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June 1996

## Canadian Cooperative Wildlife Health Centre, Volume 4-1, June 1996

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**Canadian  
Cooperative  
Wildlife  
Health Centre**



**Centre  
Canadien  
Coopératif de la  
Santé  
de la Faune**

Newsletter Vol 4 - 1, June 1996

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## **News from the CCWHC**

### **Four New Sponsors in 1996**

The CCWHC has gained four new sponsors for the 1996-97 fiscal year. Ducks Unlimited Canada became a sponsor in recognition of the wildlife health services of the CCWHC that have benefitted the programs of that organization over the years. Three manufacturers of crop protection products, AgrEvo Canada, Ciba-Geigy Canada, and DowElanco Canada, became sponsors in recognition of the national wild animal disease surveillance program of the CCWHC. We welcome these new sponsors not only for the financial contribution each is making but, equally, because these sponsorships significantly broaden the advice and council available to the CCWHC through its sponsors.

### **New Agreements with Parks Canada**

Two new Memoranda of Understanding, through which financial support is provided to the CCWHC for specified services, were established with Parks Canada in 1996: one with the Prairie and Northwest Territories Region, and one with the Yukon District. These add to agreements already established with the Ontario and Atlantic regions of Parks Canada and strengthen the good working relationship that has developed between the CCWHC and Parks Canada personnel.

### **Wildlife Disease Surveillance Workshop in British Columbia**

On March 22, a workshop on wildlife disease surveillance in British Columbia was held in Vancouver. The workshop was co-organized by the CCWHC Western and Northern Regional Centre and the newly-established Centre for Coastal Health, a cooperative project among the University of British Columbia, the Vancouver Aquarium and the Western College of Veterinary Medicine. Financial assistance was provided by the B.C. Ministry of Environment, Lands and Parks. The workshop involved about 40 participants and consisted of short presentations by groups involved in various aspects of surveillance in the province, followed by small group discussions on how surveillance might be improved. During general discussion, some specific goals for enhanced cooperation and mutual assistance among participants were established, and a second workshop in a year's time was called for to assess progress. A report on this workshop is available from the CCWHC or from the Centre for Coastal Health (\$3.00 per copy to cover duplication and postage).

## Feature Articles

### Surveillance of Wildlife Diseases

Much of what the CCWHC does can be called "surveillance" of wild animal diseases in Canada. In this article, I want to explain what disease surveillance is all about, its value to wildlife management and conservation, and the crucial role played in our surveillance program by field biologists, conservation officers and other professional and non-professional field workers.

Disease surveillance is a complicated business with at least four separate parts or phases. The first part of surveillance is detection of disease and this is the part in which field personnel play their crucial role. Someone must first observe disease in wildlife before anything else in a surveillance program can take place. Such observations might be of unusual mortality, of abnormal behaviour, of a low reproduction rate, or any other indicator of poor health. These observations must be recorded and passed along, together with appropriate specimens where possible, to those able to undertake the next phase: diagnosis of disease. Diagnosis is the identification of the disease itself. This is the work of veterinary personnel in suitably equipped laboratories able to perform the tests necessary to identify the viruses, bacteria, parasites, poisons, nutritional problems and other agents that cause diseases. The third phase of surveillance is information management. Data from detection and diagnosis must be collected and analyzed in ways that turn them into useful information. Finally, surveillance information must be used in making plans, decisions and policies on relevant issues. This means that the information progressively produced in the first three phases of surveillance must be readily available to decision-makers in appropriate government and non-government organizations.

The national wildlife disease surveillance program of the CCWHC operates according to this general plan. Field personnel are of singular importance to this program since it rests entirely on their observations and cooperation. The CCWHC fosters the interest and participation of field personnel by providing information and advice via a toll-free telephone number (1-800-567-2033), sending diagnostic reports on specimens to those who submit them, offering short courses on wildlife disease topics, and through publications such as the Wildlife Disease Investigation Manual and this Newsletter. The CCWHC is financed in such a way that no fees are charged to persons who submit wildlife specimens for diagnosis, thereby encouraging specimen submission.

Diagnosis of disease in wild animals is done at each of the four Regional Centres of the CCWHC but it also is done by the many provincial veterinary diagnostic laboratories across the country, as well as by some federal diagnostic laboratories and veterinarians in private practice. The veterinary expertise that is made available in Canada for the diagnosis of wild animal diseases is impressive and represents considerable support from federal and provincial departments of agriculture for wildlife disease surveillance.

Information management on a national scale is provided in Canada by the National Database of the CCWHC. This database consists of a computer program developed

specifically for this purpose, in which data on incidents of wildlife disease from as many different sources as possible are entered. Data from provincial veterinary laboratories across the country supplement the records from the CCWHC itself. Additional sources of data are being added as time and finances permit. The database is used by the CCWHC as a resource to respond to inquiries about disease incidents and, in this way, is available to everyone. The CCWHC does not make decisions or set policies for wildlife agencies but serves as a source of technical information about wildlife health and disease. To facilitate the use by agency personnel of the information provided by the disease surveillance program, the CCWHC makes such information available in a variety of forms. Information on urgent disease issues is provided periodically in the form of "Wildlife Health Advisories" sent by FAX to persons with specific responsibilities for the issue in question. Special reports are prepared for CCWHC sponsors on request and this Newsletter provides highlights of surveillance results on a regular basis.

Information from wildlife disease surveillance has been useful in three different areas of public and government concern: wildlife management and conservation, domestic animal health and public health. While the former is the true mandate of the CCWHC, significant contributions to the other two areas fully justify the cooperation and good will shown to the CCWHC and its surveillance program by agricultural and public health agencies. For example, the CCWHC surveillance program has tracked Newcastle disease in cormorants and infection with *Mycoplasma gallisepticum* in song birds, both important diseases to Canada's poultry industry. It also has provided information on the occurrence of zoonotic diseases such as tularemia, hantavirus and Lyme disease. On the wildlife front, there have been numerous applications of surveillance information from coast to coast to coast. Surveillance information has contributed to policy decisions in national parks, to risk assessments, to disease contingency plans and to endangered species recovery programs.

Surveillance is a continuous activity, and wildlife disease surveillance relies on the continuous observations and submissions of specimens by all Canadians who spend their professional or recreational time in the field observing nature. We thank all of you who participate in this way in the national wildlife disease surveillance program of the CCWHC, and we ask for your continued participation in the years ahead. (Ted Leighton, CCWHC Headquarters Office).

### **Giant Liver Fluke in Banff National Park, Alberta**

The next time you happen to be lucky enough to pass through Banff National Park (BNP) and see a herd of elk (*Cervus elaphus*) feeding in the marshes of Vermilion Lakes, look beyond the scene. Although natural, what may be happening in front of you may not be pretty. The elk could very well be picking up infections with the giant liver fluke (*Fascioloides magna*). This parasite may be causing serious problems in the ungulate populations of one of Canada's premier National Parks.

In less than 40 years, infections with the giant liver fluke in BNP have reached levels seen nowhere else in the world. Prior to 1958, the parasite was not found in the Park; since then, the percent of elk in the Bow Valley of Banff infected with the fluke has steadily increased from 3% in 1958, to 21% in 1963-64, and to 50% in 1964-65. Between 1984 and 1989, 89% of adult elk, 85% of moose (*Alces alces*), and 60% of white-tailed deer (*Odocoileus virginianus*) were infected with the parasite in Banff and neighboring Kootenay National Park, B.C. Even though these infections in moose and elk were often accompanied by extensive liver damage, infection levels have continued to rise. Between 1989 and spring 1995 an average of 93.5% of adult elk in BNP were infected.

The giant liver fluke has a two-host life cycle. Adult parasites, which can be up to approximately 9 cm long by 3 cm wide, are found in the liver of the definitive host, an ungulate. Eggs are shed into the environment in the ungulate's faeces. Eggs hatch into free-swimming larvae (miracidia) and enter an intermediate host, an aquatic or amphibious snail. After an asexual multiplicative phase, free-swimming larvae called cercariae are released from the snail. Cercariae encyst on vegetation as metacercariae which may survive adverse environmental conditions such as desiccation and freezing. The life cycle is completed when an ungulate ingests metacercaria encysted on forage.

In North America, the parasite has been reported from elk, moose, black-tailed (*Odocoileus hemionus columbianus*), mule (*Odocoileus hemionus*), and white-tailed deer, woodland caribou (*Rangifer tarandus*), bison (*Bison bison*), and cattle (*Bos taurus*). Infected animals have been found in British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, Quebec, Labrador, and Newfoundland. The parasite is common in white-tailed deer of the southeastern U.S. and also has been found in New York, Minnesota, Wisconsin, Michigan, Montana, Washington, and Oregon.

The parasite was imported into Europe in elk from North America in 1865. Ten years later, the fluke was officially described from elk in a Royal Hunting Reserve in Italy. It is often a fatal infection in the native roe deer and this species is now largely absent from areas in which the giant liver fluke occurs. Due to the possible recent migration of the parasite into BNP, the unprecedented infection levels, the potential effects of the parasite, and changes in ungulate populations and distributions, the giant liver fluke research project began in the spring of 1995. One objective was to determine the spatio-temporal distribution of the encysted larval stage (metacercaria) of the fluke.

The giant liver fluke rarely causes problems in white-tailed deer and caribou populations but can cause extensive damage in some individuals and has been known to directly and indirectly kill moose, elk, and black-tailed deer. Indirect mortality may result when the parasite debilitates ungulates, making them more prone to predation or collisions with vehicles. It is thought that the giant liver fluke may be influencing ungulate numbers in the lower Bow Valley of BNP. Moose can not cope with an infection of the giant liver fluke while most elk can. As a possible consequence, population levels of moose are very low throughout the Park while elk numbers are increasing near Banff townsite.

We began the search for the elusive metacercaria-in-the-marsh last year. Nearly 400 plants were collected and examined with a microscope for metacercariae. These plants were collected from six study sites that were visited tri-weekly from May to October. Low numbers of metacercariae prevented any detailed analyses. A new technique using plexiglass strips as a substrate for metacercariae was successfully pioneered to assess the distribution of metacercariae in the field. Two types of metacercariae were found on these strips placed in the field for three week periods at six study sites. One of the two types of metacercariae was tentatively identified as *Fascioloides magna* based on size and morphology. Significant differences were found in the prevalence, abundance, and density of metacercariae among study sites, suggesting that there are hotspots or foci of infection out in the marshes. Similarly, significant differences were found in the prevalence, abundance, and density of metacercariae among visits at one study site. These data strongly suggest that the bulk of cercariae are not shed until mid-September. As a result, ungulates probably do not become infected with the giant liver fluke until late fall. They may then continue to acquire infections throughout the winter.

In May 1996, a second field season began. It is hoped that a second year of data will reinforce conclusions reached last year. The most important aspect of the project so far is that the sentinel strip technique has proven invaluable in assessing the distribution of metacercariae in the field. (Dr. Lepitzki is an independent contract biologist who operates Wildlife Systems Research, Box 1311, Banff, Alberta, T0L 0C0. The giant liver fluke research project is funded by the Friends of Banff National Park and Parks Canada.)

## **Disease Updates**

### **Atlantic Region**

#### **Lungworm (*Crenosoma vulpis*) infection in wild canids on Prince Edward Island**

*Crenosoma vulpis* is a nematode (roundworm) parasite found in the bronchioles, bronchi and trachea primarily of foxes (red fox, gray fox, arctic fox), but also other canids (wolf, raccoon dog, domestic dog) and occasionally other carnivores (black bear, brown bear, marten, wolverine, badger). Red fox and coyote populations on Prince Edward Island have been surveyed for *C. vulpis* infection over the last three years. Results indicate a high rate of infection in the red fox (81.2-85.6%) - highest in young of the year (<1 year of age), which had an infection rate of 90% and a mean of 83.9 worms recovered from each animal (range = 0-483 worms), and lower in foxes over 1 year of age, which had a mean infection rate of 79.5% and a mean of 16.2 worms per animal (range = 0-156). The lower infection levels in older foxes probably reflects development of some level of host immunity with age and exposure to the parasite. It may also reflect some degree of death-loss among the most highly susceptible portion of the population.

In the 1993-94 season, 8.8% of the coyotes examined were found to be infected on P.E.I. A mean of 1.1 worms was recovered (range = 0-37) from each animal. *Crenosoma vulpis* infection has not been reported previously in coyotes. In 1994-95 an improved worm recovery technique was used which indicated an infection rate of 37.7% with a mean of 3.9 worms (range = 0-71) recovered from each coyote. *Crenosoma vulpis* infections have been diagnosed in domestic dogs on PEI and appear to be a significant cause of chronic canine respiratory disease.

*Crenosoma vulpis* has been reported in North America, Europe and China. In North America, *C. vulpis* is mainly restricted to northeastern regions (USA - New York; Canada - Nova Scotia, New Brunswick, Newfoundland, Ontario). Infection rates reported in red foxes have ranged from 17% (New York) to 50% (Nova Scotia, New Brunswick). Animals acquire the parasite by ingesting infected intermediate hosts (terrestrial snails and slugs). Infected larvae are digested free of the slug/snail tissue and migrate through the intestine to the liver and are carried in the blood to the lungs where they mature in the bronchial tree. In 19-21 days, the life cycle is completed with the production of eggs by mature females. The eggs hatch and the larvae are coughed up and swallowed. Terrestrial snails and slugs become infected while feeding on fecal matter containing the lungworm larvae. Adult worms have a life span of about 10 months in the lungs of an infected host. (Gary Conboy, Atlantic Veterinary College).

#### **Fatal infection with *Elaphostrongylus rangiferi* in Newfoundland caribou**

In February 1996, the Newfoundland Wildlife Division identified disease in several woodland caribou (*Rangifer tarandus terraenovae*) on the Avalon Peninsula of Newfoundland, approximately 120 km southwest of St. John's. Immature and adult animals of both sexes were found dead, or alive and exhibiting neurological signs. The live individuals were weak, carried their heads down, shook and staggered when walking, seemed unaware of their surroundings and showed no fear of humans. At necropsy, gross examination of affected caribou revealed that they were in very poor body condition, often had pinpoint hemorrhages scattered throughout their lungs and had variable numbers of *Dictyocaulus viviparus* (a nematode or roundworm) within airways of the lung. In a few animals, small numbers of hair-like nematodes, 4-5 cm in length, were found on the meninges of the brain. On microscopic examination, the meninges and brain were inflamed (meningoencephalitis) due to the presence of nematode larvae and eggs. The lungs had chronic inflammation centered on parasitic eggs embedded within the walls of the smallest airways (interstitial pneumonia). These eggs were in various stages of development consistent with a protostrongylid nematode.

Dr. M. Lankester, (CCWHC - Ontario Region) examined the adult parasites (3 males and 4 females) collected from the meninges and found their morphology to be compatible with previous descriptions of *Elaphostrongylus rangiferi*. This confirmed elaphostrongylosis as the cause of the neurological disease in these caribou. *Elaphostrongylus rangiferi* parasitizes the central nervous system (brain and spinal cord) and skeletal muscles of infected caribou and reindeer. Their life cycle is somewhat complex, utilizing an intermediate gastropod host. Sporadic outbreaks of neurological

disease in Scandinavian reindeer have been associated with this parasite. The problem occurs in late autumn or winter and appears to follow summers with above normal temperatures. The higher summer temperatures are believed to induce the development of large numbers of infective larvae in the intermediate gastropod host. In 1995, the mean summer temperatures were not above normal for Avalon Peninsula (Environment Canada, Atmospheric Environment Branch, St. John's, Newfoundland). However, the mean fall temperatures were slightly above normal and it is interesting to speculate that this may have contributed to the problem.

Newfoundland is the only location in North America where *E. rangiferi* is endemic. This parasite is believed to have been introduced into the island's caribou population when reindeer were shipped from Norway to Newfoundland by Sir Wilfred Grenfell in the early 1900's. It has been reported from other regions in Newfoundland but this is the first time the parasite has been identified in the isolated caribou population of the Avalon Peninsula. (S. McBurney, H. Whitney, M. Lankester and Newfoundland Wildlife Division).

## **Quebec Region**

### **Cnemidocoptic Mange in a Red-Winged Black Bird**

A young female red-winged black bird (*Agelaius phoeniceus*) was found in November 1995 with wing trauma and was housed until April 1996 at a wildlife rehabilitation centre with other passerine birds. Ultimately, it was euthanized and examined at necropsy. In addition to the wing injury, the bird had lesions typical of "scaly leg disease," or cnemidocoptic mange. Scale-like thickening of the skin was most prominent over the ankle (tarsometatarsal-phalangeal joint) and knee (tibiotarsal-tarsometatarsal joint) areas. Microscopic examination of scrapings from these areas revealed numerous mites identified as *Cnemidocoptes mutans*. Cnemidocoptic mange is a common disease of aviary birds and some poultry, and is occasionally described in wild birds. Typically, it causes thickening and overgrowth (hyperkeratosis) of the skin of the legs, and around the beak and eyelids. Occasionally, feathered portions of the skin are affected. It is not known whether this bird contracted the disease in the wild or in the rehabilitation centre. This case emphasizes that, to prevent the spread of such diseases, birds should be given thorough examinations prior to admission to rehabilitation centres and also prior to their release into the wild. (Igor Mikaelian, CCWHC-Quebec Region).

### **Tumours in Fish from Quebec Rivers**

The prevalence of tumours of the lips and the liver in some species of bottom-dwelling fish may serve as an index of the level of environmental contamination of their habitat. A study was carried out jointly by the CCWHC - Quebec Region and the Quebec Ministry of the Environment and Wildlife to evaluate the prevalence of tumours in white suckers (*Catostomus commersoni*) and brown bullheads (*Ictalurus nebulosus*) from the Richelieu

and Yamaska Rivers in Quebec. In white suckers, the prevalence of lip tumours was 0 (none in 101 fish examined) for the Yamaska River and .085 (12 in 141 fish examined) for the Richelieu River. This difference may be due to the difference in age-classes present in the two samples; only one fish greater than 400mm in length from the Yamaska River compared to 47 such fish from the Richelieu River. In general, older (longer) fish tend to have more tumours. Furthermore, the prevalence of lip tumours among fish from the Richelieu River is similar to that reported from areas considered "not polluted" elsewhere in North America. The prevalence of foci of liver cell alteration (a lesion thought to precede actual neoplasia) and of cholangiocarcinomas (a form of liver cancer) respectively was .02 and .01 in white suckers from the Yamaska River (in 101 fish examined). The prevalence of foci of liver cell alteration and of hepatic adenoma (another type of liver tumour) in white suckers from the Richelieu River was .014 and .007 respectively.

In brown bullheads from the Richelieu River, the prevalence of lip tumours was .35 ( in 34 fish examined) . Only one brown bullhead was obtained from the Yamaska River so there are no comparable data for the Yamaska. This prevalence of lip tumours is similar to that reported from waters considered "polluted" elsewhere in North America. The prevalence of foci of liver cell alteration in brown bullheads was .118 (in 34 fish examined). Future studies are planned to determine the prevalence of fish tumours in Quebec's major rivers. (Igor Mikaelian CCWHC - Quebec Region, and Yvon Richard, MEF).

## **Ontario Region**

### **Fatal trematode infection in migrating tundra swans**

This spring, there were several reports of dead tundra swans from localities in southwestern Ontario through which the swans migrate. The CCWHC lab in Guelph received swans through the Canadian Wildlife Service, Parks Canada and from Erie Wildlife Rescue, a wildlife rehabilitation centre. These birds had been retrieved from the Belle River area on the south shore of Lake St. Clair, and from the Hillman Marsh - Point Pelee area on Lake Erie. Five carcasses of an estimated two dozen deaths near Belle River were examined. These swans all had lesions of intestinal hemorrhage and inflammation due to infection with a trematode parasite, *Sphaeridiotrema globulus*. This parasite is acquired by ingestion of snails that act as the intermediate host. The flukes are small (approximately 1 mm in length) and often occur in clusters on the intestinal wall. They cause a fibrinohemorrhagic enteritis and death is due to acute severe blood loss. Relatively few flukes may be sufficient to cause death. In a previous report of death in a tundra swan due to this parasite (*Journal of Wildlife Diseases*, 19(4), 1983: 370-371), 107 flukes were recovered. In mute swans, as few as 20 flukes were found in instances of natural mortality and 6 in fatal experimental infections. In the one bird from this group in which a total worm count was conducted, 40 flukes were detected. The distribution of this parasite on the Great Lakes is not known. At CCWHC - Ontario, previous diagnoses

have been made in diving ducks during late winter and spring. A die-off of hundreds of ducks (greater scaup, bufflehead and green-winged teal) caused by this parasite occurred in Black Bay on the north shore of Lake Superior near Nipigon in the spring of 1990. *Sphaeridiotrema* also has been identified as the cause of death in mute swans from Lake Erie, east of Long Point and, this spring, in blue-winged teal from Big Creek National Wildlife Area, near Long Point. (Doug Campbell, CCWHC; John Haggeman and Jeffrey Robinson, CWS).

### **Overwintering mortality in Greater Scaup on the Niagara River**

In late February and early March, reports were received of hundreds of overwintering greater scaup dying on the Niagara River. Personnel from the Ontario Ministry of Natural Resources and from the Canadian Wildlife Service retrieved a number of carcasses and submitted them to the CCWHC in Guelph. A number of birds also were submitted to the New York State Department of Environmental Conservation. The birds received in Guelph were emaciated. Most weighed between 550 and 750 g (normal weight 850 - 1100 g), and had severe wasting of pectoral muscles. The majority were carrying moderate to large numbers of parasites, primarily cestodes (*Hymenolepididae*, *Diorchis* sp.) and trematodes (*Echinostomatidae*, *Echinostoma* sp.); however, not all birds were infected. No bacterial pathogens were detected but four of six birds had cadmium concentrations in kidney in excess of 120 ppm dry weight. Cadmium in kidney exceeded that in the liver in all birds tested, indicating chronic exposure. The scaup were likely acquiring the cadmium by ingestion of molluscs that acquired it from their substrate. With numerous industrial operations on the river, there are abundant sources of cadmium. The significance of these tissue levels is uncertain. Tissue concentrations in this range have been associated with disease and death in other avian species, but are lower than those found to be necessary in experimental exposures. These birds showed none of the renal lesions associated with cadmium toxicity. Enteric hemorrhage, present in these birds, has been described in cadmium toxicity, but is a fairly non-specific finding, and could have occurred here for other reasons. The exact cause of death of these birds remains undetermined. Prolonged negative energy balance was likely critical; the winter was relatively harsh, and few of the birds showed recent ingestion of food. Parasite burdens and heavy metal exposure may also have played a role in further debilitating these birds. (Doug Campbell, CCWHC; Ken Cornelisse, OMNR; Dave Ryckman, CWS).

### **Strychnine Poisoning in a Gray Wolf**

A young male wolf, one of a number of wolves under study by Dr. John Theberge of the University of Waterloo, was found dead near Round Lake, south of Algonquin Park, in February and was submitted to the CCWHC. The wolf was in excellent body condition. There was diffuse pulmonary hemorrhage and edema, with bloody fluid present in both thoracic and abdominal cavities. The stomach contained a mixture of fat, bones and some unidentified grey, friable material. The wolf was not rabid. Samples were sent to the toxicology laboratory at Michigan State University (MSU), where strychnine was identified by gas chromatography/mass spectrophotometry in the stomach contents. The Ontario Ministry of Natural Resources is investigating possible sources of the strychnine.

Protection of wolves and the effects of wolves on local deer populations are controversial issues in this part of Ontario. (Doug Campbell, CCWHC; Wilson Rumble, MSU; John Theberge, Univ. of Waterloo).

## **Western and Northern Regions**

### **A mortality event in Northern Fulmars in the Pacific Northwest**

During the last week of November and 1st week of December 1995, dead Northern Fulmars (*Fulmarus glacialis*) began washing ashore on beaches from Vancouver Island to Oregon. At the peak of this die-off, approximately 100 birds per linear mile (63 birds per km) were recovered from some beaches in Washington State. In B.C., most birds were collected from beaches near Tofino and Bamfield on the west coast of Vancouver Island. Although there was no attempt to systematically estimate the total magnitude of the die-off, information from ongoing beached bird surveys in B.C. indicated that this was an unusual mortality event for this species in the Pacific Northwest.

Ninety-two percent of the birds found were Northern Fulmars. The other seabirds found also were "offshore" birds such as a rhinoceros auklet, phalarope, kittiwake and albatross. No near-shore species, such as gulls or ducks, were involved. A small number of fulmars were found alive on the beach. These birds were weak, unable to stand, and emaciated. The sex ratio was approximately 1:1. Both young of the year and older birds were represented. Most showed signs of recent moulting (wing moult score average = 49).

All 52 birds examined by necropsy were emaciated and had no subcutaneous or intra-abdominal fat. Severe pectoral muscle atrophy was seen in 12% of the birds, mild atrophy in 80% and no atrophy in the remaining 8%. Three quarters of the birds had some degree of hemorrhagic material in the gastrointestinal tract. A small to moderate number of nematodes were found in the proventriculus of 25% of the birds. Nematodes also were found on histologic and gross examination of the intestinal tract of some of the birds. Foreign objects (primarily plastic beads and ceramic chips) were present in the ventriculus of 40% of the fulmars. No birds showed signs of recently ingesting any food.

Heavy metal analysis of 12 birds, lactic acid assays in 3 and brain acetylcholine evaluation in 3 birds failed to reveal any significant findings. Microbiological examination of 12 birds did not demonstrate bacterial or viral infections. No significant microscopic alterations were noted in 17 birds: all however, had been frozen, thus limiting interpretation.

Environmental conditions were assessed by reviewing 2 months of data from a weather buoy offshore of Bamfield. In the week preceding the die-off, surface water temperature increased by 1 C and there was a 8 C rise in air temperature. The wave height also increased during this time (average height 5m, maximum 12m). For the days on which birds were discovered on the beaches, the wind direction had changed to inshore. In the

month preceding the die-off, inshore winds were not associated with increases in water or air temperature. Severe winter storm activity was not noted until after the die-off. Anecdotal evidence suggests that fulmars were the predominant species residing off Vancouver Island this fall. Wildlife tour operators commented on the decreased number of seabird sightings in this area.

Ultimately, these birds died from emaciation and non-specific stress responses (as evidenced by the intestinal hemorrhage). The principle reason for the cluster of beached birds was likely the development of strong inshore winds bringing weak and dead birds onto beaches. Mass mortality of fulmars has been reported elsewhere and has been associated either with winter storms or increased water temperatures. Fulmars use sight and smell to locate prey and typically feed on cold water species. In some of the past die-offs, it was hypothesized that increased surface water temperature drove prey species deeper, rendering them inaccessible to the fulmars. The increased wave height seen in this case may also have decreased foraging success. The recent moult and parasite infections would have placed additional physiologic and nutritional stresses on these birds, thus rendering them less able to cope with decreased food availability. (Craig Stephen and Malcolm McAdie - Centre for Coastal Health; Ron Lewis and Vicki Bowes - Animal Health Centre - BC Ministry of Agriculture, Fisheries and Food).

### **Spongiform Encephalopathy in a Farmed Elk**

Chronic wasting disease (CWD), a spongiform encephalopathy of cervids, was diagnosed in a female, game-farmed elk by the Provincial Veterinary Lab, Saskatchewan Agriculture and Food, Regina, SK in December, 1995. This is the first case of CWD reported in a commercially farmed cervid and only the second case reported in Canada; the first occurred in a mule deer held at the Metropolitan Toronto Zoo which had originated from Colorado. Chronic wasting disease was first recognized in the late 1960's in deer held captive in a few wildlife research facilities in north-central Colorado and southeastern Wyoming. Since then the disease has been diagnosed in mule deer, elk and white-tailed deer in these facilities and in wild deer and elk within a 50 km radius of these facilities. This is the only known location where the disease is endemic.

Chronic wasting disease belongs to a group of diseases called transmissible spongiform encephalopathies which includes diseases such as bovine spongiform encephalopathy (BSE or "mad cow disease") of cattle, scrapie of sheep and goats, transmissible encephalopathy of farmed mink and Creutzfeldt-Jakob disease in humans. These diseases are characterized by a similar progressive degenerative process which causes vacuolation of brain tissue. In deer, the loss of brain function results in clinical signs of progressive weight loss, behavioral changes, gait abnormalities, hypersalivation, increased drinking and urination and ultimately death. There is no cure for this disease and no method of detecting infected animals other than by histological examination of brain tissue at post-mortem. The mode of transmission of CWD is unknown; however, evidence from Colorado and Wyoming would indicate that animal to animal transmission can occur and transmission from female to offspring is likely. Attempts to eradicate the disease in these facilities have been unsuccessful.

The agent responsible for this group of diseases is also unknown as no infectious agent has been visualized with electron microscopy and viral nucleic acids have not been detected. Three theories have evolved to explain these diseases. First, it has been hypothesized that the agent is a small virus which has yet to be detected by current techniques. Secondly, the virino theory hypothesizes that the spongiform encephalopathy agent consists of a small segment of nucleic acid which is too small to produce protein but instead has a regulatory function in brain cells which alters their normal function. The third and most widely supported theory is that the agent is a protein called a prion which infects the cell, altering the structure of the normal prion protein (PrP) in the cell causing it to accumulate. The accumulated PrP results in vacuolation and altered function of brain cells. If the prion theory is correct, this a new form of disease causing agent. Whatever the agent, it is small and unusually resistant to inactivation by heat, irradiation and chemicals.

Chronic wasting disease is not a reportable disease but is classified as exotic to Canada. Agriculture and Agri-Food Canada has, or is in the process, of destroying the herd from which this elk originated and all of its offspring have or will be destroyed. The likelihood that this disease was transmitted to wild deer or of it reoccurring in game ranched deer is very remote. However, if it did become established in either population there could be serious consequences for the game ranch industry and public perception of the safety of wild venison. This case highlights the importance of disease surveillance and diagnosis in wild and farmed animals. (Trent Bollinger, CCWHC Western/Northern Region. Reference: Williams, E.S and Young, S. Spongiform encephalopathies in cervidae. Rev. Sci. Tech. Int. Epiz., 1992, 11(2), 551-567.)

### **Botulism at Pakowki Lake**

It is not very often that we have an opportunity to predict the occurrence of a sizable wildlife disease outbreak. Unfortunately, in Alberta, we are in exactly that position. There is little doubt that the conditions are set for another outbreak of avian botulism at Pakowki Lake in southeastern Alberta (see CCWHC newsletters Vol. 3-1 and 3-3 for accounts of previous problems). Although water levels are likely to be somewhat higher (there was higher than average snow accumulation in the mountains and considerable late winter precipitation throughout Alberta this year), the basin is extremely flat and the added water probably will result in more rather than fewer mudflats and shallow shores. The Contingency Plan for Disease Cleanup at Pakowki Lake has been revised and plans are well under way to identify staff and equipment needs as well as to draw up a specific approach to attack the problem. Surveillance visits started in early May and the lake will be monitored closely so that we may begin cleanup as soon as there is evidence of bird losses. A cleanup coordinator, co-funded by Environment Canada, Ducks Unlimited Canada, North American Waterfowl Management Plan, and Alberta Natural Resources, has been hired and will direct the troops and battle plans in the field. All agencies are committed to trying to reduce losses and minimize the impact on avian populations at the lake. In addition, various research projects will provide information useful to determining

long-term management plans for Pakowki. Data concerning avian populations, water quality, and the effects of noise-making deterrent devices will be collected. The goal is to lay a firmer foundation for future management options. The Pakowki Lake Working Group, with representatives from various wildlife and water management agencies, is working to develop recommendations for senior managers by the fall of 1996. (M.J. Pybus, Alberta Natural Resources and Chairman, Pakowki Lake Working Group).

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