data, the relationship between lesser snow goose mortality and white-fronted goose mortality became significant (Table 6). The relationship between lesser snow goose and Canada goose carcasses remained positive, but was no longer significant (Table 6). Relative mortality (carcasses collected per 1,000 lesser snow goose carcasses) in other species of waterfowl during 1989–1999 were highest for northern pintail and white-fronted geese, and lowest for mallards. These are probably conservative estimates because detection bias likely favors greater collection of lesser snow goose carcasses relative to other smaller and less-visible species.

Coincident with the dramatic increase in lesser snow goose mortality was a significantly earlier annual detection of spring avian cholera outbreaks ($t_{11}=2.44$, $P=0.02$). Prior to the increase in lesser snow goose mortality (1976–1988), the average date on which spring cholera outbreaks (≥50 carcasses of one species) were first detected was 8 March and the first species to be collected during an outbreak varied annually (Table 1). During the latter time period (1989–1999), the average date on which spring outbreaks were first detected was 27 February and lesser snow geese were always among the first species collected (Table 1). The earlier average detection of avian cholera outbreaks from 1989–1999 coincided with an earlier end to outbreaks (as measured by the last date on which carcasses were collected; $t_{16}=1.91$, $P=0.04$). On average, during 1976–1988, the last date on which carcasses were collected was 7 April compared to 30 March during 1989–1999. When we compared the timing of outbreak detection (≥50 carcasses) for each species separately between 1976–1988 vs. 1989–1999, lesser snow geese

TABLE 3. Average number of carcasses collected for several waterfowl species from Nebraska's Rainwater Basin during 1976–1988 and 1989–1999.

Species	1976–1988	1989–1999	t	df	P
Lesser snow goose	165.5	4244.1	1.97	8	0.04
White-fronted goose	799.5	314.7	−1.13	10	0.14
Canada goose	387.4	181.3	−1.14	10	0.14
Mallard	843.6	222.3	−0.97	10	0.18
Northern pintail	897.2	498.8	−0.63	13	0.27
Other waterfowl	320.9	206.3	−0.53	13	0.30

TABLE 4. Relationship (slope) between species abundance and the number of carcasses collected from Nebraska's Rainwater Basin during 1976–1988 and 1989–1999.

Species	1976–1988 Correlation	t	df	P	1989–1999 Correlation	t	df	P
Lesser snow goose	0.3	0.83	7	0.43	0.55	1.734	7	0.13
White-fronted goose	−0.13	−0.37	8	0.72	0.03	0.074	7	0.94
Canada goose	0.43	1.07	5	0.33	0.24	0.663	7	0.53
Mallard	0.77	3.22	7	0.02	0.36	1.016	7	0.34
Northern pintail	0.6	1.83	6	0.12	0.04	−0.093	7	0.93
Other waterfowl	No data				No data			

were the only species for which we found a significantly earlier detection of avian cholera outbreaks in the latter time period (t_{12}=3.59, P=0.004). There was no trend in the timing of outbreak detection in any of the other species (all $P>0.05$). In addition, there was no relationship between the dates on which outbreaks were detected in lesser snow geese and outbreak detection in any of the other species (all $P>0.05$), suggesting that early lesser snow goose mortality was independent of other species.

DISCUSSION

Avian cholera mortality in waterfowl species at spring staging areas in the Rainwater Basin of Nebraska occurred in almost every year of the last quarter of the 20th century. Between 1976 and 1988, patterns associated with initiation of outbreaks and species mortality were inconsistent and varied among species. However, coinciding with a dramatic increase in abundance of midcontinent lesser snow geese and increasing usage of the RWB by this species, mortality in lesser snow geese species far exceeded mortality in other species. From 1989 to 1999, lesser snow geese made up the majority of waterfowl carcasses collected during avian cholera outbreaks, and annual mortality in several waterfowl species was positively related to lesser snow goose mortality. We also found an earlier detection of avian cholera outbreaks during 1989–1999 relative to 1976–1988. This earlier detection of outbreaks is consistent with observations by wetland biologists of the timing of lesser snow goose arrival to wetlands of the Rainwater Basin (Mack, unpubl. data). We note, however, that detection of an avian cholera outbreak may be influenced by detection probability of dead birds and carcass collection effort. Mortality in lesser snow geese, because of their white plumage and large size, may have been more easily detected than other species.

From 1989 to 1999, annual mortality in

TABLE 5. Relative mortality of waterfowl species (number of carcasses collected per 1,000 lesser snow goose carcasses) from Nebraska's Rainwater Basin during 1976–1988 and 1989–1999.

Species	1976–1988 Correlation	Relative mortality	t	df	P	1989–1999 Correlation	Relative mortality	t	df	P
White-fronted goose	0.01	−8.1	−0.03	8	0.98	0.06	2.3	0.17	7	0.87
Canada goose	0.04	108.3	0.12	8	0.91	0.97	46.6	11.50	7	<0.01
Mallard	0.06	−631.7	−0.17	8	0.87	0.99	64.2	20.83	7	<0.01
Northern pintail	0.08	−745.3	−0.22	8	0.83	0.97	131.4	11.46	7	<0.01
Other waterfowl	0.03	104.6	0.09	8	0.93	0.94	42.5	7.63	7	<0.01

TABLE 6. Relative mortality of waterfowl species (number of carcasses collected per 1,000 lesser snow goose carcasses) from Nebraska's Rainwater Basin during 1989–1999 with 1998 excluded.

Species	Correlation	Relative mortality	*t*	df	*P*
White-fronted goose	0.75	109.3	2.78	6	0.03
Canada goose	0.47	15.4	1.31	6	0.24
Mallard	0.86	50.5	4.18	6	0.01
Northern pintail	0.81	161.9	3.37	6	0.02
Other waterfowl	0.76	64.2	2.86	6	0.03

several waterfowl species using the RWB was positively related to annual mortality in lesser snow geese. The strongest associations with lesser snow goose mortality occurred for northern pintail, a species that appears highly susceptible to avian cholera, and white-fronted geese. Relative mortality in these two species was 162 northern pintail and 109 white-fronted geese per 1,000 lesser snow geese. Dense concentrations of highly gregarious lesser snow geese likely facilitate intraspecific transmission through bird-to-bird contact and indirectly through contamination of wetlands with virulent *Pasteurella*. The positive correlation between lesser snow goose mortality and mortality in other species of waterfowl also indicates potential interspecies transmission of disease, probably indirectly as a function of wetland contamination with *Pasteurella*.

Many of the factors that influence the initiation of avian cholera mortality or the severity of outbreaks remain poorly understood, and predicting the likelihood of large multi-species avian cholera outbreaks is difficult. However, the RWB appears to provide nearly ideal conditions for frequent outbreaks of avian cholera (Wobeser, 1992). Lesser snow goose populations using the RWB each spring now exceed 3 million birds, and nearly 1 million birds have been observed roosting on a single 700-acre wetland (Mack, US, unpubl. data). Although avian cholera mortality rates are difficult to document, they approached 10% during avian cholera outbreaks on a lesser snow goose nesting colony, and estimated mortality was ≥20% in the area with the highest nest density (Samuel et al., 1999c). Roosting and feeding lesser snow geese in the RWB can be found at much higher densities than occur on breeding colonies, thereby increasing the potential for transmission of infectious disease agents. During the 1989–1999 period we were unable to find a relationship between midcontinent species abundance and mortality from avian cholera in the RWB.

Ideally, assessments of wildlife disease outbreaks should focus on species-specific mortality rates from disease. Assessing disease mortality depends on the population at risk, severity of disease, surveillance efforts, and probability of detecting mortality. However, determination of mortality rates in waterfowl populations is challenging because of the annual variation in disease severity, spatial scale of outbreaks, high mobility of birds, interactions between disease and other mortality risks, and a number of other factors (Samuel, 1992). Local estimates of waterfowl populations using the RWB and bird density at wetlands would permit an assessment of mortality rates attributable to avian cholera during outbreaks and might help us understand the potential influence of crowding stress and contact on disease transmission. Alternatively, our assessment of disease patterns based on carcass collection data contains several potential limitations and sources of bias. We suspect that our results could be affected by differential probability of detecting carcasses of different waterfowl species, by variation in annual surveillance and carcass collection, and by limitations in our analytic methods. Regression methods comparing either temporal trends in species mortality or associations between species are likely to underestimate the relationship because the independent and/or the response measurements contain errors associated with variable carcass detection probabilities and annual surveillance efforts. Many of our analyses compare mortality trends between highly visible and detectable snow geese and other less detectable species (e.g., ducks). This differential detection will also underestimate the strength of this association. However, if the level of surveillance and carcass detection increases with higher disease mortality, which we suspect because management efforts are designed to remove carcasses to reduce disease transmission, this might cause an overestimate of the regression coefficient. Overall, we believe the significant temporal trends and relationships between mortality in snow geese and other species are likely conservative estimates compared to actual mortality levels; however, nonsignificant results should be viewed with a degree of uncertainty because the potential biases and limitations described likely reduced our ability to detect significant trends.

Understanding the dynamics of disease transmission in multi-host systems, such as avian cholera in waterfowl communities, is extremely complex and challenging. These dynamics are further complicated by potential variations among species in abundance, susceptibility, mortality rates, mobility, behavior, and wetland use patterns that influence the risk of exposure to disease. Current information on avian cholera indicates that some waterfowl species serve as reservoirs and carriers of the disease, stress-related conditions play an important role in initiating outbreaks, and disease is transmitted among birds or through environmental contamination. Lesser snow geese have been documented as carriers of virulent *P. multocida* (Samuel et al., 2005), and these birds likely play an important role in spreading and transmitting the disease. We suspect that lesser snow geese have become one of the species responsible for annually transporting the disease agent to the RWB, played a role in the earlier occurrence of disease outbreaks, and transmit disease to other species. However, the positive correlation in mortality patterns among species using the RWB could reflect a common unknown mechanism responsible for disease severity and similar avian cholera mortality patterns among species.

Currently, it is not possible to evaluate whether avian cholera mortality is likely to be substantial enough to reduce the overabundant lesser snow goose population, nor is it completely clear what the long-term impact of the increasing abundance of this carrier species will have on other waterfowl species susceptible to avian cholera. A better understanding of local waterfowl community dynamics as well as of the ecology of avian cholera is needed, including research to identify which species are competent carriers of *P. multocida*, ecologic factors that promote outbreaks, and how bird density, species composition, carcass density, and wetland contamination affect transmission of the disease. Additional research on waterfowl community dynamics before and during avian cholera outbreaks at winter or spring staging areas will provide a means for increasing our understanding of rates and mechanisms of multi-species disease transmission and persistence.

ACKNOWLEDGMENTS

We thank R. R. Cox, Jr, K. E. Church, M. P. Vrtiska, and T. Moser for their reviews and improvements to this manuscript. We especially thank the past and present staff of the Rainwater Basin Wetland Management District and the Nebraska Game and Parks Commission for their dedication to waterfowl management and sharing historic records. We thank the USGS–National Wildlife Health Center for partial funding. We are grateful to three anonymous reviewers for their helpful comments and suggestions on a previous version of this manuscript.

LITERATURE CITED

Abraham, K. F., and R. L. Jefferies. 1997. High goose populations: Causes, impacts, and implications. *In* Arctic ecosystems in peril: Report of the Arctic goose habitat working group, B. D. J. Batt (ed.). Arctic Goose Joint Venture Special Publication, US Fish and Wildlife Service, Washington, D.C., and Canadian Wildlife Service, Ottawa, Ontario, pp. 7–72.

Blanchong, J. A., M. D. Samuel, D. R. Goldberg, D. J. shadduck, and L. H. Creekmore. 2006. Wetland environmental conditions associated with the risk of avian cholera outbreaks and the abundance of *Pasteurella multocida*. Journal of Wildlife Management. In press.

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. Journal of Wildlife Management 48: 399–406.

Farrar, J. 1982. The rainwater basin, Nebraska's vanishing wetlands. Nebraska Game and Parks Commission, Lincoln, Nebraska. 14 pp.

Friend, M. 1981. Waterfowl management and waterfowl disease: Independent or cause and effect relationships? Transactions of the North American and Natural Resources Conference 46: 94–103.

———. 1987. Avian cholera. *In* Field guide to wildlife diseases, Vol. 1. General field procedures and disease of migratory birds, M. Friend and C. J. Laitman (eds.). US Fish and Wildlife Service, Resource publication 167, Washington, D.C., pp. 69–82.

———. 1992. Environmental influences on major waterfowl diseases. Transactions of the North American Wildlife and Natural Resources Conference 57: 517–525.

Gersib, R. A., K. F. Dinan, J. D. Kauffeld, P. J. Gabig, J. E. Cornely, G. E. Jasmer, J. M. Hyland, and K. J. Strom. 1992. Rainwater Basin Joint Venture implementation plan, Nebraska Game and Parks Commission, Lincoln, Nebraska, 56 pp.

Humberg, D. D., D. Graber, S. Sheriff, and T. Miller. 1983. Estimating autumn-spring waterfowl nonhunting mortality in north Missouri. Transactions of the North American Wildlife and Natural Resources Conferences 48: 241–256.

Samuel, M. D. 1992. Influence of disease on a population model of mid-continent mallards. Transactions of the North American Wildlife and Natural Resources Conference 57: 486–498.

———, D. J. Shadduck, D. R. Goldberg, V. Baranyuk, L. Sileo, and J. I. Price. 1999a. Antibodies against *Pasteurella multocida* in snow geese in the western arctic. Journal of Wildlife Diseases 35: 440–449.

———, J. Y. Takekawa, V. V. Baranyuk, and D. L. Orthmeyer. 1999b. Effects of avian cholera on survival of lesser snow geese *Anser caerulescens*: an experimental approach. Bird Study 46: S239–S247.

———, ———, G. Samelius, and D. R. Goldberg. 1999c. Avian cholera in lesser snow geese nesting on Banks Island, Northwest Territories. Wildlife Society Bulletin 27: 780–787.

———, D. J. Shadduck, D. R. Goldberg, and W. P. Johnson. 2005. Avian cholera in waterfowl: the role of lesser snow and Ross' geese as disease carriers in the Playa Lakes region. Journal of Wildlife Diseases 41: 48–57.

Smith, B. J., and K. F. Higgins. 1990. Avian cholera and temporal changes in wetland numbers and densities in Nebraska's Rainwater Basin Area. Wetlands 10: 1–5.

———, ———, and W. L. Tucker. 1990. Precipitation, waterfowl densities and mycotoxins: their potential effect on avian cholera epizootics in the Nebraska Rainwater Basin Area. Transactions of the North American Wildlife and Natural Resources Conference 55: 269–282.

Stuzenbaker, C. D., K. Brown, and D. Lobpries. 1983. An assessment of the accuracy of documenting waterfowl die-offs in a Texas coastal marsh. Special Report, Federal Aid Project W-106-R, Texas Parks and Wildlife Department, Austin, Texas, 21 pp.

Tiner, R. W. 1984. Wetlands of the United States: Current status and recent trends. US Fish and Wildlife Service, Washington, D.C.

Wilkins, K. A., and E. G. Cooch. 1999. Waterfowl population status, 1999. US Fish & Wildlife Service, Department of the Interior, Washington, D. C., 33 pp. + appendices.

Windingstad, R. M., J. J. Hurt, A. K. Trout, and J. Cary. 1984. Avian cholera in Nebraska's rainwater basins. Transactions of the North American Wildlife and Natural Resources Conference 49: 577–583.

———, S. M. Kerr, R. M. Duncan, and C. J. Brand. 1988. Characterization of an avian cholera epizootic in wild birds in western Nebraska. Avian Diseases 32: 124–131.

———, M. D. Samuel, D. D. Thornburg, and L. C. Glaser. 1998. Avian cholera mortality in Mississippi Flyway Canada geese. *In* Biology and management of Canada geese, D. H. Rusch, M. D. Samuel, D. D. Humburg, and B. D. Sullivan (eds.). Proceedings of the International Canada Goose Symposium, Milwaukee, Wisconsin, pp. 283–289.

Wobeser, G. 1992. Avian cholera and waterfowl biology. Journal of Wildlife Diseases 28: 674–682.

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

ZINKL, J. G., N. DEY, J. M. HYLAND, J. J. HURT, AND K. L. HEDDLESTON. 1977. An epornitic of avian cholera in waterfowl and common crows in Phelps County, Nebraska, in the spring, 1975. Journal of Wildlife Diseases 13: 194–198.

Received for publication 3 January 2005.