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**Centre Canadien
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News from the CCWHC

Quebec Pathologist

Congratulations to Igor Mikaelian, pathologist at the Quebec Regional Centre, for successfully completing the arduous certifying examination of the American College of Veterinary Pathology. Igor managed to prepare for this examination while working full-time for CCWHC; his success is a great personal and professional accomplishment.

Italian Conference

Gary Wobeser, Western/Northern Region, participated in a meeting of the Società Italiana di Ecopatologia della Fauna (Italian Society for Ecopathology of Wildlife) in Bormio, Italy in October. This society is sponsored by 5 of Italy's 14 veterinary colleges as well as many agencies involved with wildlife and domestic animal health. It deals with an array of wildlife health concerns similar to those in Canada, with greater emphasis on the role of wildlife in diseases shared with livestock, such as classical swine fever. The meeting provided an insight into the amount and high quality of wildlife health work being done in Europe, of which we in North America hear very little.

Feature Article

A Critical Need for Information on Diseases of Wild Amphibians

There is increasing concern over declines in amphibian populations throughout the world. Habitat destruction and alteration, acid rain, pollution, disease, and introduction of predators have all been reported as causes of local population declines. In other situations, declines are occurring in apparently pristine environments. The loss of some amphibian species and population declines in others are thought to be part of a global loss of biodiversity brought about by environmental degradation. However, some researchers suggest that amphibian declines are unique and that amphibians are unusually sensitive indicators of environmental degradation, because of characteristics of their biology and physiology, such as permeable eggs, skin and gills and complex life-cycles, which make them more susceptible to these effects. They suggest that amphibians are analogous to canaries in coal mines and are harbingers of the effects of environmental degradation on other vertebrate species. Whether this is true or not, and how much of the reported amphibian population declines is due to natural population variation is currently being debated; undoubtedly more research will be required.

Although diseases have been implicated in local population declines, our understanding of diseases affecting amphibians is very rudimentary. *Aeromonas septicemia* or "red-leg" is probably the most widely recognized cause of amphibian mortality and has been reported to cause die-offs of amphibians in the wild. *Aeromonas hydrophila*, the bacterium responsible for this disease, is widespread in aquatic environments and can be isolated from the skin of healthy fish and from water. Generalized infection is thought to occur secondary to some other disease process or in animals whose immunity has been suppressed by disease or stress. The visible signs of red-leg are similar to other diseases and without a complete necropsy other diseases will be missed.

Fungi have been implicated as the cause of mortality in various amphibians. Recently, *Saprolegnia ferax*, a water mold that commonly attacks fish, has been implicated as the cause of

mortality in egg masses and embryos of the western toad (*Bufo boreas*) in Oregon. Increased ultraviolet B radiation, because of depletion of the ozone layer, has been proposed as a cause for the increased susceptibility to these pathogens but the evidence is contradictory. Infection by chytrid fungi has been associated recently with large die-offs of frogs in Queensland, Australia, Costa Rica and Panama. This type of fungus is ubiquitous in soil and water but has not been previously shown to cause disease in vertebrates. Spread of this disease to new locations over time has been reported in some cases.

Newly recognized diseases also have been reported recently in Canada. A high prevalence of hindlimb deformities has been reported from wild-caught green frogs (*Rana clamitans*), northern leopard frogs (*R. pipiens*), American toads (*Bufo americanus*) and bullfrogs (*R. catesbeiana*) from the St. Lawrence River Valley, in Québec. The average prevalence of deformities was 12% among amphibians from agricultural sites, compared to 0.7% at non-agricultural control sites. Although the differences were not statistically significant due to high variation in prevalence among sites, research is underway to determine if agricultural practices may be causing limb deformities.

On the prairies, a new viral disease has been recognized as a cause of mortality in tiger salamanders (*Ambystoma tigrinum*). This virus, called Regina Ranavirus (RRV), is similar to other iridoviruses recently shown to be the cause of mortality among captive and wild fish, and amphibians in several areas of the world. A closely related virus, *Ambystoma tigrinum* virus (ATV), was reported to cause a 1995 -1996 die-off in the endangered Sonoran tiger salamander of Arizona. In 1998, iridoviruses were responsible for mortality of tiger salamanders and wood frogs (*R. sylvatica*) in Saskatchewan, and tiger salamanders in Manitoba. Iridoviruses also were thought to be involved in die-offs of tiger salamanders in the northern USA this past summer. In Australia and California, specific iridoviruses have been shown to be infectious for both fish and amphibians. This raises the possibility that movement of fish and/or amphibians may be responsible for introduction of viruses to new locations, with the potential for dramatic mortality in populations that have had no prior experience with the virus.

The emergence or discovery of many new diseases recently is, at least to some degree, a result of more thorough investigation of mortality in amphibians. It also stresses how little we know of the diseases affecting wild amphibians and fish. As habitat for amphibians becomes more fragmented, the potential for local population extinction will increase. As has occurred with other species, programs to translocate amphibians from geographically distant sites have and will be implemented to reestablish populations. The perils of introducing diseases to naive wildlife populations through translocation, or of exposing translocated animals to diseases already present at the release site, are well documented in other wildlife species. We need to avoid making mistakes such as the translocation of tuberculosis in bison to Wood Buffalo National Park, the introduction of whirling disease in trout to new areas, and the death of caribou (*Rangifer* sp.) introduced to areas where meningeal worm (*Parelaphostomum tenuis*) was present in white-tailed deer (*Odocoileus virginianus*). Information on diseases of amphibians is needed to prevent future introduction of new diseases, and to ensure the success of introduction programs. The potential for introducing diseases to other aquatic species through movement of fish for fish farming and sport fishing warrants further investigation. Improved investigation of

fish and amphibian die-offs is the first step in understanding how disease may be affecting amphibian populations. [Trent Bollinger, Western/Northern Regional Ctr].

Selected References:

Pechmann, J. H. K. and H. M. Wilbur. 1994. Putting declining amphibian populations in perspective: natural fluctuations and human impacts. *Herpetologica* 50 (1): 65-84.

Mao, J., D. E. Green, G. Fellers, and V. G. Chinchar 1998. Iridoviruses from sympatric amphibians and fish: Isolation, pathology and molecular characterization. *Virus Research* (in press).

Update on Zoonoses

Chlamydiosis in Wildlife Workers

Chlamydia psittaci is an unusual bacterium that occurs in many species of birds. It usually does not cause disease, and healthy birds may shed the organism in their droppings. The bacterium can remain alive in the environment for long periods and may be inhaled with dust from dried bird feces. Human infection has been known to occur for many years; two recent cases in western Canada demonstrate a risk for wildlife personnel. In the spring of 1998, an enforcement officer inspected premises of 15 aviculturists in Saskatchewan. Birds in the collections were predominantly native waterfowl. He subsequently developed stiffness and muscle aches, chills and a fever that persisted until he consulted his physician a week later, when pneumonia was diagnosed. Eleven days later, the individual asked his physician to do a blood test for antibodies to *C. psittaci*. This test was positive. In total, the individual was sick for 4 weeks. The second individual was involved in waterbird carcass cleanup during botulism outbreaks in northern Alberta in the summer of 1998. He developed aggressive flu-like symptoms followed by a prolonged hacking cough, generalized weakness and difficulty in breathing. A blood test was positive for *C. psittaci*. Individuals usually recover well after antibiotic therapy but in some cases the infection can be persistent and recurrent, requiring aggressive and consistent treatment.

The exact source of infection for these individuals will never be known, but it is likely they were exposed to airborne stages of *C. psittaci* while examining buildings in which waterfowl were held and while handling bird carcasses. Because human chlamydiosis of this type is uncommon, most physicians are unfamiliar with the disease or only associate it with exposure to parrots, because it has been referred to as "parrot fever". Another name, "ornithosis", more clearly indicates that any bird might be a source of infection. It is difficult to avoid exposure to dust when working with birds, but a suitable mask could be used in high-risk situations such as confined spaces in buildings. If someone develops clinical signs of the type described above after working with birds, it is important to inform the physician that the person has been working with birds and might have been exposed to chlamydia, so that appropriate tests can be done and proper antibiotic therapy can be started. [M. Pybus, Alberta Natural Resource Services; G. Wobeser, CCWHC].

Hantavirus in Canada

In 1993, a previously unrecognized pneumonic disease was identified in humans in the southwestern USA and soon was linked to a virus carried by the deer mouse *Peromyscus maniculatus*. The virus, which is one of a group called hantaviruses, does not harm mice but infection can cause a severe and often fatal respiratory disease in humans that is now called "Hantavirus Pulmonary Syndrome" (HPS). Evidence accumulated since 1993 indicates that the causative virus is widespread in deer mice throughout North America and has been present for many years. In addition, three other hantaviruses in the United States associated with other rodent species (white-footed mouse, cotton rat, rice rat) have been shown to be capable of causing HPS. Approximately 200 human cases have been recognized in the USA. As of November 20, 1998 there have been 30 human cases in Canada: 19 in Alberta, 6 in British Columbia and 5 in Saskatchewan; 10 of the individuals have died of HPS in Canada. Infected deer mice have been found in eight provinces and the Yukon. No infected mice have been found in Prince Edward Island, Nova Scotia or the Northwest Territories to date, but based on the discontinuous distribution of infection in deer mice sampled throughout the country, no area can be assured to be free of infection.

Hantavirus infection is a rare disease in humans in Canada but it is serious because of the high fatality rate. Infection occurs through inhalation of aerosols from urine, feces or saliva of deer mice. Human cases have been associated with rodent contact primarily in infested buildings or with handling deer mice. Rodent infestation of buildings should be prevented, and infested buildings should be disinfected with 10% chlorine bleach and thoroughly aired before they are cleaned. Care must be taken in handling live or dead deer mice. Detailed information on preventive measures is available from local public health authorities. [Information provided by Dr. H. Artsob, Laboratory Centre for Disease Control, Health Canada, Winnipeg].

Disease Updates

Atlantic Region

Listeriosis in a Canada goose

An adult male Canada goose was submitted for necropsy examination in September, 1998. A conservation officer found the goose dead in a bird sanctuary near Milltown Cross, Prince Edward Island, and it was the second mortality in this location identified in 3 weeks. The bird was in good body condition (body weight = 3.6 kg), but had excessive fluid beneath the skin, in the visceral membranes and in the lungs, as well as necrosis and inflammation affecting approximately 40-50% of the liver. The bacterium *Listeria monocytogenes* was isolated in high numbers from the liver, kidney and lungs. The cause of this goose's death was attributed to generalized infection with this bacterial pathogen.

Listeria monocytogenes is a small, Gram-positive bacterium that is ubiquitous in the environment and is able to propagate outside of a host species. This organism can be isolated from the feces of healthy individuals in a variety of bird species, suggesting either routine ingestion of the bacterium or that healthy birds can carry and spread the infection. *L. monocytogenes* also is reported to cause sporadic deaths, due to both acute and chronic infections, in a number of avian hosts. The clinical disease is associated with lesions of the heart,

liver and brain, although, in birds that die acutely, no gross lesions may be found. A confirmed diagnosis requires the isolation of *L. monocytogenes* from affected tissues. Other species of *Listeria* (*L. ivanovii*, *L. innocua* and *L. seeligeri*) are recovered commonly from birds. There is little information on the pathogenicity of these *Listeria* spp., although *L. innocua* is considered apathogenic. While listeriosis is a documented cause of sporadic avian mortality, there is little known about the epidemiology of the disease in wild bird species. [Scott McBurney, CCWHC, Atlantic Region and Paul Walker, Fisheries and Environment, PEI].

Bowhead whale (*Balaena mysticetus*) stranding on the northeast coast of Newfoundland

In mid October 1998, a baleen whale was reported drifting and repeatedly beaching along a fjord in northeastern Newfoundland, finally coming aground near Rattling Brook (49 40'N; 56 10'W). The whale had been tentatively identified as a right whale (*Eubalaena glacialis*) when partly submerged; the lack of callosities on its head and the white color of its chin identified it as a bowhead whale. It was a female, and its relatively small size (length of 9 m from rostrum to tail notch, as compared to 14-18 m in adults) indicated that it was immature. Numerous large bites had been taken from the blubber along its belly, although its fluke and flippers were largely intact. It remains unclear whether this extensive loss of blubber was attributable to scavengers (such as Greenland sharks) or to predators (such as killer whales). This animal was estimated to have been dead for at least 2-3 weeks when necropsied, and advanced postmortem decomposition of its internal organs greatly limited gross and microscopic examination. However, the biology of the species, some of the gross findings, its relatively young age and, therefore, presumed inexperience suggest that it was at least in a moderate degree of emaciation. The bowhead whale is "adapted to live in the loose edges of the north polar sea ice. Its annual migrations roughly track the advance and retreat of the [ice] floe edge" (Dyke et al., *Arctic* 49:235-255, 1996). Its southernmost range in winter in Davis Strait may reach the northernmost part of Labrador (approx. 60 N; 60 W) (Kinley, *Arctic* 43:137-152, 1990); its summer range is much further north. Therefore, this whale was far out of its normal range and, thus, its normal food source for that time of year, even when accounting for the amount of time that the carcass may have been drifting southward. According to Dyke et al. (1996), "bowheads feast in [the summer- fall feeding grounds] on zooplankton ..., and eat little if anything for the rest of the year". Therefore, one should have expected maximum fat reserves in this animal in October, in preparation for its winter fast. Again according to Dyke et al. (1996), "the bowhead has the thickest blubber layer of any mammal (up to 50 cm)", although, admittedly, these authors are likely referring to adult animals. The maximum blubber thickness recorded in this whale was 33 cm, but, at several other sites, it was only 16 to 20 cm. Moreover, there was no evidence of fat around the heart, whereas, at least in some other species of baleen whales that undergo a winter fast, the amount of fat in this location can be very abundant.

Because of the fast pace of postmortem decomposition in large whales, interpretation of necropsy results is often speculative, as was the case here. However, opportunities for such necropsies are uncommon and should not be missed. This particular animal represents the southernmost record of a bowhead whale in the northwest Atlantic. It was a unique specimen which deserved scrutiny.

[Pierre-Yves Daoust, CCWHC - Atlantic Region; Jon Lien, Memorial University, St. John's, Newfoundland; Amy Knowlton, New England Aquarium, Boston, Massachusetts].

Quebec Region

Diseases of beluga whales (*Delphinapterus leucas*) from the St. Lawrence Estuary: 1998

The 1998 health assessment program of beluga whales from the St. Lawrence estuary has been supported by Parks Canada, the World Wildlife Fund Canada, and the Department of Fisheries and Oceans to whom we are grateful. We summarize the results of necropsy of 8 beluga.

Two adult females had mammary gland cancer (adenocarcinoma), which had spread to viscera and bone. This brings to three the number of mammary cancers in beluga whales from the St. Lawrence estuary from 1983 to 1998 and represents a prevalence of about 8 % in adult female beluga whales (n = 39). In humans, it has been proposed that endocrine disruptors could have an important role in the development of mammary tumors. Tissue levels of organochlorine compounds in these two animals are presently being assessed, and will be compared with those in other belugas that were free of mammary tumors.

Three adult whales died of bacterial infections: a young adult female that had just calved had hemorrhages on all internal surfaces and in the subcutis. *Aeromonas hydrophila*, a bacterium common in water, was isolated from all organs. An adult whale had infection and inflammation of the heart valves (valvular endocarditis) with spread to the lung. *Vibrio fluvialis* was isolated from the lesions. This bacterium is isolated occasionally in fish but its pathogenicity in marine mammals is unknown. An adult male had a large abscess that replaced about two-thirds of his right lung. The bacterium *Edwardsiella tarda* was isolated from the abscess.

Death was attributed to verminous (parasitic) pneumonia in a juvenile female. Lungworms, such as *Halocercus monoceris*, have been incriminated in the death of 18 % of beluga whales found stranded on the shores of the St. Lawrence estuary from 1983 to 1997 (n = 85).

A < 1 year-old whale had systemic toxoplasmosis. This is the second case of generalized infection with the protozoan parasite *Toxoplasma gondii* in a beluga whale. The first case was observed in an aged female in 1988. (See report of toxoplasmosis from the Western/Northern Region for more details on this parasite).

Death of an adult male was attributed to severe bacterial infection secondary to a large, rectangular, purulent wound located close to the anus. The cause of this wound was not determined.

In addition to necropsies, studies performed this year concentrated on the herpesvirus of beluga whales as a possible cause for necrotizing dermatitis, a severe skin disease found in three different beluga whales, and on the possible role in beluga cancers of the gene known as p53, an important player in many forms of cancer in humans and other species. The delay between death of the animals and sampling of tissues in the necropsy room may have hampered the search for these factors. A previous study (Environ. Health Persp. 1993, 101, 371-377) found that about 60

% of beluga whales from the St. Lawrence estuary had antibodies against herpesviruses. [Igor Mikaelian, Daniel Martineau (CCCSF-Quebec), Lena Measures, Michel Lebeuf (Institut Maurice Lamontagne, Peches et Oceans Canada)].

Syngamiasis in an American robin (*Turdus migratorius*)

Death of a yearling American robin, found dead in a public park in Montreal, was attributed to about 15 adult *Syngamus* sp. nematodes that almost totally obstructed the upper portion of the trachea. Also, skeletal muscles of the bird were very pale, indicative of severe anemia. During banding studies in Montreal parks, we have noted that most young American robins had marked respiratory difficulty, especially those captured during the hottest periods of summer. Clinical signs in these birds included open-mouth breathing, coughing, respiratory sounds, and rapid respiration. These signs were presumably caused by infection by this same syngamid worm. Other bird species and adult American robins captured at the same locations did not have these clinical signs.

Adult female *S. trachea* nematodes live attached to the wall of the trachea where they feed on blood. Males are permanently attached to females. Parasite eggs transported to the bird's mouth by coughing and by the mucociliary escalator are swallowed and passed in the bird's droppings. Larva undergo two molts within the egg and become infectious after about 2 weeks. If eggs or larvae are eaten by an earthworm or other invertebrate, the larvae encyst in the muscles of the invertebrate where they can persist indefinitely. Birds become infected by ingesting eggs or larvae or infected earthworms. Larvae, once ingested, migrate to the lung where they develop. Males and females copulate in the lung and migrate up the trachea. The first eggs are laid a week later.

In North America, *Syngamus* sp. is a common parasite that preferentially affects American robins rather than other species of birds. In Delaware, 57 % of young American robins were infected. There is strong evidence of "self-curing" in the majority of young birds³, which explains why infection is less prevalent in older birds.

Wehr, E.E.. Endoparasites. In: Infectious and parasitic diseases of wild birds. Davis JW, Anderson RC, Karstad L, Trainder DO, eds. Iowa State University Press, Ames, Iowa, 1971, 185-233; 2 *Avian Dis.* 1986, 30, 736-739; 3 *J. Wildl. Dis.* 10:397-398. [Corinne Tastayre (La Maison de l'Arbre), Igor Mikaelian, Daniel Martineau (CCCSF)].

Ontario Region

Lead Poisoning in Canada Geese Due to Skeet Shot Ingestion

Two separate incidents of lead poisoning in Canada geese due to ingestion of lead skeet shot were diagnosed in southern Ontario this fall. In the first incident, two geese were seen with neurologic signs and subsequently died on a small pond near Terra Cotta. Canadian Wildlife Service investigators submitted one bird to the CCWHC. Thirteen lead shot, all less than 2mm diameter, were found in the gizzard.

In the second incident, 22 geese were seen with neurologic signs or were found dead over a one week period on a small pond adjacent to a propane supply depot in Blyth, Ontario. Ministry of Natural Resources personnel retrieved and submitted 6 birds for necropsy. Four birds were in emaciated condition, with marked esophageal and proventricular dilation and impaction. The two other birds were not emaciated and had no evidence of impaction. All birds, however, had large numbers of small lead shot in their gizzards. The birds in good body condition, which were among the first to die, had ingested 80 to 100 shot.

The shot was assumed to have been used for skeet on the basis of its small diameter, and the uniform size of the shot present in the birds' gizzards, without significant evidence of abrasion. If the shot were originally of a larger size, there would be a range of sizes present in the gizzard, varying with the degree of erosion. There are several shooting clubs in the area where the birds may have picked up this shot. Geese in the area are a mixture of resident and migratory birds, and it is not known whether these birds were migrants. If so, they may have ingested the shot some distance north of where the incident occurred. [Caroline Brojer, Doug Campbell, CCWHC - Ontario Region; Andrew Taylor, CWS; Rick Williams, OMNR]

Canine Distemper Virus in Raccoons (*Procyon lotor*) and Striped Skunks (*Mephitis mephitis*)

Since late summer, there have been reports of large numbers of raccoons and lesser numbers of skunks found ill with clinical signs suggestive of canine distemper or found dead in areas where affected animals have been observed. The CCWHC has received animals from the Niagara Peninsula, western end of Lake Ontario, and eastern Ontario, and reports of affected animals have been received from most parts of the metropolitan Toronto area. Since the beginning of May, 172 sick or dead raccoons have been picked up within the city of Burlington, west of Toronto. In Toronto, 60 to 80 sick raccoons have been picked up monthly during the autumn. The majority of these are likely canine distemper cases.

For observers and submitters of these animals, the confusing and worrisome aspect of the disease is that it is difficult to distinguish distemper from rabies on the basis of the clinical signs and behaviour of the animals. Many of the animals appear stupefied, while others are extremely aggressive. The range of behaviour observed likely reflects the area of the brain affected by the virus.

Brains have been submitted to the Canadian Food Inspection Agency laboratory in Nepean for rabies testing from all animals submitted to necropsy and all have been negative. In all of the animals necropsied, there have been lesions compatible with, or confirmatory of, canine distemper. The Ontario Ministry of Natural Resources is conducting a trap-vaccinate-release program, aimed at prevention of raccoon rabies, in the Niagara peninsula and eastern Ontario. Concern regarding incursion of rabies into these areas has led to a greater degree of surveillance and heightened concern regarding raccoons with neurologic disease. [Caroline Brojer, Doug Campbell, CCWHC - Ontario Region; Sherri Griswold, Burlington Animal Control; Paloma Plante, Toronto Humane Society].

Oral masses in mourning doves (*Zenaidura macroura*)

The Ontario Regional Centre has received numerous reports of mourning doves with large masses obstructing their oral cavities. A small number of birds with this condition have been examined at necropsy. The lesion consists of a large, cheese-like mass that microscopically is a mixture of necrosis and inflammation with a mixture of heterophils and macrophages. There is often secondary bacterial infection of the necrotic inflamed surface. The suspected cause is infection with *Trichomonas* spp. (a flagellate protozoan parasite) but no organisms have been identified from the specimens received.

Hairless grey squirrels (*Sciurus carolinensis*)

Several reports have been received of grey squirrels with little or no hair. Gross and microscopic findings have been unremarkable in the few animals that have been submitted for necropsy. In the skin samples examined to date, there has been excess dry keratinized cells on the surface of the skin with no evidence of inflammation. Hair follicles, some of which are tortuous, contain hair shafts. This phenomenon is apparently not restricted to southern Ontario and a website has been set up by a wildlife rehabilitation organization to attempt to assemble information and reports on the occurrence of this condition. Information may be submitted to:
<http://www.squirrel-rehab.org/misc/questions.html>

Western/Northern Region

Chronic Wasting Disease in A Game Farm Elk (*Cervus elaphus*) in Saskatchewan

In late March 1998, a male elk on a game farm in Saskatchewan was noted to be depressed and not coming to eat with other animals. The animal, born in 1996, was one of 68 elk (all males) on the farm, and had been on the farm since January, 1998. On April 1, the animal died while being examined by a veterinarian. It was submitted for necropsy to the Department of Veterinary Pathology, Western College of Veterinary Medicine. The tentative diagnoses were emaciation and suppurative bronchopneumonia. When tissues were examined microscopically, lesions compatible with a spongiform encephalopathy were found. Brain tissue was submitted to the Canadian Food Inspection Agency (CFIA) for immunohistochemical examination. Accumulation of disease-specific prion protein was found in the brain and a diagnosis of chronic wasting disease (CWD) was made.

Following the diagnosis, the dam of the animal and five siblings were collected by CFIA from three other game farms in Saskatchewan, euthanized, and tested for CWD by immunohistochemistry. All were negative.

This animal was the third case of CWD in Canada and the first in an animal born in this country. The previous cases, a mule deer (*Odocoileus hemionus*) in an Ontario zoo and a game farm elk diagnosed with CWD in 1996 in Saskatchewan, were imported from the USA. The dam of the current animal was imported from South Dakota in 1988. CWD behaves like a transmissible disease, and it has been suggested that lateral transmission may occur among captive elk¹. The source of disease in the current case remains unknown. Saskatchewan Environment and Resource Management and the Western/Northern Region of CCWHC began a survey for CWD in hunter-killed wild deer in Saskatchewan in 1997. This has been expanded to include more

intensive surveillance of mule deer in the vicinity of the game farms involved. 1 Miller, M.W., M.A. Wild, and E.S. Williams, 1998. Epidemiology of chronic wasting disease in captive Rocky Mountain elk. *Journal of Wildlife Disease* 34:532-538.

Toxoplasmosis in a Merlin (*Falco columbarius*)

Toxoplasma gondii is an intestinal protozoan parasite of cats that utilizes a wide variety of other animals as alternate hosts. Infection is very common but, in most situations, host animals do not develop clinical signs, although they develop microscopic cysts that persist for months in their tissues. Disease (called toxoplasmosis) has been described in many mammals but rarely is reported in birds. During July, two adult female merlins were found dead near a school in Saskatoon. One was severely autolyzed and unsuitable for examination. The other bird was in good body condition; the only gross lesion was mild swelling of the spleen. Microscopically, there was inflammation and necrosis within the liver, with occasional small oval structures suggestive of protozoan parasites present in some cells. Liver tissue was examined by an immunohistochemical method which identified these as *Toxoplasma gondii*. Animals can become infected either by consumption of food contaminated with feces from infected cats, or by consuming prey animals that have cysts in their tissues. The latter is probably what happened to the merlin, as tissue cysts have been found in both passerine birds and small rodents. [H. Philibert, G. Wobeser, CCWHC].

Botulism Mortality on the Canadian Prairies

Avian botulism continued to cause high waterfowl losses in the prairies this year but, rather than being restricted primarily to a few large lakes in the southern regions of the provinces, as occurred in 1997, the disease was more widespread (see figure and table). In Alberta, approximately 165,000 avian carcasses were collected, with 80% of the losses occurring on lakes in the boreal forest ecoregion of northern and north-central Alberta. Approximately 19,400 carcasses were collected in Saskatchewan. The statistical estimate of mortality at Old Wives Lake was 52,000, and an estimated mortality of 10,000 occurred at Crane Lake. Approximately 20,000 avian carcasses were collected in Manitoba. The total number of carcasses collected in the summer of 1998 from lakes in the Canadian prairies was 204,000. Preliminary results of a study to determine the efficiency of carcass cleanup showed that less than 25% of avian carcasses on a marsh are retrieved; therefore, total estimated avian botulism mortality for known outbreaks in the 3 prairie provinces was likely in excess of 1 million birds. Mortality by species is not available at this time. (Provincial contacts: Alberta - Margo Pybus, Alberta Fish and Wildlife, Edmonton; Saskatchewan - Trent Bollinger, CCWHC, Saskatoon; Manitoba - Garth Ball, Manitoba Natural Resources, Winnipeg).

Avian Cholera in Double-Crested Cormorants (*Phalacrocorax auritus*)

Avian cholera (infection with the bacterium *Pasteurella multocida*) occurred on four colonies of Double-Crested Cormorants in Alberta and Saskatchewan in the summer and fall of 1998. Very high mortality was documented at three of these colonies and is suspected at the fourth. The disease was first observed among hatch-year cormorants at a large breeding colony on Doré Lake, Saskatchewan (54°46'N, 107°17'W) and another on Lavallée Lake, in the northwest

quadrant of Prince Albert National Park (54°19'N,106°33'W), on 30 July and 13 August 1998, respectively. Doré Lake was revisited on 18 August, and 1523 carcasses of full-grown hatch-year cormorants were counted. Multiple specimens of freshly-dead cormorants from both locations and a Common Raven (*Corvus corax*) found on the colony at Lavallée Lake were examined by the CCWHC and found to have avian cholera. Culture and histopathology found no evidence of Newcastle disease. In late September 1998, sick and dead cormorants were observed at a nesting colony on Lac La Biche, in east-central Alberta (54°52'N, 112°05'W). Subsequent investigation found 1000 dead birds on one colony on 2 October and 300 on a second colony on 7 October, at which time further mortality since the 2 October visit also was noted at the first colony. Dead immature gulls were also noted at both colonies. Freshly-dead cormorants were examined by Dr. D.K. Onderka, Alberta Agriculture, Food and Rural Development, Edmonton, and found to have died of avian cholera. Total mortality at Lac La Biche was estimated to be 1,500 cormorants.

This is the second recognized occurrence of avian cholera in Double-crested cormorants in Canada; the first was in 1988, also on a colony at Lac La Biche and also with mortality of about 1500 birds (Mutalib and Hanson, 1989. *Canadian Veterinary Journal* 30:350) Since 1990, cormorant colonies have been under scrutiny for mortality due to Newcastle disease. However, avian cholera was not noted among cormorants during this period until this year, when it occurred simultaneously at widely-separated locations. [M. Pybus, F. Kunnas, Alberta Natural Resources Service; D. Fransden, Prince Albert National Park; S. Lightfoot, H. Philibert, T. Leighton, CCWHC]