Canadian Cooperative Wildlife Health Centre

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Centre Canadien Coopératif de la Santé de la Faune

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In this issue:

News from the CCWHC

Type E Botulism in Fish-Eating Birds on Lake Huron and Lake Erie 1998-2003

Chronic Wasting Disease in free-ranging cervids in western Canada

Disease Updates

Atlantic Region

Esophageal Lesions in Crows

Surplus killing of roseate terns and common terns by a mink

Quebec Region

West Nile Virus infection in Québec Raptors : 2003 season

Ontario Region

Mortality in Great Black-Backed Gulls near Presqu'ile

Type E Botulism Update

West Nile Virus Update

Adenovirus Encephalitis in a Wild Fox

Western/Northern Region

Newcastle Disease Virus in Double-crested Cormorants in Alberta and Saskatchewan in 2003

West Nile Virus in Sage Grouse

The Research Group for Arctic Parasitology (RGAP), November 2003

Death of a Grizzly Bear caused by capture and handling

Announcements

Diagnosing Disease in Wild Animals. February 25-27, 2004, Saskatoon, SK

Feature Articles

Type E Botulism in Fish-Eating Birds on Lake Huron and Lake Erie: 1998-2003

Type E botulism is a form of food poisoning caused by ingestion of one of the seven different toxins produced by different strains of the bacterium *Clostridium botulinum*. This bacterium exists in two forms: a durable spore that persists in the environment, and a growing form into which the spore develops when environmental conditions become favourable for growth. Suitable conditions for growth include a complete lack of oxygen and a rich organic source of nutrients. Decaying carcasses meet both these requirements, and in carcasses the organism proliferates and produces a toxin. The toxin blocks nerve impulse transmission and causes paralysis of muscle. Affected animals may die from paralysis of breathing muscles, and water birds may drown because they cannot hold their heads out of the water.

Each type of botulism is associated with different environmental circumstances and different animal hosts. For example, Type C botulism is best known because of large-scale die-offs of waterfowl on alkaline prairie lakes. Type E botulism is strongly associated with lake and marine environments. It is a regular source of human food poisoning when fish or marine mammals are eaten without thorough cooking, and particularly when prepared by smoking, air drying or fermentation. Any of these methods can create ideal conditions for bacterial growth and toxin production. Numerous local outbreaks of Type E botulism have occurred in Arctic communities where this type of food preparation is common, and there have been cases in temperate North America and Europe involving commercially prepared smoked or air-dried fish.

In North American wildlife, Type E botulism primarily is recognized as a disease of water birds on the Great Lakes. In the early 1960's on Lake Michigan and western Lake Huron, there was repeated annual mortality of thousands of common loons and smaller scale events involving various species of gulls and grebes. The disease disappeared for a dozen years, then resurfaced on Lake Michigan from 1981-83, with annual mortalities of loons numbering in the thousands.

The first diagnosis of botulism in loons in Canada was made on Lake Huron in 1998. That year, a few dozen loons were reported dead along the beaches on and near Pinery Provincial Park on southeastern Lake Huron during late October and November. In late October 1999, as many as 7,000 birds died in the same area. Common loons and red-breasted mergansers were the species most frequently affected, but numerous other species, including grebes, gulls and diving ducks also were involved. That same fall, botulism occurred in the western basin of Lake Erie, in the area between Point Pelee National Park and Rondeau Provincial Park. Thousands of birds of many species were found dead along the beaches and shorelines; red-breasted mergansers were the most commonly affected species.

Since 1999, Type E botulism has become an annual event on the lower Great Lakes (Erie and Ontario). On Lake Huron, none was detected from 1999 until the fall of 2002, when hundreds of birds of multiple species died along the shore near Kincardine.

The centre of the current epidemic of Type E botulism is Lake Erie. Since 1999, there has been a repeated pattern of annual occurrence. In late July or early August, there are small-scale die-offs of gulls, and, less commonly, of other species such as cormorants and terns, which share colony sites. Then migrating shorebirds, such as sanderlings, plovers and sandpipers, are affected.

Large-scale die-offs begin in late September, peak in late October and November, and involve primarily predatory fish-eating species: common loons, mergansers and grebes. More recently, long-tailed ducks (oldsquaw) have been involved in these events. Geographically, the centre of botulism activity has shifted from western to eastern Lake Erie, although sporadic events still occur in all parts of the lake. In fall 2002, there were suspected cases in gulls on the Niagara River, and in long-tailed ducks on Lake Ontario (Table 1).

Confirmation of Type E botulism involves demonstrating the presence of ingested Type E toxin in the bird's body. The ideal sample for testing is a blood sample taken from a live, sick bird. Serum from the blood is injected into a mouse; if there is botulinum toxin present, the mouse will soon die with the typical signs of botulism. The strain of toxin can then be identified by taking a group of mice, protecting individual mice, each with a different botulism anti-toxin, and then injecting these mice with the bird's serum. The mouse protected with type E anti-toxin will survive and the other mice will die if Type E botulism is in the bird's blood.

Mouse inoculation has confirmed Type E botulism in a live, sick loon captured during the 1999 event and in numerous gulls from Lake Erie. However, in most instances, live, sick birds are not available, and the test must be done with post-mortem material. In freshly dead birds, the test can be performed using liver or clotted blood found inside the heart. Once putrefaction of the carcass begins, tests for toxins are difficult to interpret. Type E *Clostridium botulinum* grows in putrefying carcasses and produces toxin, so a positive test may only mean that putrefaction has begun. Conversely, not all birds that die of botulism will test positive. In many of these birds, the toxin may already have exerted its effect, and be bound up in tissue and not present in liver or serum in sufficient quantity to produce a positive test. The mouse inoculation test for botulism is labourious, expensive and imperfect, but remains, at the present time, the best test available.

Because it is possible to have false positive tests on carcasses, it is necessary to exclude other possible causes of death of birds found dead during one of these mass die-offs. Complete post mortem examinations and a wide range of tests have been done on birds from these epidemics on the Great Lakes. Enough of these birds have been tested for enough other possible causes of death to satisfy most observers that it is botulism, and not some other disease or toxin, that is the cause of the epidemic.

The source of type E toxin for these birds remains an open question. In the case of the gulls, it is reasonable to believe that they acquire the toxin through scavenging decomposing carcasses containing toxin. Die-offs of fish, and also of mudpuppies, occur every summer on Lake Erie and likely provide a rich source of botulinum toxin for scavenging birds. Shorebirds are likely intoxicated through consumption of insects that have absorbed toxin from feeding on carcasses.

The source of toxin for predatory fish-eating birds presents more of a conundrum: birds such as loons are not known to scavenge carcasses. However, it is known that they will pursue and consume injured fish that swim abnormally, and it is quite likely that they will also eat sick and weak fish. In an attempt to determine the source of toxin for these birds, gizzards were collected from birds dying during these die-offs, and the contents were identified. Results are summarized in Tables 2 and 3. Gobies constituted a large proportion of fish that were identifiable to species.

A variety of other items, including zebra and quagga mussels, were found in the gizzards of the other species of affected birds.

These findings have led to speculation that invasive species are playing an important role in the current Type E botulism epidemic. Round Gobies are fish native to the Caspian and Black Seas that were inadvertently introduced into the Great Lakes, likely through the discharge of ship's ballast water. They were first detected in Lake St. Clair in 1990, have since dispersed widely in the lower Great Lakes, and now have reached the north channel of Lake Huron and the eastern end of Lake Ontario. Among their primary foods are zebra and quagga mussels, also alien invasive species in the Great Lakes. Zebra or quagga mussels also have been identified in the gizzard contents of dead birds, particularly the diving ducks. In recent years, there have been reports of extensive die-offs of mussels, leading to speculation that decomposing mussels have provided the nutrient substrate for the production of Type E toxin, which subsequently has moved up the food chain to birds directly, as in the case of ducks, or indirectly through fish, particularly gobies, in the case of fish-eating birds.

At the present time, these connections are speculative. Research is needed to identify the components of this food web, the production of botulinum toxin within these components, and its subsequent movement through trophic levels, terminating in the annual toll of thousands of bird carcasses littering the shores of Lake Huron and Lake Erie. There also is need to assess the effects of this level of annual mortality on the populations of some of the affected species, particularly the Common Loon. Our knowledge of this epidemic has been gained through a cooperative effort among many individuals and agencies. Jeff Robinson of the Canadian Wildlife Service has taken the initiative and done most of the work of reporting and collecting of carcasses, and has received assistance from many agencies, including Parks Ontario (Pinery and Rondeau), Parks Canada, the Ontario Ministry of Natural Resources, the Canadian Wildlife Service, and many members of the public. Post-mortem examination of the carcasses was done by the Canadian Cooperative Wildlife Health Centre in Guelph. Mouse inoculation tests were done at the Animal Health Laboratory of the University of Guelph. The identification of gizzard contents of the birds was done by the Fisheries Research Laboratory of the Ontario Ministry of Natural Resources in Wheatley. (Doug Campbell, CCWHC, Ontario/Nunavut Region).

Table 1. Type E Botulism Bird Species Affected

Confirmed	Suspected
Common Loon	Sanderling
Red Breasted Merganser	Sandpipers
Ring Billed Gull	Pied Bill Grebe
Herring Gull	Horned Grebe
Great Black Backed Gull	Greater Scaup
Bonaparte's Gull	Caspian Tern
Double Crested Cormorant	Great Blue Heron
Red Necked Grebe	Long Tailed Duck

American Golden Plover	

Table 2. Items Identified in Gizzards of Birds Dying During Type E Botulism Events

Species	Fish	Zebra/Quagga Mussels	Other	Empty
Common Loon	70	9	17	24
Mergansers	11	2	1	1
Grebes	7	1	2	3
Long Tailed Ducks	9	24	3	1
Gulls	15	2	16	0

Table 3. Fish Identified in Bird Gizzards

Species	Number of Gizzards
Gobies	34
Smelt/Alewives	8
Perch	7
Gizzard Shad	2
Salmonids	2
Sheepshead	2
Cyprinids	9
Shiners	2
Unidentified Fish	53

Figure 1. Loons dead of Type E botulism, Lake Erie, November 2002.

image pending

Update on Chronic Wasting Disease in Free-Ranging Cervids in Western Canada

Chronic wasting disease (CWD) is a fatal brain disease of mule deer, white-tailed deer and elk. CWD is caused by an abnormal protein molecule called a prion and is a disease closely related to scrapie in sheep, bovine spongiform encephalopathy (BSE) in cattle and Creutzfeld-Jakobs disease (CJD) in humans. The disease is thought to have been introduced into Saskatchewan farmed elk in the late 1980s via affected elk imported from the United States, but it was not recognized in farmed elk until 1996. Shortly after its detection on game farms, wildlife agencies in the prairie provinces began surveillance programs to determine if the disease was present in free-ranging deer and elk. From 1996 up to August of 2003, Manitoba has tested approximately 2,500 wild deer and elk, Alberta has tested approximately 3,000 and Saskatchewan has tested

approximately 11,000 (Table 1). CWD has been found in a total of 14 free-ranging deer in Saskatchewan, in three geographically distinct areas in the province (Figure 1). The largest number of CWD affected deer has been from an area north of Swift Current on either side of the South Saskatchewan River near Saskatchewan Landing Provincial Park. A total of 9 affected mule deer and one affected white-tailed deer have been found in this area. CWD has not been detected in deer and elk in Manitoba; Alberta has detected CWD on two deer/elk farms, but none in wild elk or deer.

Chronic wasting disease is a difficult disease to detect in wild deer. Initially, very few deer are likely to be affected in most areas and these deer are likely to occur in relatively small geographic areas. Therefore large numbers of animals must be tested from many different areas in order to determine the geographic range of this disease. One method of detecting CWD in new areas is to ensure that all deer that appear ill are submitted to a veterinary diagnostic laboratory for complete autopsy and CWD testing.

There are many unanswered questions regarding CWD. How did the disease first occur in North America and in Saskatchewan? Did it occur spontaneously in deer or did the elk and deer acquire the disease from some other wild or domestic species? How and when is the disease transmitted from animal to animal? What are the long-term effects on deer populations? How fast will the disease spread? Will CWD spread to other species, including people, under certain circumstances? The answers to these questions are critical in order to develop an appropriate management response to this disease. Although research on CWD is underway in some parts of the United States, more research is needed, particularly in Canada. (Trent Bollinger, CCWHC Western and Northern Region).

Table 1. Summary of chronic wasting disease surveillance in Saskatchewan, 1997 to August 2003 (as of October 31, 2003 two additional mule deer have tested positive for CWD during the ongoing surveillance program).

Year	White-Tailed Deer	Mule Deer	Elk	Total/year	Test Negative	Test Positive
1997	36	2	0	38	all	none
1998	18	91	2	111	all	none
1999	57	79	44	180	all	none
2000	726	185	89	1000	999	1 mule deer
2001 spring	58	155	0	213	212	1 mule deer
2001 fall	2,236	1,077	340	3,653	all	none
2002 spring	23	162	0	185	184	1 mule deer
2002 fall and 2003 winter	2,416	3,224	163	5,841	5,832	9 (7 mule deer & 2 white-tailed deer)
Fall 2003 as of Nov 20, 2003	82	291	20	400	396	4 mule deer
Total to date	5,652	5,266	658	11,621	11,605	16

Figure 1. Locations where chronic wasting disease has been detected in wild deer in Saskatchewan.

image pending

Disease Updates

Atlantic Region

Esophageal Lesions in Crows

The submission of American Crow carcasses for West Nile virus testing has provided a unique opportunity to observe a range of disease problems affecting crows. Four crows from Prince Edward Island, submitted to the CCWHC - Atlantic Region in the spring of 2002 and 2003, had interesting pathological changes of the esophagus (gullet).

Two crows had zones of severely thickened and inflamed tissue in the upper esophagus. In both cases, the tissue damage was associated with the presence of adult nematode worms and their eggs, identified as *Capillaria* sp.. In one of the birds, proliferation of new tissue in the abnormal area was very pronounced, resulting in a highly irregular surface with folds up to one centimeter in length.

These two crows were found during the same week in March, on opposite sides of Prince Edward Island. Despite the examination of thousands of crows during the 2002 and 2003 West Nile Virus Surveillance Programs, no other crows with similar abnormalities were identified. The infective larval stage of *Capillaria* sp. is usually found in earthworms, suggesting that these crows may have become infected in the previous summer or fall, before the ground froze.

Two other crows had cancer of the upper esophagus. These were identified as: 1) adenocarcinoma (from the glands of the esophagus) and 2) squamous cell carcinoma (from the surface of the esophagus). In both cases, the tumors had spread to the lungs (adenocarcinoma) and liver (squamous cell carcinoma). Tumours of the esophagus are rarely reported in domestic animals or birds, and even less so in free-ranging wildlife. (Scott McBurney and María Forzán, CCWHC Atlantic Region)

Surplus killing of Roseate Terns and Common Terns by a mink

The Brothers Islands, two small islands off the southwestern shore of Nova Scotia, hold Canada's largest colony of the threatened Roseate Tern. In early summer 2003, a total of 85 Roseate Tern nests were located on these two islands. On July 3, while on the islands to band the chicks, biologists found five dead adults and only 18 live chicks among the 85 nests. The dead adults were found next to their nests. Necropsy done at CCWHC - Atlantic Region strongly suggested predator attack, possibly by mink (*Mustela vison*). With the help of the Nova Scotia Department of Natural Resources, efforts were immediately taken to catch the predator.

More carcasses were found when the islands were visited to set traps. In total, 47 bird carcasses from the island were examined: eight adults (seven female, one male) and 18 chicks of Roseate Terns, one adult (female) and 19 chicks of Common Terns, and one adult (male) Arctic Tern. 25 birds had obvious fractures and hemorrhage of the skull or neck, nine had only hemorrhage of soft tissues over the skull

and/or neck, and four had laceration of portions of their pectoral muscles or hemorrhage in their thorax and abdomen. Nine chicks had no gross or microscopic lesions. Traumatic injuries in one particularly well preserved adult Roseate Tern suggested that it had died from asphyxiation following a bite to its throat. There was very little, if any, evidence of consumption of the flesh in any of the carcasses. A large male mink was caught on one of the islands around July 14th. By then, adult terns were still active at the colony, but no live Roseate Tern chicks, and very few Common Tern chicks, could be found.

Surplus killing, defined as a predator killing more prey than it can consume at the time, can occur when prey species become easily accessible, such as when ungulates are attacked by wolves or coyotes during severe winters with thick snow conditions that immobilize the prey (Patterson, Canadian Field-Naturalist 104:484-487, 1994; DelGiudice, Journal of Mammalogy 79:227-235, 1998). It is a rare event under natural conditions, but mink are notorious surplus killers, particularly of colonial birds. One report describes surplus killing of Eared Grebes in central British Columbia, in which 50 adult birds were found dead on their nest (Breault and Cheng, Canadian Field-Naturalist 102:738-739, 1988). Only three carcasses showed any sign of having been eaten. All carcasses examined had small puncture wounds on the skull and neck; in some cases, the skull was partly crushed. The size and distribution of the puncture wounds suggested that one or more mink had been responsible for the killing. In the present case, the good cooperation among all parties involved led to quick remedial action which may

have prevented complete abandonment of the colony. (Pierre-Yves Daoust, CCWHC - Atlantic region; Andrew Boyne, Canadian Wildlife Service; Ted D'Eon, Nova Scotia).

Quebec Region

West Nile Virus infection in Québec Raptors : 2003 season

West Nile Virus infection was confirmed in 18 individuals of eight different species of raptorial birds in Quebec in 2003 (Table 1). Infected birds of prey were submitted from mid August to mid October, which corresponds with the peak of WNV-associated corvid mortality in Québec. This peak was from mid August to mid September and gradually declined until the end of October. The geographic location of infected birds of prey was similar to that of infected corvids in 2003: the southwestern part of the province, in a broad band along the St Lawrence Valley. One infected raptor was found north of this zone: a Merlin in Sullivan, adjacent to Val D'Or in the Abitibi-Témiscamingue Health Unit. WNV infected raptors were submitted either as sick individual birds to the Clinique des Oiseaux de Proie (COP) of the Faculté de médecine vétérinaire (Université de Montréal) for treatment or as dead specimens to the CCWHC – Québec Regional Center for necropsy. The Union québécoise des oiseaux de proie (UQROP) annually submits to the COP over 350 raptors of various species for treatment and rehabilitation. Of these, approximately 150 individuals are released back into the wild.

Birds infected with WNV most often displayed dehydration, lethargy, unawareness, general weakness, head tremor and

anorexia. One Northern Harrier, two Sharp-shinned Hawks, two Red-tailed Hawks and two Great Horned Owls survived suspected WNV infection. Confirmation that WNV was the cause of their illness by serology is pending. Birds dead of WNVmost often were emaciated and had pale hearts. The most prominent microscopic finding was destruction of the heart muscle with minimal inflammation and replacement by fibrous tissue. Other changes found in some individuals included inflammation of the breast muscle and inflammation of the brain and its outer membrane. WNV infection was confirmed in all 18 birds of prey by molecular analysis (PCR). Microscopic lesions of WNV infections were much more severe in these raptors than in crows, probably relating to the raptors' relative resistance to the infection. [See related reports from Ontario/ Nunavut and Western/Northern Regions] (André D. Dallaire, Guy Fitzgerald and Stéphane Lair CQSAS - CCWHC Quebec Region).

Table 1. Quebec Raptor Sopecies with Confirmaed WNV Infection in 2003

Family	Species	N
Accipitridae	Sharp-shinned Hawk	5
	Cooper's Hawk	1
	Northern Goshawk	4
	Northern Harrier	1
	Red-tailed Hawk	2
Falconidae	American Kestrel	2
	Merlin	1
Strigidae	Great Horned Owl	2

Ontario Region

West Nile Virus Update

In 2003, all raptors submitted to the CCWHC Ontario-Nunavut Region for post-mortem have been tested for West Nile virus (WNV) by polymerase chain reaction (PCR). More than 50 raptors have been tested, and, as of late October, 12 have tested positive. They include: Sharp-Shinned Hawks (5), American Kestrels (2), Northern Goshawk (1), Merlin (1), and Red-Tailed Hawk (2). Most of these birds had severe microscopic tissue changes often involving heart and brain, and virus was abundant in these affected tissues, indicating that WNV was the cause of death. Several of these species prey upon other birds and thus may have acquired infection through eating infected prey. Such transmission of West Nile Virus in birds has been demonstrated experimentally. The total number of raptors confirmed with WNV infection is fewer in 2003 than 2002. However, it is not possible to draw any conclusions from this observation.

In addition to the raptors, infection was confirmed in Ring Billed Gulls and in a Grey Squirrel (*Sciurus carolinensis*). The tissues from this squirrel tested negative for the virus by PCR, but it had microscopic lesions in the brain which have tested positive for virus by a different procedure.

A small number of bats and raccoons have been tested but all have been negative. [See related articles under Quebec and Western/Northern Regions] (Doug Campbell - CCWHC, Ontario/Nunavut Region).

Mortality in Great Black-Backed Gulls near Presqu'ile

Great Black-Backed Gulls are an uncommon species on Lake Ontario. This fall, there have been at least 3 separate episodes of mortality between early September and late October, at the eastern end of Lake Ontario between Presqu'ile Provincial Park and Scotch Bonnet Island to the east. Only 40-50 pairs of this species nest in this area, although more birds move into the area during the post-nesting period when these mortalities were observed. The total number of affected birds is at least 15, and may be considerably greater. Only this one species appears to be affected. Sick birds cannot fly or walk, and eventually die. Post-mortem findings in birds submitted to the CCWHC lab in Guelph have been unremarkable. The birds were in good body condition, although they had not eaten recently. There were no significant gross or microscopic abnormalities.

The birds have tested negative for West Nile virus and serum from one live bird had no antibodies to Newcastle Disease virus. Tissues also have tested negative for botulinum toxin.

The first nesting of Great Black-Backed Gulls on Lake Ontario was recorded in 1954, but the species has not increased in numbers as have Herring Gulls and Ring-Billed Gulls. During the period of the 1950s to 1970s, it was possible to attribute this lack of growth to the effects of contaminants, but as contaminant levels have fallen, population levels have not increased at the same rate as for other fish-eating birds. Causes of mortality, particularly during the post-nesting, post-colony departure period, have not been investigated. It is possible that high levels of

mortality during this period play a role in the slow growth of the population of this species. (Chip Weseloh, Kate Gee, Jeff Robinson - Canadian Wildlife Service; Doug Campbell - CCWHC).

Adenovirus Encephalitis in a Wild Fox

During late June, 4 dead red foxes were found on the property of an electricity generating station at Bowmanville, east of Toronto. Two animals were submitted for necropsy 3 weeks apart. The first animal was rotten and no significant findings were made at post-mortem. The second fox was an adult female in poor nutritional condition. Gross dissection revealed only mild pulmonary congestion. A variety of material - porcupine quills, aluminum foil and feathers - was present in the stomach. Microscopically, there was mild inflammation of the brain in the form of inflammatory cell cuffing of numerous blood vessels. In these blood vessels, there were numerous large, basophilic inclusion bodies in endothelial cells, typical of adenovirus infection and Canine Adenovirus (Infectious Canine Hepatitis virus) was confirmed in these cells by immunological tests.

Infectious Canine Hepatitis is well-recognized as a disease in captive foxes ("fox encephalitis") but has rarely been reported from wild foxes. This is the first case recognized in wild foxes in Ontario. (Doug Campbell - CCWHC, Ontario Region; Keith West - Prairie Diagnostic Services, Saskatoon).

Western/Northern Region

Newcastle Disease Virus in Double-crested Cormorants in Alberta and Saskatchewan in 2003

Newcastle Disease outbreaks occurred on colonies of Double-crested cormorants in both Alberta and Saskatchewan in July and August of 2003. Approximately 1500 sick or dead cormorants, largely juveniles, were seen at seven colonies in the vicinity of Lac La Biche and St Paul , Alberta. Birds were observed in various degrees of partial, often unilateral, paralysis and were weak and disoriented. There were multiple reports of cormorants occurring in unusual situations including walking along roads as well as landing in yards and on golf courses and washing up on recreational shorelines. Avian scavengers, particularly turkey vultures, were common at some colonies. There were no reports of undue mortality in domestic poultry.

In Saskatchewan, small numbers of sick hatch-year birds were found in the Island A colony site of Doré Lake on July 21, and on 17 August, approximately 500 dead hatch-year birds were found. In early September, about 1300 dead cormorants were found at the colony site in LaVallée Lake in Prince Albert National Park.

Fresh specimens from Alberta colonies and from Doré Lake were examined for cause of death. These birds had inflammation of the brain typical of Newcastle Disease. Virus isolation and characterization was done by the National Centre for Foreign Animal Diseases (CFIA) in Winnipeg and confirmed the presence of highly pathogenic avian paramyxovirus-1, the agent of Newcastle Disease.

Newcastle disease virus appears to be maintained in double-crested cormorants and has caused outbreaks on colonies in the prairie provinces recurrently since is was first

recognized in 1990. This disease also occurred in double-crested cormorants in New York State and Vermont in the summer of 2003. MJ Pybus (Alberta Fish and Wildlife Division), T. Inglis (Poultry Health Services Limited, Edmonton), Dan Fransden and Fiona Moreland (Parks Canada), Ted Leighton (CCWHC).

West Nile Virus in Sage Grouse

West Nile virus (WNV) was identified in early August 2003 in Sage Grouse in Wyoming and Montana. In August of 2003, seven of 29 radio-collared birds in southeastern Alberta were found dead and were found to be infected with WNV. As of October 24th, WNV is considered the probable cause of death of a total of 27 sage grouse in North America: 19 in Wyoming, 5 in Alberta, 3 in Montana. Results are pending on 8 additional carcasses from WY and MT. Most of the WNV-related mortality occurred from late July to mid August.

The Sage Grouse is listed as an endangered species nationally and provincially in Alberta and Saskatchewan. The species is extirpated in British Columbia. There are only about 350 birds in Alberta and about 250 in Saskatchewan. Mortality associated with WNV may put the two provincial populations at greater risk. Mortality in Alberta during July and August 2003 was significantly greater than was the case in previous years.

The Alberta Cooperative Conservation Research Unit [ACCRU] and the Alberta Sage Grouse Recovery Team are assessing management responses prior to the next West Nile

season. (MJ Pybus, Alberta Fish and Wildlife; Cameron Aldridge, University of Alberta; Trent Bollinger, CCWHC W/N Region, and Sue McAdam, Saskatchewan Environment).

The Research Group for arctic Parasitology (RGAP), November 2003

The Research Group for Arctic Parasitology (RGAP), organized within CCWHC, is a collaborative group of wildlife parasitologists, biologists, veterinarians, and user groups concerned with parasites of northern mammals and birds. A developing focus for RGAP is the effect of climate change on arctic parasites and other infections of wildlife. Previous RGAP studies described many aspects of the life cycle of the newly-discovered lungworm of muskox (*Umingmakstrongylus pallikuukensis*). A similarly comprehensive approach is now being applied to *Parelaphostrongylus odocoiei*, another nematode parasite affecting Dall's Sheep in the Mckenzie Mountains. For both parasites, larval development and exposure of animals to infection are highly sensitive to summer temperatures, and field and laboratory experiments have been used to test models and make predictions about new patterns of infectious diseases that may emerge as the arctic climate warms. Recent publications describing this work include Kutz *et al*. 2002. *Can.J.Zool*. 80:1977-1985 and Dobson, Kutz *et al*., 2003 in *Advances in Applied Biodiversity Science (Conservation International)* pp 33-38. (Susan Kutz, Emily Jenkins and Lydden Polley - Western and Northern Regional Centre).

Death of a Grizzly Bear caused by capture and handling

In early June 2003, an adult male Grizzly Bear was captured by leg-hold snare in west-central Alberta, and was restrained in the snare for as long as 24 hours before being anesthetized,handled and released. The snare was anchored by a short cable (1.2m) to a tree and restrained the animal around its left wrist. The bear was anesthetized with a mixture of xylazine and Telazol® administered using a 3 ml rapid-injection dart (plunger advanced by explosive charge) fired into the muscles of the right shoulder. The bear was in good body condition and weighed 245 kg. Superficial injuries noted at the time of handling were a small diameter puncture at the site of dart injection and a 2.5 cm laceration on the medial aspect of the carpus. Routine measurements and samples were collected, including blood, and a Global Positioning System (GPS) collar was fitted around the neck of the bear prior to release. At the conclusion of handling, the bear was administered yohimbine to reverse anesthesia.

The bear was sighted by helicopter three times over a 15-day period following release: one day later, moving through forest approximately 1.3 km from the capture site; 5 days later, at 2.8 km from the capture site; and 10 days later, when the bear was found dead at the same location as on day five.

Post mortem examination revealed greater damage to the left wrist by the snare than had been recognized previously; the laceration had cut deeply and one wrist bone was fractured and partially missing. The muscles of the upper left forelimb and chest were swollen and very pale due to extensive necrosis (confirmed by microscopy) typical of

muscle damage caused by excessive exertion (capture myopathy). The lungs were congested and wet, and there were two litres of fluid in the chest cavity; both findings suggest death by heart failure. The dart wound site in the right shoulder had become a large (3-4 cm) abscess with some associated muscle necrosis and heavy infection with the bacterium *Clostridium perfringens* (identified by PCR).

Analysis of blood taken at the time of capture showed increased numbers of neutrophils exhibiting toxic change. The blood contained high concentrations of muscle enzymes, indicating that muscle injury already had occurred when the blood sample was taken. The analysis also indicated that the bear was dehydrated when it first was handled.

Leg-hold snares, drugs and dart rifles are tools commonly used for wildlife management, research, and conservation, and are generally believed to cause minimal injury to animals when employed correctly. Nevertheless, for each method, it is difficult to determine the proportion of animals that experience prolonged suffering, due to their use.

Health and movement data gathered during the long-term conservation project for which this bear was captured show three lines of evidence to suggest that our standard capture techniques for grizzly bears may commonly cause significant injury more often than previously thought. First, results from blood analyses indicate bears captured by leg-hold snare often have elevated white blood cell counts and serum concentrations of muscle enzymes comparable to values recorded for this bear that died from capture myopathy. Second, analyses of body condition over time indicate bears

captured multiple times over different years are in poorer body condition than bears captured once only. Third, analysis of movement rates for individual bears indicates that many bears move little in the days immediately following capture, with movement rates remaining sub-normal for approximately 3 weeks. Although these results are preliminary, they emphasize the need for wildlife personnel to continue to work to improve capture methods to minimize deleterious effects on wildlife and to use every opportunity to assess the potential impact of the methods used. (Marc Cattet, Trent Bollinger – CCWHC W/N Region; Gordon Stenhouse – Alberta Sustainable Resource Development Fish and Wildlife Division and Foothills Model Forest Grizzly Bear Research Program).

Announcements

Diagnosing Disease in Wild Animals, February 25-27, 2004, Saskatoon, SK

This short-course will provide an overview of the processes involved in determining the cause of disease in sick or dead animals. Lecture and lab sessions will cover: necropsy techniques, how to recognize lesions, proper collection of samples for further diagnostic testing, what tests are available, and how to obtain the most information from a wildlife disease occurrence. This course will emphasize a practical hands-on approach to assist field personnel in dealing with diseased wildlife or in undertaking wildllife disease surveillance programs.