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POSTMORTEM EVALUATION OF REINTRODUCED MIGRATORY WHOOPING CRANES IN EASTERN NORTH AMERICA

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ABSTRACT: Reintroduction of endangered Whooping Cranes (*Grus americana*) in eastern North America has successfully established a migratory population between Wisconsin and Florida. Eighty birds (47 males, 33 females) were released between 2001 and 2006, and all birds were tracked following release with satellite and/or VHF monitoring devices. By the end of 2006, 17 deaths (12 males, five females) were recorded from this population. Postmortem findings and field data were evaluated for each bird to determine the cause of death. Causes included predation ($n=8$, 47%), trauma ($n=2$, 12%), and degenerative disease ($n=1$, 6%); the cause of death was undetermined for 35% ($n=6$) of the birds. Based on physical evidence, the primary predator of the birds was the bobcat (*Lynx rufus*). Limited roosting habitat availability or bird behavior were likely prime factors in the occurrence of predation. Traumatic injuries and mortality were caused by gunshot, electrical utility lines, and an unknown source. The lone case of degenerative disease was due to chronic exertional myopathy associated with translocation. Available postmortem testing did not indicate the presence of infectious disease in this limited sample.

Key words: Bobcat, *Grus americana*, gunshot, *Lynx rufus*, predation, trauma, utility line, Whooping Crane.

INTRODUCTION

The Whooping Crane (*Grus americana*) is one of the world's most endangered avian species, with only one remnant migratory population in central North America currently numbering approximately 260 individuals. Historically, Whooping Cranes in eastern North America migrated along the Appalachian Mountains from coastal New Jersey, South Carolina, and more southerly river deltas to the nesting grounds in the Hudson Bay area of Canada (Wisconsin Department of Natural Resources Whooping Crane Management Plan, 2006). In 1911, the last population of Whooping Cranes ($n=14$) in eastern North America was observed in Alachua County, Florida. The Whooping Crane Eastern Partnership (WCEP), a public-private consortium, was formed to conduct a multiyear reintroduction effort aimed at establishing a geographically separate, self-sustaining migratory population of Whooping Cranes in eastern North America.

We reviewed postmortem data from this population to identify causes of death and assess the health management of the reintroduction effort. Postmortem studies such as this are essential to understanding the success of endangered species reintroduction programs and are recommended by worldwide conservation planners (International Union for the Conservation of Nature, 1998). Published reports of the postmortem findings of reintroduced Whooping Cranes are limited but include case reports of lead and zinc toxicosis, avian cholera, mycobacteriosis, parasite infections, and a review of mortality factors for nonmigratory Whooping Cranes in Florida (Stroud et al., 1986; Snyder et al., 1991; Spalding et al., 1997; Varela et al., 2001; Spalding, 2003). The objective of this paper is to describe the

pathologic changes and causes of mortality observed in reintroduced migratory Whooping Cranes in eastern North America.

MATERIALS AND METHODS

Between 2001 and 2006, 80 captive-bred Whooping Cranes were reintroduced by one of two methods to create an eastern migratory population (EMP). The first method utilized costume-reared cranes (Wellington et al., 1996; Duff et al., 2001; $n=71$, 42 males, 29 females) trained to fly with ultralight aircraft along an artificial migration route between Necedah National Wildlife Refuge (NNWR; 44°3′N, 90°9′W), Wisconsin, and Chassahowitzka National Wildlife Refuge (CNWR; 28°43′N, 82°38′W), Florida, between October and December of their hatch year (Duff et al., 2001; Hartup et al., 2005). Cranes were maintained for variable lengths of time in a top-net covered satellite pen upon arrival at CNWR. Eventually, cranes were released to an uncovered pen and provided supplemental food until they departed on northward migration the following spring. Release was considered the date of removal from the top-netted pen at CNWR. One bird was withdrawn from the ultralight cohort in 2004 because of acute loss of primary remiges at NNWR. The bird was later released in October at NNWR after the feathers had regrown. In the second method, costume-reared cranes ($n=9$, five male, four female) were released at NNWR in October of their hatch year. This release method has been described for Sandhill Cranes (*Grus canadensis*; Urbanek et al., 2005). The birds were not trained with ultralight aircraft and generally followed other wild adult cranes on their first migration. Release was considered to be the date when they were no longer kept overnight in a top-netted pen at the rearing area in NNWR.

All released birds were equipped with a VHF radio transmitter (Advanced Telemetry Systems, Isanti, Minnesota, USA) mounted on an individually color-coded leg band. A few birds (two to six birds per year) also carried similarly mounted satellite transmitters on the leg opposite the VHF transmitter (Microwave Telemetry, Columbia, Maryland, USA). All birds were tracked by VHF telemetry when possible throughout the migration route by personnel in ground tracking vehicles or aircraft.

All released individuals from the EMP that died by 31 December 2006 are included in this review. Postmortem assessment of each Whooping Crane included a two-part evaluation. The first was a field assessment at the carcass location. Field personnel photographed the remains and mortality site and collected the following data: date, time of carcass collection, identification and band numbers, degrees and minutes of latitude and longitude, county and state where the carcass was recovered, carcass position, habitat, presence and characteristics of water, environmental conditions (current weather, drought, flood, etc.), nearby human constructions (utility lines, buildings, fences, roads), sighting and tracks or other signs of potential predators and scavengers, last sighting of the bird (or valid transmitter signal) prior to death, and any unusual antemortem behavior if known.

Because most carcasses were recovered at sites presumed to have been used for roosting, the proximity to proper crane roosting habitat was evaluated. Roosting cranes most commonly use wetlands that have a seasonal or semipermanent water source. Roosting sites used by migratory Whooping Cranes have several consistent characteristics: average water depth of 20 cm (range 5–46 cm), clear to turbid water, sand or soft mud substrate, slight shoreline slope, unobstructed visibility of >90 m in all directions, and often concurrent use by other cranes (Austin and Richert, 2005). We deemed dry land or wetlands with minimal water, a hard substrate, steep shoreline slope, proximity to tall terrestrial vegetation that could conceal a potential predator, and poor visibility as inappropriate roosting habitat for Whooping Cranes. We defined poor roosting behavior as an apparent choice of the bird to roost in inappropriate habitat when an appropriate roosting location was available nearby. Inadequate roosting habitat was defined as a lack of proper roosting habitat in the vicinity of the site of carcass retrieval, suggesting the bird had no choice but to roost in inappropriate habitat. We were unable, however, to determine the time of day when death occurred in many cases, and remain unsure whether deaths were associated with roosting, loafing, or foraging activities.

For the second part of the evaluation, necropsies were performed. Carcasses were chilled and shipped by overnight commercial carrier or hand-carried for necropsy to the US Geological Survey–National Wildlife Health Center (NWHC), Madison, Wisconsin; the University of Florida, Gainesville, Florida; or the US Fish and Wildlife Service Forensics Laboratory, Ashland, Oregon (frozen carcasses only), depending on the location and circumstances of death. A complete gross necropsy was performed from suitable carcasses, sam-

ples of brain, heart, major artery, lung, trachea, liver, kidney, spleen, thyroid gland, adrenal gland, esophagus, proventriculus, ventriculus, and intestine were fixed in 10% buffered formalin, paraffin embedded, sectioned at 5 μm, and stained with hematoxylin and eosin for light microscopy. Sections of tissues containing granulomatous lesions were stained by the Ziehl-Nielssen acid-fast procedure (Luna, 1968). At necropsy, tissue samples were collected and submitted to appropriate laboratories for microbiologic, virologic, parasitologic, and toxicologic tests based on gross lesions and differential diagnoses, and these varied per case. In general, tissue samples were embedded in Sabouraud dextrose medium for fungal culture (Baron and Finegold, 1990) or enriched for *Mycoplasma* spp. in SP4 broth (Waites et al., 2004). Brevetoxin analysis was performed by the Florida Fish and Wildlife Conservation Commission Research Laboratory, St. Petersburg, Florida. Other diagnostic laboratory methods were similar to routine testing and performed as follows.

Routine screening tests were performed on each carcass, to the extent possible depending on carcass condition and availability. All carcasses were radiographed for evidence of metallic foreign bodies prior to dissection. Bacterial isolations from the upper and lower intestinal tract were done by routine aerobic culture techniques using trypticase soy agar containing 5% sheep blood and eosin methylene blue agar (BD Diagnostic Systems, Franklin Lakes, New Jersey, USA) and by a *Salmonella*-selective culture method using both xylose lysine tergitol 4 agar (Miller et al., 1991) and Miller-Mallinson agar (BD Diagnostic Systems) after enrichment in Rappaport-Vassiliadis broth (Remel, Lenexa, Kansas, USA). General viral isolations from liver and lower intestine were attempted in embryonated specific-pathogen-free (SPF) chicken eggs 9 days of age (Senne, 1998) and Muscovy duck embryo fibroblast culture (Docherty and Slota, 1988). West Nile virus (WNV) isolation was attempted in Vero cell culture from brain, feather pulp, or combined spleen and kidney; cultures were screened for WNV by reverse transcriptase–polymerase chain reaction (RT-PCR; Docherty et al., 2004). Beginning in 2005, tracheal and/or cloacal swab samples in viral transport media were screened for avian influenza virus via culture in embryonated SPF chicken eggs (Senne, 1998) and RT-PCR toward the matrix gene (Spackman et al., 2002). Serum collected from clotted heart blood was tested using virus neutralization methods for antibodies against inclusion body disease of cranes virus (IBDC; Docherty and Romaine, 1983), WNV (Docherty et al., 2006), and eastern equine encephalitis (EEE) virus (Olsen et al., 2005). Liver, lung, and kidney were examined under a dissecting microscope, and squash preparations of these tissues, as well as Giemsa-stained blood smears and scrapings of the gastrointestinal mucosa were examined microscopically for parasites. Feces were examined microscopically for parasite eggs or oocysts after concentration using Sheather's sugar flotation technique (Garcia and Bruckner, 1993). Representative adult parasite specimens were deposited in the Harold W. Manter Laboratory of Parasitology (HWML, University of Nebraska State Museum, Lincoln, Nebraska, USA). Lead concentration in the liver was measured by atomic absorption spectrophotometry (Boyer, 1984). Brain cholinesterase activity was measured to detect organophosphate and carbamate pesticide toxicity (Hill and Fleming, 1982); values were compared with activity levels previously determined from 10 Whooping Cranes in which pesticide toxicity had been ruled out (NWHC 2005 cumulative average: 15.40± 1.80 μmoles/min/grams of tissue).

Potential predation events were evaluated based on available physical evidence and field signs. Predation of Whooping Cranes is known to occur by several species including bobcat (*Lynx rufus*), coyote (*Canis latrans*), alligator (*Alligator* sp.), and Golden (*Aquila chrysaetos*) and Bald Eagles (*Haliaeetus leucocephalus*) (Ellis et al., 1999; Spalding, 2003).

Based upon observed attack and over 80 presumed bobcat predation events in a resident Whooping Crane population in Florida, the following progression of events appear to be characteristic depending upon the amount of time the bobcat spends with the carcass: 1) a crushing injury of distal cervical vertebral bodies, commonly cervical vertebrae 10–13, 2) the pectoral muscle is consumed, 3) the head and neck are disarticulated near base of neck, 4) the cranial coelomic cavity is entered through ribs and tissues are consumed, 5) the musculature and dorsal spinous processes of thoracic vertebrae are consumed and chewed on, respectively, and finally 6) the tissues of the caudal coelomic cavity are consumed (M. Spalding, unpubl.). Feathers are found at the site of the kill, and the carcass may be dragged to a location with vegetative cover where vegetation, dirt, or snow is scraped over the carcass (i.e., cached; Hansen, 2007). Bobcats rarely break long bones (M. Spalding, unpubl.). During gross examination, vertebral body crushing injury, hemorrhage from major vessels and soft tissues, and

associated soft tissue damage in the cervical region are apparent.

Coyote predation has similar pathology to the bobcat, with the addition of long bone fractures; however, the carcass is not usually dragged or cached (M. Spalding, unpubl.). Alligator predation is characterized by twisting of skin around the limb or region initially attacked, as alligators often rotate their whole body immediately after obtaining prey. A Bald Eagle has been observed preying on young chicks in central Florida (M. Folk, pers. comm.). Golden Eagles attack midflight from above and behind, grasping the crane and riding it toward the ground, and releasing it as it strikes the ground (Ellis et al., 1999). Expected postmortem lesions would include laceration of skin and deep tissues along the dorsum associated with the grip of the talons, puncture wounds to viscera in the posterior coelom, and fractures and internal injuries associated with impact trauma (Windingstad et al., 1981).

Pertinent field factors, necropsy findings, and other abnormal conditions that may have contributed to mortality were evaluated, when available, to arrive at a cause of death. We define the cause of death as the event that initiated the bird's removal from the population (ultimate cause), not necessarily the condition responsible for termination of life (proximate cause). The cause of death was left undetermined for cases in which both the pathologic lesions and field observations were nonspecific or where interpretation was precluded by postmortem change, scavenging, or other disturbance.

RESULTS

Seventeen deaths were recorded (12 males, five females) among the 80 migratory Whooping Cranes released in eastern North America by the end of 2006. All 63 remaining birds were confirmed alive at the end of the study period. Gross necropsies were performed on all 17 carcasses and six histopathologic examinations were performed. Ten carcasses were decomposed and/or scavenged such that insufficient soft tissue remained for histopathology. One of the fresh carcasses did not have histopathology performed as the postmortem examination was limited to collection of forensic data. One of the decomposed carcasses had intact feathers with vascular pulp sufficient for WNV testing. In total, seven of the cases had at least one additional diagnostic test applied with gross necropsy ± histopathology.

Causes of death include predation ($n=8$, 47%), trauma ($n=2$, 12%), degenerative disease ($n=1$, 6%), and undetermined ($n=6$, 35%). Individual causes of mortality are presented in Table 1. All deaths with an identified cause of mortality were due to noninfectious causes (11/17, 65%). Deaths attributed to predation occurred at the following locations: CNWR, Florida ($n=4$), Cape Romain National Wildlife Refuge (CRNWR), South Carolina ($n=1$), and central Wisconsin surrounding NNWR ($n=3$). All predatory deaths at CNWR and CRNWR were attributed to bobcats; the predator involved in the three Wisconsin cases remains undetermined.

Carcasses from six of the eight predation mortalities were recovered in habitat deemed inappropriate for roosting (none appeared to have been moved from the presumed kill site). Of these, four carcasses were found in inappropriate roosting locations with proper roosting habitat within 1 km, suggesting poor roosting behavior by the birds. Two carcasses were found in an inappropriate roosting location with no nearby proper roosting habitat, suggesting only inadequate roosting habitat was available at the time of death.

Traumatic deaths included one bird that died following impact trauma with an undefined object, which caused acute hepatic hemorrhage and necrosis (bird 04-03) secondary to crush injury, and one bird (bird 15-02) with gunshot trauma sustained during the waterfowl hunting season. Radiographic diagnosis confirmed bird 15-02 had at least nine gunshot wounds in various regions of the body, including the head. Recovery and examination by a firearms examiner revealed multiple sizes of pellets indicating that the bird may have been shot by at least two different shotgun pellet loads.

Nonfatal trauma was identified in five

TABLE 1. Causes of mortality in reintroduced migratory Whooping Cranes in eastern North America, 2001–2006.

Bird identification	Gender	Date of death	Age at death (yr)	Location of death (US state)	Carcass status	Postmortem condition	Field factors	Cause of mortality	Additional details	Diagnostic tests performed[a]
4-01	M	Dec. 2001	<1	FL	Partial	Fresh	Poor roosting behavior	Predation		R, G, H, P
6-01	M	May 2005	4	WI	Partial	Fresh	Power line	Predation	Impact trauma	R, G, H, M, P, VS, VI, T
10-01	F	Jan. 2002	<1	FL	Scant	Decomposed	Poor roosting behavior	Predation		R, G
3-02	F	July 2006	4	WI	Scant	Decomposed	Inadequate roosting habitat	Undetermined		R, G
7-02	F	Aug. 2003	1	WI	Complete	Fresh	Translocation	Chronic exertional myopathy	Aspiration pneumonia	R, G, H, M, P, VS, VI, T
8-02	M	Dec. 2006	4	IN	Complete	Fresh	Power line	Undetermined	Aspiration of ingesta; healed skull fracture	R, G, H, M, P, VS, VI, T
14-02	F	Feb. 2005	2	FL	Scant	Decomposed	Intraspecific aggression	Predation		R, G
15-02	F	Dec. 2004	2	AL	Partial	Fresh	Waterfowl hunting	Gunshot trauma		R, G, P
2-03	M	July 2006	3	WI	Partial	Decomposed	Inadequate roosting habitat	Predation		R, G
4-03	M	Oct. 2005	2	WI	Complete	Fresh		Impact trauma	Hepatic necrosis	R, G, H, M, P, VS, T
5-03	M	Nov. 2004	1	SC	Scant	Decomposed	Poor roosting behavior	Predation		R, G
19-03	M	July 2004	1	MI	Partial	Decomposed		Undetermined	Gunshot trauma; nonlethal	R, G, P
5-04	M	Mar. 2005	<1	FL	Partial	Fresh	Poor roosting behavior	Predation	Mild encephalitis	R, G, H, VS, VI, T
14-04	M	May 2005	1	WI	Scant	Decomposed	Inadequate roosting habitat	Predation	Left femur fracture	R, G, M
17-04	M	May 2006	2	WI	Partial	Decomposed		Undetermined	Impact trauma	R, G, VS
18-04	M	July 2005	1	WI	Scant	Decomposed	Power line	Undetermined		R, G, VS
22-05	M	July 2006	1	MI	Scant	Decomposed		Undetermined		R, G

[a] G = gross necropsy, H = histopathology, M = microbiology, P = parasitology, R = radiology, T = toxicology, VI = virus isolation, VS = virus serology.

additional birds, including gunshot ($n=1$), impact trauma from an unknown source ($n=1$), and utility line collision ($n=3$). Bird 19-03 sustained a single no. 2 lead shot pellet wound in the foot, fracturing the first phalanx of the right third digit. The wound was not considered life threatening. Bird 17-04 was found with a right humero-ulnar joint luxation, which we believe was caused by impact trauma with an unidentified object. Bird 06-01 sustained a comminuted fracture of the left tarsometatarsus secondary to a presumed utility line strike (present on adjacent property) and was observed in woody cover that was considered inappropriate habitat for roosting. The bird was killed by a predator 11 days following discovery of the injury. Localized osteomyelitis at the fracture site was diagnosed postmortem. Bird 08-02 was found sternally recumbent under a utility line unable to stand or flee capture when approached. The bird was non–weight bearing on the right leg and subcutaneous emphysema was noted about the proximal left leg. The bird was transported to a zoologic facility for veterinary care but died 3 days later following acute aspiration of feeding formula. Antemortem diagnostic testing failed to reveal a cause for the bird's clinical presentation. Gross and microscopic examination found no evidence of significant trauma, electrocution or exertional myopathy, but revealed a small, healed, depressed skull fracture beneath a cutaneous scar. No disease agents or toxins were identified by bacterial and viral cultures of various organs, serology, or toxicology in this case. Only a small number of gastrointestinal parasites were present. Spinal cord injury was not ruled out in this case, however, and subcutaneous emphysema commonly results from impact trauma in birds. The proximity of the bird to the utility line suggests impact was likely. Bird 18-04 was found with left proximal humerus and right distal radius and ulna fractures immediately below a utility line. The decomposed state of the carcass precluded determination of a definitive cause of death, but the fractures and proximity of the utility line suggest direct mortality from a collision.

One of the 17 birds was diagnosed with a degenerative disease of iatrogenic origin: chronic exertional myopathy following a translocation effort. Details of this case have been published previously (Hanley et al., 2005).

The cause of death for six birds remains undetermined. The carcasses of most of these birds ($n=5$) were severely scavenged and/or decomposed, greatly limiting postmortem examinations and laboratory testing. The carcass of bird 08-02 was complete and well preserved (see above); however, postmortem evaluation failed to determine the cause of the bird's condition.

Infectious disease was not found to be a cause of mortality in any of the 17 deaths. Results of routine screening tests are summarized in Table 2. No antibodies against EEE virus ($n=3$) or IBDC virus ($n=3$) were detected in the serum and WNV antibodies were detected in only one bird without evidence of infection (bird 06-01). In addition, serum from bird 05-04 diagnosed with mild cerebral vascular lymphocytic cuffing showed no evidence of antibodies to Highlands J virus or St. Louis encephalitis virus, and virus isolation from the brain did not detect any virus. Virus isolation was attempted from the liver and intestine when possible, and no viruses were detected. Avian influenza virus testing (initiated in 2005) did not detect any evidence of these viruses.

Gastrointestinal parasites were present in small numbers in all carcasses that had an intact gastrointestinal tract ($n=4$), but no deaths were attributed to parasites. The trematodes *Clinostomum* sp. were found in esophagus ($n=1$), and *Echinoparyphium* sp. ($n=1$) and *Echinostoma trivolvis* ($n=1$) were found in intestine, HWML nos. 48888-20098-001, 48887-20098-001, and 48886-19611-001, respectively. Bro-

TABLE 2. Results of routine postmortem screening tests performed on reintroduced migratory Whooping Cranes from eastern North America, 2001–2006.

Laboratory	Test[a]	Total no. birds tested	Positive results	Description of results
Radiology	Whole body radiographs	17	2	Metal shot found in foot (1) and various locations (1)
Virology	Serology			
	WNV	6	1	West Nile antibody titer 1:20
	EEE	3	0	
	IBDC	3	0	
	Virus isolation			
	Liver	4	0	
	Intestine	2	0	
	Kidney + spleen—WNV	2	0	
	Feather pulp—WNV	2	0	
	Cloacal/tracheal swab—AI	2	0	
Toxicology	Brain cholinesterase activity	3	0	
	Lead (liver)	4	0	
Parasitology	Lung	3	0	
	Liver	4	0	
	Kidney	4	0	
	Upper gastrointestinal tract	2	1	*Clinostomum* sp.
	Intestine	2	2	*Echinoparyphium* sp., *Echinostoma trivolvis*
	Ectoparasites	7	0	
	Blood smear	5	0	
Microbiology	Aerobic culture			
	Upper small intestine	2	2	*Escherichia coli*, *Enterococcus* sp., *Plesiomonas shigelloides*, *Edwardsilla tarda*
	Lower intestine	2	2	*Eschericia coli*, *Enterococcus* sp., *Enterobacter* sp., *Aeromonas hydrophila*, *Citrobacter braakii*, *Edwarsdiella tarda*, *Plesiomonas shigelloides*, *Clostridium* sp., *Clostridium perfringens*, *Lactobacillus* sp.
	Salmonella culture			
	Intestine	3	0	

[a] AI = avian influenza virus, EEE = eastern equine encephalitis virus, IBDC = inclusion body disease of cranes virus, WNV = west Nile virus.

ken or degenerated nematodes resembling *Tetrameres* sp. and *Capillaria* sp. were found in proventriculus ($n=1$), and intestine ($n=1$), respectively, but the specimens were too degraded for positive identification. Two birds (birds 04-03 and 05-04) had incidental granulomas in lung and/or liver consistent with disseminated visceral coccidiosis (DVC) (Spalding, 2003; Novilla and Carpenter, 2004), although the organisms (*Eimeria gruis*, *E. reichenowi*) were not detected by histopathologic examination.

Microbiologic evaluation of the upper and lower gastrointestinal tract yielded normal flora (Carpenter, 1993; Hoar et al., 2007). Bird 07-02 had bilateral conjunctivitis characterized by grossly visible pale plaques on the nictitans; no *Mycoplasma* sp. was isolated from culture of the lesion.

No lead was detected in liver samples ($n=4$; detection limit <0.25 ppm, wet weight). Brain cholinesterase activity ($n=3$) was considered within normal limits. A liver sample from one bird that was found dead in coastal Florida had no detectable brevetoxin.

DISCUSSION

The primary cause of mortality in reintroduced Whooping Cranes in the EMP was predation. Based on the available physical evidence, we attributed all of the predatory deaths that occurred in South Carolina and Florida in this migratory population to bobcats. In central Wisconsin, predation occurred in locations adjacent to NNWR, but with fewer indications (field factors and postmortem findings) of what predatory species was involved. Potential predators include coyote (locally abundant), domestic dog (*Canis lupus familiaris*), wolves (*Canis lupus lycaon*), Fisher (*Martes pennanti*), and bobcat; however, the last two are less abundant in central than in northern Wisconsin (Dhuey and Olson, 2006; Hansen, 2007). Avian predators were deemed unlikely due to lack of physical evidence, and in the case of Golden Eagles, the lack of breeding or resident eagles at the time of the Whooping Crane mortalities. Bobcats have been documented in two of three counties where Whooping Crane predation events were recorded and a county adjacent to NNWR by state biologists (R. E. Rolley, pers. comm.). Postmortem analysis in the three Wisconsin cases was not consistent with lesions specific to a particular species. The femur fracture observed in bird 14-04 may suggest a larger canid predator or scavenger; the carcass of bird 03-02 was cached, a common bobcat behavior.

In most of the deaths caused by predation, the habitat in which the carcasses were recovered was deemed inappropriate for roosting Whooping Cranes. These circumstances, whether created by bird choice (poor roosting behavior) or limited availability of roosting habitat, likely influenced the probability of the predation event. As most of the predators of Whooping Cranes are nocturnal, predation likely occurred at night while the birds were roosting. On a landscape level, there may be no practical management solution to increase the availability of adequate roosting habitat for cranes. When a mosaic of habitat is present however, Whooping Cranes may show improved orientation to appropriate roost sites following release if they are provided adequate roosting conditions while captive or in a protected environment during the rearing and soft release process (such as at CNWR). Such strategies have greatly improved the first-year survival rates of released captive-reared Whooping Cranes in central Florida (Gee et al., 2001).

It is not known the extent to which costume-rearing affects the birds' behavior. A study of the same captive-reared Whooping Cranes in central Florida had increased postrelease survival when the chicks exhibited two behaviors during rearing: more frequent foraging and less frequent walking (Kreeger et al., 2006). That study also suggested, in released birds, that frequent vigilant behavior (alert body posture, visually scanning environment) contributed positively to their survival. Behavior of the birds in the EMP was not studied, but it could be postulated that a lack of parent-taught behaviors (i.e., infrequent vigilance) may have increased predation susceptibility.

Trauma was a significant cause of morbidity in this population, but only two birds, birds 15-02 and 04-03, had a confirmed traumatic cause of mortality other than predation. The two birds with gunshot injury, birds 15-02 and 19-03, indicate the need for continued hunter education, especially during open waterfowl seasons. We also documented three deaths that were likely influenced by the presence of utility lines, birds 06-01, 08-02, and 18-04. Electrical utility lines have been a known threat to cranes for over 50 yr (Walkinshaw, 1956). In comparison to Sandhill Cranes or waterfowl, the Whooping Crane population has been shown to be more vulnerable to morbidity and mortality associated with utility lines (Brown et al., 1987). These published reports describe blunt force trauma from collision, rather than electrocution, which

is consistent with our findings. Modifications to existing utility lines, such as placing lines away from major roosting sites or loafing areas, using visibility enhancement devices (marker balls and bird diverters) and removal of the static wire, may be of benefit (Howard et al., 1985; Brown and Drewien, 1995), but will be difficult to assess with a low density population of Whooping Cranes.

Infectious diseases were not confirmed as a cause of mortality for any of the 17 deaths. Birds released in 2001 were vaccinated for EEE, and all were vaccinated for WNV and EEE after 2003. No birds were vaccinated in 2002. Of the three birds serologically evaluated for antibodies to EEE virus, two had been vaccinated. Of the five birds serologically evaluated for WNV antibodies, three had been vaccinated. One of the two unvaccinated birds, bird 06-01, had detectable antibodies, suggesting the bird had natural exposure to the virus. This bird had no postmortem lesions typical of WNV infection, and virus isolation attempts from tissue (kidney, spleen, lower intestine, and liver) were unsuccessful. The brain was not evaluated as the carcass was found decapitated. Due to the lack of lesions in any tissue, we have no evidence that this bird was experiencing clinical disease associated with WNV infection at the time of its death. In bird 05-04, encephalitis may have predisposed this bird to attack by other Whooping Cranes and/or predation.

Parasites discovered postmortem were not associated with overt disease or lesions. The parasites found in the upper and lower gastrointestinal tracts were either nematodes or trematodes. While nematodes and trematodes are commonly reported in cranes (Windingstad, 1978; Gaines et al., 1984; Spalding et al., 1996; Mowlavi et al., 2006), this is the first report of these particular parasites in Whooping Cranes. Trematodes are generally transmitted indirectly via molluscs, fish, amphibians, and aquatic invertebrates. Their presence is not surprising because free-ranging Whooping Cranes regularly forage in aquatic environments and probably consume these intermediate hosts. Nematodes may be of some concern, as *Capillaria* sp. are known to be pathogenic in Sandhill Cranes (Carpenter, 1993), although no lesions were seen in these birds. Although DVC is a common pathogenic condition of cranes, especially chicks in captivity (Novilla and Carpenter, 2004; Kwon et al., 2006; Sarashina et al., 2006), both cases reported here appear incidental. All Whooping Cranes in the EMP were fed an artificial diet containing the coccidiostatic drug monensin from hatching until direct release at NNWR (5 mo of age) or departure from the CNWR pensite (10 mo of age). We expect this treatment limited the development of clinically relevant disease from DVC in the reintroduced birds.

Undetermined causes of mortality are expected in a postmortem study of a free-ranging population with a wide dispersal or migratory range. Five of six of these cases were decomposed and consisted of partial or scant remains. The EMP is distributed across multiple states, especially during spring and fall migration, making regular monitoring less frequent for each bird. Determination of carcass location can take from a day to a few weeks depending on the last known location of the bird and accessibility of the site (private lands, terrain features, etc.); however, carcass retrieval occurred within days once a stationary signal was identified. Additionally, elevated ambient temperature increases the rate of decomposition. Of the 10 decomposed carcasses, seven were collected during summer months (May–August) and three in southern climates, which can be warm even in the fall and winter (November, South Carolina; January and February, Florida).

The preliminary EMP mortality data reported here are similar to the distribution of factors reported over a 12-yr period from a reintroduced, nonmigratory central Florida population (FP) which involved the release of captive-reared Whooping

Cranes released at 6–10 mo of age (M. Spalding, unpubl.). Predation mortality was 47% in the EMP and 58% (108/186) in the FP. Similar to the EMP, bobcat predation caused the majority of diagnosed mortality in the nonmigratory population. Traumatic injury accounted for 12% of mortality in the EMP and 7.5% (14/186) in the FP. Of the traumatic deaths in the FP, nearly all were due to gunshot or utility line strike injuries. Cause of death was undetermined in 35% of the EMP deaths and 27% (50/186) of the FP deaths. The primary difference between the EMP and FP appears to be the lack of infectious disease-related mortality: 0% in the EMP versus 7.5% (14/186) in the FP. Mortality in the FP has been attributed to EEE, DVC, aspergillosis, and a wasting syndrome associated with infectious bursal disease virus exposure (Spalding et al., 2004). The absence of infectious diseases in the EMP may be due to the small population size (resulting in reduced disease transmission, sporadic exposure to disease agents, and/or detection bias due to small sample size) or affected by limited diagnostic opportunities available from decomposed and scavenged carcasses.

This paper reports the comprehensive postmortem findings in a reintroduced population of captive-reared Whooping Cranes in North America. We observed no overt disease problems in the carcasses examined, and therefore no indication of a population-limiting disease of captive origin at this time that may jeopardize the success of the reintroduction. Lack of exposure to infectious agents following release seems unlikely however, as these birds have a very broad dispersal range along their migratory route. Additional postrelease health screening is warranted to substantiate these findings.

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