WHOOPING CRANE MORTALITY AT PATUXENT WILDLIFE RESEARCH CENTER, 1982-95

Glenn H. Olsen
Patuxent Wildlife Research Center

Joanna A. Taylor
Patuxent Wildlife Research Center

George F. Gee
Patuxent Wildlife Research Center

Follow this and additional works at: http://digitalcommons.unl.edu/nacwgproc

Part of the Behavior and Ethology Commons, Biodiversity Commons, Ornithology Commons, Population Biology Commons, and the Terrestrial and Aquatic Ecology Commons

http://digitalcommons.unl.edu/nacwgproc/230

This Article is brought to you for free and open access by the North American Crane Working Group at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in North American Crane Workshop Proceedings by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.
WHOOPING CRANE MORTALITY AT PATUXENT WILDLIFE RESEARCH CENTER, 1982–95

GLENN H. OLSEN, Patuxent Wildlife Research Center, 11610 American Holly Drive, Laurel, MD 20708, USA
JOANNA A. TAYLOR,1 Patuxent Wildlife Research Center, 11510 American Holly Drive, Laurel, MD 20708, USA
GEORGE F. GEE, Patuxent Wildlife Research Center, 11510 American Holly Drive, Laurel, MD 20708, USA

Abstract: Whooping cranes (Grus americana) have been reared at Patuxent Wildlife Research Center since 1966. During 1982–95 there were 103 mortalities caused by infectious and parasitic diseases (46%), trauma (21%), anatomic abnormalities (17%), miscellaneous conditions (12%), and open or no diagnoses (5%). The implications that disease may have on new whooping crane flocks in Florida and Canada are discussed, based on these mortality factors in captivity.

PROC. NORTH AM. CRANE WORKSHOP 7:243-248

Key words: congenital deformities, crane, disease, Grus americana, infectious disease, mortality, parasites, scoliosis, whooping crane.

The endangered species research program was established in 1965 (Brickson 1968) at the Patuxent Wildlife Research Center (Patuxent), Laurel, Maryland. Captive propagation forms an integral part of gene pool preservation, research, and reintroduction mission of the program. Researchers develop effective husbandry, medical, and reproductive techniques to build self-sustaining captive populations of critically endangered native species. Eventually, captive-bred animals are released to augment and restore wild populations. The most intensive effort in captive propagation research has focused on the whooping crane. The first whooping crane (an injured young-of-the-year) was transferred from Canada to the U.S. Bureau of Sport Fisheries and Wildlife in 1964 and moved to Patuxent in 1966. Patuxent received 6 eggs removed from nests in Wood Buffalo National Park, Northwest Territories, Canada in 1967 and 173 eggs in subsequent years.

The first successful breeding of whooping cranes at Patuxent occurred in 1975 (Ellis et al. 1992). Although a steadily increasing number of eggs has been produced annually, mortality factors, including disseminated visceral coccidiosis (Carpenter et al. 1980), eastern equine encephalitis virus (Dein et al. 1986), mycotoxins (Olsen et al. 1995), and delayed sexual maturity have kept recruitment rates low. Also, 73 eggs (61 fertile) produced at Patuxent were used in an unsuccessful reintroduction project at Grays Lake National Wildlife Refuge in Idaho (Ellis et al 1992). These factors slowed the growth of the captive flock. In 1989, 22 of 58 captive whooping cranes were transferred to the International Crane Foundation in Baraboo, Wisconsin, to minimize the impact a stochastic disaster would have on the captive breeding program and gene pool. From 1992 through 1995, 11 whooping cranes were transferred to the Devonian Science Center of the Calgary Zoo, Alberta, Canada, to establish a third captive flock. Other than papers documenting specific diseases, there has been 1 summary report of captive whooping crane mortality covering the years 1966–81 (Carpenter and Derrickson 1982).

Mortality information from captive flocks provides an understanding of the impact of disease on life span, reproduction, and population demographics. Mortality information can also be examined to estimate its impact on proposed releases. Whooping cranes are being released in Florida to establish a non-migratory flock, and plans are underway for a release in the Prairie Provinces of Canada. We discuss possible implications of mortality on releases in these 2 locations, as well as captive mortality.

We thank M. T. Childress and J. L. Kodak for help with preparation and editing of this manuscript, J. M. Nicoich for her help in preparing the information used in this study and for presenting the paper at the workshop, and J. Lewis for his editorial comments. The necropsies were performed by the authors and veterinarians at Patuxent, the National Wildlife Health Center (NWHC), Madison, Wisconsin, and the Maryland Department of Agriculture, College Park, Maryland. These veterinarians were J. W. Carpenter, F. J. Dein, C. Driscoll, D. Green, C. Franson, P. Klein, and N. Thomas.

METHODS

Patuxent has reared whooping cranes by using various techniques. For the first 11 years (1967–77), whooping crane eggs were artificially incubated for 28 days and then transferred to a mechanical hatcher after pipping. Crane chicks were reared indoors by caretaking staff, then moved to outdoor pens as they grew older. Between 1978 and 1989, some whooping crane eggs were incubated by cochin chickens and sandhill cranes to improve embryo survival.

1Present address: Okefenokee National Wildlife Refuge, Route 2, Box 3330, Folkston, GA 31537, USA.

243
Hatching took place in artificial hatchers or under sandhill cranes. Chicks were reared as before, by caretakers or outdoors by surrogate sandhill crane parents. Beginning in 1990, whooping crane chicks were no longer reared by sandhill cranes to avoid cross-species imprinting. However, since 1990 some whooping crane chicks have been successfully reared by whooping crane pairs. Due to the low number of experienced whooping crane pairs, most chicks continue to be reared by staff, most recently by individuals wearing shrouds and masks to camouflage their human form. Sandhill cranes continue to act as surrogate incubators.

Adult whooping cranes are housed in outdoor pens of various sizes. Most of these are covered with nylon flight netting. Each pen has a gravity-flow poultry feeder and fully reared by whooping crane pairs. Due to the low number of experienced whooping crane pairs, most chicks continue to be reared by staff, most recently by individuals wearing shrouds and masks to camouflage their human form. Sandhill cranes continue to act as surrogate incubators.

Cause of death for each bird was determined from a combination of clinical findings, gross pathological changes observed at necropsy, and microbiological, parasitological, serological, and histopathological results. Gross necropsies were performed at Patuxent, although cases were occasionally referred to the NWHC. Radiographs were taken when trauma, foreign body, or skeletal deformity was suspected. Specimens from liver, lungs, kidneys, spleen, heart, brain, gastrointestinal tract, and other appropriate organs were obtained for microbiological testing. Samples were processed at Maryland Medical Laboratory in Baltimore, Maryland, Maryland State Board of Agriculture, Animal Health Department Laboratory in College Park, Maryland, or the NWHC.

We conducted parasitological analysis (both direct smear and flotation techniques) for isolation and examination of intestinal parasites. Parasites were preserved in 70% isopro- pyl alcohol for later identification.

Representative tissue samples collected at necropsy were preserved in 10% buffered formalin for histopathological study and sent to the NWHC or the Maryland State Board of Agriculture. When appropriate, additional tests were performed, including analysis of tissues for suspected chemical residues, and food and water for contaminants and toxins.

RESULTS

During the 14 years of this study, 103 whooping cranes died and were examined (Table 1). Nearly one-half of these cranes (46%) died from infectious or parasitic diseases. Fewer died from trauma (21%), anatomic abnormalities (17%), or from miscellaneous causes (12%). Cause of death was unknown in 5 cases. The causes of death were similar to those reported earlier (Carpenter and Derrickson 1982).

Whooping cranes were assigned to 3 age categories based on earlier studies: chicks (<2 months), juveniles (2-12 months), and adults (>12 months) (Carpenter and Derrickson 1982). Most deaths occurred in chicks <2 months of age (62%, n = 63) (Table 1). Ten chicks that died during the hatching process are included in the 63 deaths. Four of the chicks that died during hatching died from scoliosis, 3 from omphalitis, and 1 each from malpositioning and delayed hatching. No definitive cause of death was discovered in 1 death.

There were 15 juvenile (2-12 months) crane mortalities, and 25 subadult/adult (1 yr) mortalities between 1982 and 1995. Of these 25, 12 were males and 13 were females. The subadult/adult cranes ranged from 1 to 23 years of age at death.

The largest concentration of deaths occurred in spring and summer (April n = 6, May n = 39, June n = 17, and July n = 12, August n = 9) and was associated with chick hatching and early growth. The mortality rates in the autumn (September n = 7, October n = 3, and November n = 5) were elevated due to eastern equine encephalitis in 1984 (7 birds), and mycotoxins in 1987 (3 birds). Mortality was lowest during the cold months of the year (December n = 2, January n = 2, February n = 1, and March n = 0).

DISCUSSION

The primary causes of death for captive whooping cranes were infectious and parasitic diseases. Several of these diseases are of importance to the future of the captive propagation program and subsequent release efforts. Pneumonia and associated respiratory diseases were the most common infectious diseases (9%). Aspergillosis was also a common sequel to respiratory infections (7% of the cases). Infectious diseases caused 31.7% of deaths during 1966–81 (Carpenter and Derrickson 1982) and 32% of deaths in this study. However, there has been a shift in causative agents. Aspergillus spp. was not reported in the earlier study but caused 7% of deaths in this study. Factors contributing to this rise in incidence were improved diagnostic tests and the increased use of powerful antibiotics, which will control bacterial infections but may make the cranes more susceptible to secondary fungal infections. Although one-third of all infectious diseases were diagnosed as either pneumonia or aspergillosis, young chicks accounted for most of these deaths. Respiratory disease is an important mortality factor in any wild flock, but the extent of such mortality has been and will remain unknown. When there is whooping
Table 1. Causes of mortality in whooping cranes by age class at Patuxent Wildlife Research Center, Laurel, Maryland, 1982–95.

<table>
<thead>
<tr>
<th>Cause of mortality</th>
<th>Age class(^a) (n)</th>
<th>All ages</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chick</td>
<td>Juvenile</td>
</tr>
<tr>
<td>Trauma</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Aggression</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Spinal trauma</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Fractures</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Congenital abnormalities</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>Scoliosis</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Heart defects</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Leg deformities</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Infectious or parasitic disease</td>
<td>33</td>
<td>6</td>
</tr>
<tr>
<td>Pneumonia/respiratory disease</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>Omphalitis/rupture</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Disseminated visceral coccidiosis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Peritonitis/intestinal rupture</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>Acanthocephalans</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Eastern equine encephalitis</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aspergillos</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Miscellaneous infections</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Miscellaneous mortality factors</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Mycotoxins</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Nutritional deficiency</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Drug reaction</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Capture myopathy</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Malposition/delayed hatching</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>No diagnosis/open</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td>63</td>
<td>15</td>
</tr>
</tbody>
</table>

\(^a\) Chicks (<2 months); juveniles (≥2 and <12 months); adults (≥12 months).

Crane reproduction at the Florida release site, we should be able to document chick mortality and determine if respiratory disease is a major factor affecting chick survival.

During 1966–81, 4 deaths (10%) were attributed to disseminated visceral coccidia (DVC). Anti-coccidial medication was incorporated into the pelleted feed after identification of this disease in the late 1970’s. Between 1982 and 1995, only 1 whooping crane death resulted from DVC, a parent-reared chick <2 months of age that may not have received adequate amounts of medicated pelleted feed to prevent the infection. The 2 coccidia protozoans, *Eimeria gruis* and *E. riechenowi*, found in whooping cranes are found in both Florida and Canada, and adults can act as carriers. The disease is primarily of concern in chicks; both the enteric and visceral forms can be severe. Symptoms of the enteric form include diarrhea, weight loss, bloody stools, and electrolyte and fluid imbalances. Small white nodules (2–4 mm diameter) in various internal organs, including heart, liver, lungs, and kidney, and in the soft tissues of the mouth, are observed in the visceral form. Coccidiosis is unlikely to be a major problem in new wild populations; nevertheless, occasional chick mortality may be encountered. Although *Eimeria* organisms occur in Canada, harsh winters should limit their spread, especially to chicks.

Eastern equine encephalitis (EEE), an arbovirus spread by mosquitoes (primarily *Culiseta melanura*), can lead to lethargy, incoordination, and acute death in whooping cranes (Dein et al. 1986). Pathology is rather nonspecific: ascites, hepatomegaly, splenomegaly, and congestion or mild hemorrhage of the gastrointestinal tract. This agent does not cause disease in most native birds, but it does in whooping cranes and is potentially significant to the Florida release because the arbovirus and mosquitoes occur in the release area. At Patuxent, 5 of the 7 deaths occurred in adult females. Any
outbreak could have serious consequences for a reintroduction program, affecting young and older birds of breeding age. EEE has not been reported in the Prairie Provinces of Canada. However, a similar disease (western equine encephalitis) does occur, but this entity has never been documented as causing disease or death in whooping cranes.

Acanthocephalans and the associated category of peritonitis/intestinal rupture in chicks are important causes of mortality (14% total, Table 1). Acanthocephalans are small parasites (2–3 mm long) that lose torpor and detach from the intestinal wall when the host dies, causing difficulty in locating the actual parasite (Greiner and Ritchie 1994). The cause of death is usually attributed to intestinal rupture and peritonitis. The parasite life cycle is thought to involve invertebrate hosts that cranes are exposed to in the field. Most deaths occur in birds < 1 year of age. Adult acanthocephalans are controlled by benzimidazole anthelmintics such as oxfendazole, oxibendazole, and thiabendazole (Macwhirter 1994). Thiabendazole is used to control nematodes in whooping cranes in the non-breeding season, but its use in combination with monensin medicated feed during the breeding season should be investigated. There is nothing readily apparent in the captive situation that enhances the incidence of acanthocephalan infestation. On the contrary, if invertebrates do act as intermediate hosts, the pelleted diet reduces the number of invertebrates a crane may be capturing and eating. In 1966–81 only 4.8% of deaths were due to acanthocephalans (Carpenter and Derrickson 1982). However, adding the 2.4% of mortalities caused by peritonitis would give a mortality rate of 7.2%, still half of what we found in 1982–95. Because this is a disease found in young cranes, the increase may be associated with the increasing numbers of chicks being raised in recent years. Regardless of the cause of the increase, this mortality factor requires further research to learn more about control measures.

Trauma as observed in captivity would generally not occur in the wild. Trauma was the direct cause of 21% of whooping crane mortalities; however, nearly every instance was an artifact of captive propagation. Aggression was primarily of 2 types. The first, and most common, was 1 or more cranes attacking and killing a penmate. When a pair forms in a community pen (large pen enclosures housing multiple unpaired adult cranes), the 2 may become territorial and attack other cranes. Also, the adult crane may direct aggression against a chick. Inexperienced parents have been known to kill the chick they are raising. Chicks that wander and enter an occupied pen are at an even higher risk of being attacked. Trauma/aggression accounted for 6% of deaths in this study and 7.3% during 1966–81 (Carpenter and Derrickson 1982). This indicates trauma continues to be a factor in the captive propagation program that has not been resolved.

Fractures and associated complications, especially leg fractures, are often fatal (Olsen 1994). Fractures are usually associated with handling or other aspects of the captive situation. In wild cranes, collisions with powerlines and fences often result in fractures and death. Indeed, several whooping cranes received by Patuxent from the wild flock came as injured adult birds. However, leg fractures are not seen as often in the wild as in the captive flock. In the period 1966–81, there was only 1 fatality (2%) associated with a fracture (Carpenter and Derrickson 1982). We reported 6 cases, indicating a rise in this type of mortality, possibly associated with increased efforts to rear wild, non-imprinted cranes for release.

Spinal trauma occurs when a crane runs or flies into a fence, luxating or fracturing 1 or more vertebrae. If this injury is not immediately fatal, the paralyzed crane has to be euthanized. Recently, this type of mortality has increased as juveniles are raised to be wilder (to enhance survival after release), resulting in increased injury after birds are startled by occasional disturbances.

Mycotoxic-related mortality can occur in wild sandhill cranes (Windingstad et al. 1989) and is a potential threat to released whooping cranes. Mycotoxins are produced by certain aflatoxin or tricothecene fungi. Clinical signs are variable (Olsen et al. 1995) but can include inappetence, weight loss, oral ulceration, and paralysis of wings or neck. Clinical pathology may include elevated uric acid levels. There are no significant gross pathological lesions, and toxin detection can be difficult. Mycotoxin distribution is sporadic and unpredictable, and concentrations often are related to abnormal weather cycles, such as excess moisture. There are no reports of the disease in cranes in Canada, though it is likely that mycotoxins occur, especially where abundant waste grain is a common food source for cranes. Mycotoxins can be a potential problem for any release, but the sporadic nature of the toxin production indicates any problem would be stochastic.

Anatomic abnormalities resulting in fatalities at hatching and of young chicks occur in captivity and may occur in wild cranes. Scoliosis resulted in the death of 9 whooping cranes. We have seen very few instances of scoliosis in the 3 subspecies of sandhill cranes raised at Patuxent (Olsen and Gee 1997). Scoliosis was first diagnosed in 1983 and is a lethal congenital factor in the gene pool of the whooping crane. Therefore, scoliosis will be a likely mortality factor in other whooping crane populations.

Several types of leg deformities are common in the captive setting, including hock rotation associated with valgus deformity, slipped gastrocnemius tendons, and deformed feet. The exact congenital nature of these conditions is not well.
understood, may not affect the wild populations, and requires further investigation. Anatomic abnormalities resulted in 19.5% of mortalities from 1966 through 1981 (Carpenter and Derrickson 1982) and had a similar incidence (17%) during the years 1982–95 of this study.

Several diseases known to occur in the Canadian Prairie Provinces (Wobeser 1985) are not seen in the captive flock. Avian choler caused by the bacterial organism *Pasteurella multocida* occurs in lesser snow (*Chen hyperborea*) and Ross’s geese (*C. rossii*) in the Kindersley area of Saskatchewan during April and early May. Other waterfowl and sandhill cranes occasionally succumb to avian choler. The disease is peracute; most birds do not appear clinically ill before death. Some birds may exhibit symptoms such as weakness, depression, inability to fly, and convulsions prior to death. Avian cholercauses scattered hemorrhagic lesions on the viscera, and white or yellow 1- to 2-mm-diameter necrotic foci on the liver. Diagnosis is confirmed by bacterial culture. The outbreaks in Saskatchewan are not as explosive as others seen in waterfowl wintering areas; but because the outbreaks occur in spring with occasional sandhill crane mortality, released whooping cranes may encounter some losses to avian choler.

Salmonellosis caused by bacteria of the genus *Salmonella* (most commonly *S. typhimurium*) occurs in the Canadian prairies. House sparrows (*Passer domesticus*) around Saskatoon, Saskatchewan, have died from this disease (Wobeser 1985). Generally, sick birds appear depressed, are dehydrated, and have diarrhea. A postmortem examination reveals caseous degeneration of the crop, liver, spleen, kidney, or brain. Yolk sac or intestinal inflammation is seen in young birds. Culture and strain identification is important because virulence varies with strain. Sandhill cranes in Mississippi have been affected by salmonellosis (Langenberg et al. 1994). An occasional non-virulent strain has been isolated individuals.

Avian tuberculosis, a chronic, debilitating disease caused by the bacteria *Mycobacterium avium*, has been reported in wild birds in Saskatchewan (Wobeser 1985). A sick bird appears weak and has an elevated white blood cell count with elevated monocyte levels. White caseous nodules are found in the liver, spleen, and gastrointestinal tract on postmortem examination. The disease occurs in whooping cranes in Idaho and Texas (Snyder and Richard 1994) and therefore would be of concern for a release program. However, avian tuberculosis concerns do not preclude the use of the present release sites in Florida or future releases in Canada.

Newcastle disease is caused by a paramyxovirus and there are 4 strains of the virus. The most virulent (viscero-tropic velogenic Newcastle disease strain [VVND]) causes high morbidity and mortality in poultry, psittacines, raptors, and waterfowl. There are no pathognomonic signs, and young birds develop a peracute disease resulting in high mortality. Symptoms include yellowish or hemorrhagic diarrhea, coughing, sneezing, dyspnea, incoordination, or ataxia. Gross pathologic findings include enlarged liver and spleen, airsacculitis, and generalized inflammatory response of visceral organs. In the past, North America had been considered free of VVND, but in 1990 about 9,000 birds (7,000 double-crested cormorants [*Phalacrocorax auritus*], 2,000 gulls [*Larus spp.*], and a small number of white pelicans [*Pelecanus erythrorhynchos]*) died in the Canadian Prairie Provinces (Wobeser et al. 1993). This disease must be monitored because any occurrence in the area of the proposed whooping crane reintroduction would pose a threat to the released birds.

In summary, some mortality factors seen in the captive flock at Patuxent, such as infectious and parasitic diseases and congenital abnormalities, will have similar impacts on reintroduction programs. Other conditions are artifacts of the captive propagation situation. Some mortality factors in the captive breeding flock require further investigation to facilitate better understanding and reduction of mortality.

**LITERATURE CITED**


Captive Whooping Crane Mortality, Olsen et al.


