Original Research

The Causes of Eagle Mortality in Saskatchewan, 1992-2012

Steven J. SCOTT1 and Trent K. BOLLINGER1

Abstract

Diagnostic records of 227 bald eagles (Haliaeetus leucocephalus) and 78 golden eagles (Aquila chrysaetos) collected in Saskatchewan, during 1992 to 2012, were reviewed to identify common causes of mortality. The most frequent cause of death in bald eagles was toxicosis (53%), followed by trauma (22%), electrocution (6%), infectious disease (4%), malnutrition (3%), and idiopathic disease (2%). Trauma and toxicosis were equally responsible for the majority of golden eagle mortalities (28%). Overall, 77% of toxicosis cases were attributed to organophosphate and carbamate pesticides, and 22% were attributed to lead. Automobile collision was responsible for the majority (18%) of trauma-related mortalities in which a cause was known. Pasteurella multocida and Aspergillus fumigatus were the most common cause of infectious disease in bald eagles and golden eagles, respectively. One golden eagle infected with A. fumigatus was also diagnosed with osteosarcoma. This study shows that anthropogenic factors are the most significant cause of eagle mortality in Saskatchewan, and that organophosphate and carbamate insecticides pose a significant risk to resident and migrant eagle populations.

Key Words: Aquila chrysaetos, Bald eagle, Golden eagle, Haliaeetus leucocephalus, Saskatchewan, Insecticides.

INTRODUCTION

Bald eagles (Haliaeetus leucocephalus) and golden eagles (Aquila chrysaetos) are sensitive to human activities. This was demonstrated in the early to mid-1900s when bald eagles became endangered in the United States due to a combination of human-related factors, including persistent use of pesticides, illegal shooting, habitat fragmentation and human disturbance (Fraser 1985; Richardson and Miller 1997). Subsequent banning of dichlorodiphenyltrichloroethane (DDT) allowed eagle populations to recover in the United States, and ensured that eagle populations in Canada would continue to thrive. The Committee on the Status of Endangered Wildlife in Canada (COSEWIC) currently reports that the status of bald and golden eagles in Canada is “not at risk”
Saskatchewan is an important breeding and migratory area for bald eagles and golden eagles (Gerard et al. 1978). Their presence has not gone unnoticed as both species are highly valued by bird enthusiasts, the general public, and First Nations people. As a result, sick or dead eagles are often submitted to the Canadian Wildlife Health Cooperative (CWHC) Western/Northern for postmortem examination.

Several studies have summarized the causes of morbidity and mortality in free-ranging raptorial birds in North America; however, these were mainly restricted to birds from the United States and few have focused specifically on eagles (Coon et al. 1970; Morishita et al. 1998; Deem et al. 1998; Wendell et al. 2002; Driscoll et al. 2004; Harris and Sleeman 2007). The purpose of this retrospective study was to determine the most common causes of mortality in eagles from Saskatchewan examined over a 21-year period (1992-2012).

### MATERIALS AND METHODS

Complete diagnostic records of eagles collected in Saskatchewan between 1992 and 2012 were obtained from the CWHC national database (www.ccwhc.ca). Data collected from each case included the case identification number, location where the eagle was found, date of collection, species, age class, sex, postmortem and histologic findings, diagnostic test results, and final diagnosis. Fifty-four cases were not included in this study because carcasses that were confirmed poisoned. A diagnosis of suspicion of toxicosis was based on 20–49% reduction in brain cholinesterase activity and/or demonstration of the insecticide residues in crop contents (Fairbrother 1996). Additionally, these cases included non-tested eagles that were found near dead avian or mammalian carcasses that were confirmed poisoned. A diagnosis of suspicion of toxicosis was based on 20–49% reduction in brain cholinesterase in one or more eagles in an area. Birds assigned to these sub-categories of confirmed and suspicious insecticide toxicosis were included within the toxicosis category as there were no other gross or histological lesions to suggest an alternative etiology. Diagnosis of lead toxicosis was based on liver lead levels ≥5ppm (wet weight), kidney lead levels of ≥10ppm (wet weight), or blood lead levels of ≥3ppm, together with compatible gross and/or histologic lesions (i.e., emaciation, myocardial necrosis and fibrosis, and proximal tubular necrosis in the kidneys) (Locke and Thomas 1996). Strychnine toxicity was based on demonstration of the toxin in crop contents, and mercury toxicity was based on liver mercury levels ≥20 ppm (wet weight) (Heinz 1996).

Trauma was sub-categorized into automobile collision, gunshot, traps, predation/scavenging, and unknown origin. Automobile collision was defined as an eagle with soft tissue injury and/or fractured bones, which was found on, or next to, a road. Gunshot was defined as an eagle with radiographic evidence of bullet fragments, or in which bullets were identified on postmortem examination. Entrapment included eagles with soft-tissue injury resulting from a snare or trap, and scavenging/predation included eagles that died from complications associated with feeding on prey animals. Eagles that had soft tissue damage and/or fractured bones for which the cause could not be determined were classified as trauma of unknown cause.

The case definition for electrocution was an eagle found beneath, or next to, a power line, and which had lesions compatible with thermal injury (i.e., singed feathers). Infectious disease was diagnosed by demonstrating pathogenic bacteria, viruses, or fungi using microbiological, immunological or molecular techniques, and parasites were identified on postmortem and histopathologic examination. Malnutrition was defined as an emaciated eagle with no evidence of other categories of underlying disease. Idiopathic disease was diagnosed when the origin of a condition was uncertain. When no cause of death had been determined, the bird was placed in the category of unknown cause. Typically, such birds were in good body condition and had no evidence of underlying disease or trauma.

A chi-square test was used to analyze age class and sex differences in mortality categories. Differences were deemed to be significant at *P*<0.05.

### RESULTS

Records from 305 eagles collected in Saskatchewan were reviewed: 227 bald eagles and 78 golden eagles (Table 1, Figure 1). On average, 18 eagles were received annually (Figure 2), and submissions were most frequent during spring (April-May) and late fall (October–December).

Toxicosis was the most common cause of mortality in bald eagles followed by trauma, electrocution, infectious disease, malnutrition, and death from unknown cause (Table 2). The most frequent cause of toxicosis was OP/CB insecticides, which occurred most
Table 1. Age and sex composition of bald eagles (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*) submitted for necropsy from Saskatchewan from 1992 to 2012.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Bald Eagles&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Golden Eagles&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adult</td>
<td>119 (52.4%)</td>
<td>38 (48.7%)</td>
</tr>
<tr>
<td>Juvenile</td>
<td>93 (41.0%)</td>
<td>29 (37.2%)</td>
</tr>
<tr>
<td>Not available</td>
<td>15 (6.6%)</td>
<td>11 (14.1%)</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>108 (47.6%)</td>
<td>30 (38.5%)</td>
</tr>
<tr>
<td>Female</td>
<td>82 (36.1%)</td>
<td>32 (41.0%)</td>
</tr>
<tr>
<td>Total available</td>
<td>37 (16.3%)</td>
<td>16 (20.5%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>227</td>
<td>78</td>
</tr>
</tbody>
</table>

<sup>a</sup> Number of bald eagle and golden eagle mortalities (% for each species of eagle).

commonly in April and November. The diagnosis was confirmed in 72% of these cases. Carbofuran was identified in 10 eagles, a combination of Carbofuran and Terbufos was identified in 1 eagle, and 1 eagle was found near a pig carcass in which a farmer confessed to lacing with Furadan. The diagnosis in 28% of cases was suspicious. Lead toxicity was the second most frequent cause of toxicity and cases were most common in November and December. Strychnine toxicity was diagnosed in 2 bald eagles that recently had ingested viscera from Richardson ground squirrels (*Urocitellus richardsonii*); one of these eagles ultimately died from colliding with an automobile, but was still classified in the toxicity category. Mercury toxicity was diagnosed in 1 bald eagle that was found dead and emaciated with elevated mercury levels in the liver (41.2ppm). The majority of trauma-related mortalities in bald eagles resulted from traumatic events of unknown cause, followed by gunshot. One bald eagle was discovered captured in a coyote (*Canis latrans*) snare, which resulted in gangrenous necrosis of the foot.

The most common infectious disease in bald eagles was due to *Pasteurella multocida* infection (avian cholera); 3 of the 4 cases occurred in November. Two cases of West Nile virus (WNV) were diagnosed during the summer months. Fungal pneumonia due to *Aspergillus fumigatus*, encephalitis due to an unidentified spirochaete bacterium, and avian herpesvirus occurred as single cases. Idiopathic disease included peritonitis secondary to gastrointestinal perforation (*n=2*), esophageal perforation (*n=1*), and mesenteric torsion (*n=1*).

In golden eagles, trauma and toxicosis were the most frequent causes of mortality (Table 2). The majority of trauma cases were attributed to unknown causes and automobile collision. In 2 instances, golden eagles died from complications of predation or scavenging on porcupine (*Erethizon dorsatum*); in 1 case, porcupine quills pierced several vital organs in a juvenile eagle and in a separate case, occlusion of the oropharynx with quills resulted in starvation of an adult eagle. One golden eagle was found with its leg caught in a coyote snare, while 2 presented with lesions on the distal legs that were suspected to represent snare injuries (i.e., skin laceration and swelling of the tarsometatarsus with associated loss of deep pain reflex and apparent inability to use the hind feet). Organophosphate and carbamate pesticides were the most frequently diagnosed toxicosis; 46% and 54% of these diagnoses were confirmed and suspicious, respectively. The remaining toxicosis cases resulted from lead.

Fungal pneumonia caused by *Aspergillus fumigatus* was the most common infectious disease in golden eagles. In one case, an adult eagle was concurrently diagnosed with osteosarcoma of the shoulder. Severe parasitism was implicated as the cause of death in 3 cases. In 1 case, an eagle’s emaciated state was attributed to a heavy burden of intestinal *coccidia* and a severe infection with blood-borne protozoa (suspect *Haemoproteus* spp.). Severe myocarditis caused by an unidentified *Sarcocystis* spp., and severe intestinal trematodiasis caused by an unidentified trematode, comprised the remainder of the fatal parasitic diseases. There were single cases of West Nile virus, and of avian pox complicated by secondary encephalitis due to *Candida albicans*. One record of chronic encephalopathy in an adult eagle, which was negative for WNV on immunohistochemistry, was categorized as a death from unknown cause.
Figure 1. Location of eagles that were collected and submitted from necropsy in Saskatchewan from 1992 to 2012.
Figure 2. Number of bald eagle (*Haliaeetus leucocephalus*) and golden eagle (*Aquila chrysaetos*) carcasses from Saskatchewan that were submitted to the Canadian Wildlife Health Cooperative, 1992-2012.

Table 2. Causes of morbidity and mortality in bald eagles (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*) in Saskatchewan from 1992 to 2012.

<table>
<thead>
<tr>
<th>Cause of Mortality</th>
<th>Bald Eagles*</th>
<th>Golden Eagles*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Toxicosis</td>
<td>121 (53.3%)</td>
<td>23 (29.5%)</td>
</tr>
<tr>
<td>Organophosphate/carbamates</td>
<td>98 (43.2%)</td>
<td>13 (16.6%)</td>
</tr>
<tr>
<td>Lead</td>
<td>20 (8.8%)</td>
<td>10 (12.8%)</td>
</tr>
<tr>
<td>Strychnine</td>
<td>2 (0.9%)</td>
<td></td>
</tr>
<tr>
<td>Mercury</td>
<td>1 (0.4%)</td>
<td></td>
</tr>
<tr>
<td>Trauma</td>
<td>50 (22.2%)</td>
<td>22 (28.2%)</td>
</tr>
<tr>
<td>Unknown</td>
<td>33 (14.5%)</td>
<td>8 (10.2%)</td>
</tr>
<tr>
<td>Automobile collision</td>
<td>7 (3.1%)</td>
<td>6 (7.7%)</td>
</tr>
<tr>
<td>Gunshot</td>
<td>9 (3.9%)</td>
<td>3 (3.8%)</td>
</tr>
<tr>
<td>Traps</td>
<td>1 (0.4%)</td>
<td>3 (3.8%)</td>
</tr>
<tr>
<td>Predation/scavenging</td>
<td></td>
<td>2 (2.6%)</td>
</tr>
<tr>
<td>Electrocution</td>
<td>14 (6.2%)</td>
<td>13 (16.7%)</td>
</tr>
<tr>
<td>Infectious Disease</td>
<td>9 (3.9%)</td>
<td>7 (8.9%)</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>7 (3.0%)</td>
<td>6 (7.7%)</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>4 (1.8%)</td>
<td>1 (1.3%)</td>
</tr>
<tr>
<td>Unknown</td>
<td>22 (9.6%)</td>
<td>6 (7.7%)</td>
</tr>
<tr>
<td>Total</td>
<td>227</td>
<td>78</td>
</tr>
</tbody>
</table>

* Number of bald eagle and golden eagle mortalities (% for each species of eagle).
When bald eagles and golden eagles were combined, a greater proportion of OP/CB toxicosis cases involved adult eagles ($P=0.0002$) and male eagles ($P=0.01$) relative to the sex distribution in the sample of birds.

**DISCUSSION**

The majority of eagle mortalities in this study were due to human-related activity (66%), which is consistent with similar studies of mortality in eagles and other raptorial birds in North America (Coon *et al.* 1970; Deem *et al.* 1988; Driscoll *et al.* 2004; Harris and Sleeman 2007). The high prevalence of human-associated mortality reflects the fact that the majority of the eagle carcasses were collected in the southern more populated areas of the province (Figure 1).

The biggest difference between our findings and those of previously published reports is that trauma was not the most common reason for Saskatchewan eagles to be submitted to the CWHC Western/Northern. Instead, toxicosis accounted for 52% of eagles examined in Saskatchewan, which is considerably higher than what is reported in previous studies in the United States, in which prevalence of toxicoses ranged from 0-10.5% in eagles and 0-40% in all raptor species (Coon *et al.* 1970; Deem *et al.* 1988; Morishita *et al.* 1998; Driscoll *et al.* 2004; Harris and Sleeman 2007).

The majority of toxicosis cases were attributed to OP/CB insecticides, which is similar to other studies in the United States (Deem *et al.* 1988; Morishita *et al.* 1998; Wendell *et al.* 2002; Harris and Sleeman 2007). These insecticides are a well-known cause of eagle morbidity and mortality in western Canada, and in Saskatchewan many of these cases represent secondary poisoning from scavenging on coyotes that have been illegally poisoned (Wobeser *et al.* 2004). We are not aware of any reports that compare OP/CB toxicosis between the 2 eagle species, but we suspect that bald eagles were more commonly diagnosed with OP/CB toxicosis due to their higher population numbers, and the increased likelihood of multiple individuals being involved in a poisoning incidence, compared to golden eagles in Saskatchewan. The seasonal distribution of OP/CB toxicosis cases has been recognized and corresponds to the migratory pattern of eagles in the province (Wobeser *et al.* 2004). The differences in age class and sex suggest that adult eagles and male eagles are more susceptible to OP/CB toxicosis; however, we are unaware of any sex- or age-related differences in the inherent susceptibility to OP/CB pesticides or in feeding habits.

Although demonstration of insect residues was not possible in most OP/CB toxicosis cases due to the high cost of testing, >50% reduction in brain AChE activity is generally considered diagnostic for such poisoning (Fairbrother 1996). Several published studies excluded our category of suspect OP/CB toxicosis, and instead classified them as unknown cause of death (Deem *et al.* 1988; Morishita *et al.* 1998; Wendell *et al.* 2002; Harris and Sleeman 2007). We included these cases because the circumstances of death were typical of such poisoning, no other causes of death could be detected and reactivation of carbamate-inhibited cholinesterase in carbamate poisonings is a well-recognized phenomenon that complicates diagnostic interpretation of AChE levels (Fairbrother 1996). Although this may overestimate the prevalence of mortality directly caused by OP/CB pesticides, 20-50% reduction in brain AChE is consistent with exposure to a cholinesterase-inhibiting agent, which in turn can predispose birds to other causes of mortality (i.e., trauma) (Fairbrother 1996).

The prevalence of lead toxicosis in eagles was consistent with previous reports from Canada and the United States (Wayland and Bollinger 1999; Miller *et al.* 2001). A greater proportion of golden eagles died from lead toxicosis compared to bald eagles. This dissimilarity can likely be attributed to differences in diet between the two species. Bald eagles commonly feed on waterfowl, which are not as likely to have lead embedded in their tissues due the ban placed on the use of lead shot for hunting game birds in 1999 (Wayland and Bollinger 1999). The golden eagles’ diet is dominated by small and large game mammals, which would be more likely to contain lead shot (Brown and Watson 1964).

The prevalence of lead toxicosis in bald eagles was highest between 1998-2001 (30-50%), and ranged between 10-20% in the years afterwards. Lead shot was found in the stomach in 1 bald eagle, but the source of lead toxicosis was not determined in the remainder of these cases.

Strychnine poisoning is not reported frequently in raptors, but its prevalence may be underestimated since sub-lethal concentrations could potentially predispose birds to other causes of mortality (i.e., trauma), as was the case for 1 eagle in our study. The presence of a Richardson’s ground squirrel in the stomach of 1 of these eagles is suggestive of secondary poisoning as farmers in southern Saskatchewan have been reported to use large quantities of 0.4% strychnine to poison Richardson’s ground squirrels (Proulx 2011). Our findings along with Proulx (2011) indicate that strychnine does pose a risk to non-target species.

Eagle mortality associated with mercury toxicity was not common in this study, which is similar to what was reported in a retrospective study of morbidity and mortality of raptors in Florida (Deem 1998). It is important to note that mercury poisoning may be underestimated in this study since testing mercury levels is not routinely performed on submissions. In addition, mercury levels are difficult to interpret since the adverse health effects and pathologic lesions are poorly described in eagles. Axonal degeneration has been reported in mallards that were experimentally poisoned with methylmercury chloride (Pass *et al.* 1975). These lesions were not identified in the bald eagle in our study, but there was moderate freezing artifact and autolysis in this specimen that could have masked subtle lesions in the central
nervous system. Trauma was the most common cause of mortality in golden eagles, which is consistent with previous findings in Maryland (Driscoll et al. 2004). In cases in which the cause of trauma was known, automobile collision and firearms were the greatest contributors in both species, which is consistent with findings from 2 other studies in the United States (Coon et al. 1970; Driscoll et al. 2004). Non-target snare captures have been recently identified as a cause of mortality in bald eagles and golden eagles in Canada (Proulx et al. 2015). Although not statistically significant, prevalence of electrocution was higher in female eagles compared to males, and juveniles compared to adults. Sexual dimorphism is most likely responsible for the sex difference in electrocution cases, since female eagles are typically larger than males, and thus more likely to span electrical components (Lehman et al. 2007). Juveniles are reported as predisposed to electrocution because they are less experienced with flight and more prone to accidents when landing or taking-off from a power line (Lehman et al. 2007). Other studies have demonstrated a seasonal variation in electrocution cases; the peak occurrence in October in our study may represent increased populations of post-fledging eagles on their first migration southward (Harness and Wilson 2001; Lehman et al. 2007).

Infectious disease was not a significant contributor to mortality in eagles, which is similar to findings in other raptor species (Coon et al. 1970; Deem et al. 1988; Morishita et al. 1998; Wendell et al. 2002; Driscoll et al. 2004; Harris and Sleeman 2007). In this study, avian cholera was the most frequently diagnosed infectious disease and carcasses were typically found in association with avian cholera die-offs in geese as suggested by Morishita et al. (1996). All cases of WNV contained characteristic lesions consisting of nonsuppurative encephalitis (Wünschmann et al. 2014), and were diagnosed in the summer months coinciding with the time of year when mosquito (Culex spp.) populations are at their highest (Rappole et al. 2000). Eagles are remarkably susceptible to West Nile virus, which was recently demonstrated in a 2013 mortality event in Great Salt Lake, Utah, that involved more than 40 bald eagles (Ipl et al. 2014).

We are not aware of any previous reports of osteosarcoma in golden eagles, and the relationship between neoplasia and fatal aspergillosis was undetermined in the case described in this study. The identity of several parasites causing fatal disease in eagles in this study was not known at the time of diagnosis, but some recent publications have identified the causal agents in very similar cases. For example, Sarcozystis falculata has been recognized as a cause of fatal myocarditis in free ranging bald and golden eagles (Wünschmann et al. 2010) and the trematode Neodiplometomum reflexum was the cause of occlusive enteritis in a Japanese golden eagle (El-Dakhly et al. 2012). The golden eagle that died from avian pox complicated with C. ablicans was previously published as a case report (Shrubsole-Cockwill et al. 2010).

This is the first study on all causes of eagle mortality in Canada. Our findings indicate that human-related activities are the greatest contributor to eagle mortality in Saskatchewan and that OP/CB pesticides are a significant threat to resident and migrant eagles. In light of these findings, there is a need for increased surveillance for organophosphate and carbamate insecticides use in Saskatchewan, and possibly stricter fines for individuals who are prosecuted for illegal poisoning of wildlife species. Public outreach programs to increase awareness of the issue of poisoning of non-target species is also recommended.

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LITERATURE CITED


Harness, R., and K. Wilson. 2001. Electric-utility structures...

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Steven Scott is a veterinary pathologist with the Western and Northern region of the Canadian Wildlife Health Cooperative in Saskatoon, Saskatchewan. He received his Doctor of Veterinary Medicine from the Atlantic Veterinary College in 2011, after which he completed a Masters of Veterinary Science at the Western College of Veterinary Medicine in 2013. In 2014, Dr. Scott became a diplomate with the American College of Veterinary Pathologists. He has previously researched infectious causes of fish mortality in Saskatchewan
lakes and *Baylisascaris procyonis* in raccoons in New Brunswick.

Trent Bollinger is a wildlife veterinarian who specializes in the pathology and epidemiology of diseases of wildlife and fish. He is a Professor in the Department of Veterinary Pathology and the Director of the Western and Northern Region of the Canadian Wildlife Health Cooperative, a partnership between the five Canadian Veterinary Colleges; Wildlife, Agriculture and Public Health Departments across Canada; and NGOs.

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