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CCWHC News

CCWHC Newsletter on the World Wide Web

This Newsletter, English version, now is available on the Internet at the ProMed/AHEAD web site. The address is http://www.fas.org/ahead/. Click on the CCWHC logo for access to the Newsletter. ProMed is a project of the Federation of American Scientists to monitor and track emerging diseases and AHEAD is its animal health subsection. The CCWHC is a cooperator in the ProMed program.

Five-Year Review of the CCWHC

As of August 1997, the CCWHC will have been in operation for five years. In preparation for a second five-year mandate, the directors and regional coordinators prepared a written review of the first five years. This review was published in the May issue of the Canadian Veterinary Journal (Leighton, F. A., et al. 1997. The Canadian Cooperative Wildlife Health Centre and surveillance of wild animal diseases in Canada. Canadian Veterinary Journal 38: 279-284). Copies of this review are available from the CCWHC Headquarters Office.

Centre for Coastal Health moves to Nanaimo

The Centre for Coastal Health (CCH,) has moved to the campus of Malaspina College in Nanaimo, on Vancouver Island. The CCH undertakes research and educational activities focussed on health in and of the coastal ecosystem, and is the on-site west coast cooperator of the CCWHC. Contact Information: Craig Stephen (Director), Centre for Coastal Health, Faculty of Science and Technology, Malaspina University-College, 900 5th St., Nanaimo, BC V9R 5S5. Tel.: 250-753-3245 (local 2642); FAX: 250-755-8749.

International News

Newcastle Disease in Cormorants, California, USA - May 1997

Newcastle Disease (ND) has once again occurred among Double-Crested Cormorants (DCC) in North America. During the week of 12 May, personnel at the Salton Sea National Wildlife Refuge, located approximately 150 kilometres east of San Diego, reported finding approximately 1600 dead nestlings and fledglings in a colony of DCC and Caspian Terns. Most of the dead were DCC. An investigative team from the National Wildlife Health Centre (NWHC - U.S. Geological Survey, Madison, WI) secured samples for diagnostic examination and noted some young-of-the-year DCC at the colony with unilateral wing or leg paralysis. NWHC pathologists found lesions in the brain typical of ND, and ND virus was isolated from the affected tissues. Readers will recall that ND occurred in DCC in Canada in 1990, 1992, 1995 and possibly 1996 (see Atlantic Region, this issue); the only previously recognized occurrence was among DCC in the St. Lawrence estuary in 1975. Until this California outbreak, all previously recognized occurrences have been in DCC populations east of the Rocky Mountains, although the CCWHC found antibodies to ND virus in eggs taken from DCC nests in British Columbia in 1993. Although there may be some migration over the mountains, the DCC populations of the west coast are thought generally to be separate from populations that nest east of the Rockies. ND now has occurred in DCC across the full east-west breadth of the species' range in North America and at least four times between 1990 and 1997. All virus strains isolated and tested thus far have proven to be highly pathogenic to domestic poultry. Thus, there are important biological, economic and regulatory reasons to establish collaborative research programs aimed at a complete understanding of the ND virus in wild bird populations in North America.

Feature Articles

Chronic Wasting Disease of Wild Deer

Chronic Wasting Disease is a fatal disease known to occur among wild deer in only one small geographic area of the United States. It belongs to the group of diseases known as Transmissible Spongiform Encephalopathies (TSE's). Considerable public interest and anxiety have arisen about TSE's over the past few years, stimulated largely by the emergence of Bovine Spongiform Encephalopathy (BSE), also sometimes referred to as "mad cow disease" - another TSE. Creutzfeld-Jacob Disease (CJD) of people, and Scrapie of sheep and goats also are TSE's. Public anxiety about these diseases is understandable and justified. These are terrible diseases in which people and animals suffer mental and physical deterioration over long periods of time and then inevitably die. Compared to many other diseases, very little is known about the TSE's. Their cause(es) is/are enigmatic. It is not known with certainty whether or not people can acquire CJD from animals with TSE's, but there is considerable suspicion that this may be the case

with BSE. Scientists issue contradictory statements on this issue because the evidence available can be interpreted in different ways. Hard proof of anything about the TSE's is elusive. Into this climate of anxiety and uncertainty have tumbled popular books depicting the TSE's as the major human health threat now and in the future, and government advisories admonishing the public to eat, or not to eat, meat according to the politics and culture of the particular ministry. There is no easy resolution of this confusing situation. Wildlife personnel should be as informed as possible about Chronic Wasting Disease, but also must live with the fact that all aspects of this and the other TSE's currently are plagued with uncertainty.

Chronic Wasting Disease (CWD) is known to affect Mule Deer, Elk (Wapiti) and White-tailed Deer. It was first recognized as a disease in a herd of captive Mule Deer and Mule Deer hybrids at Fort Collins, Colorado in 1967 and was determined to be a form of TSE in 1977. It subsequently was diagnosed in other captive deer and elk in the Colorado facility and in a research facility in neighbouring Wyoming. CWD has been recognized in free-ranging wild deer since 1981. From that year to June 1995, the disease was diagnosed in 41 Mule Deer, 6 Elk and 2 White-tailed Deer, all from free-ranging populations in north-central Colorado within a 100 km radius of Fort Collins. A total of 11 diseased wild animals of the same three species have been found in an adjacent area of southeastern Wyoming. In Canada, the disease has never been identified in wild deer, but two cases have occurred in captive deer: in 1978 in a Mule Deer at the Metro Toronto Zoo and in 1996 in an elk imported from the United States and held on farms in Saskatchewan. No further cases have occurred at the zoo and a comprehensive eradication program was undertaken by Agriculture and Agri-Food Canada in cooperation with the game farming industry with respect to the case in Saskatchewan.

CWD gets its name from the progressive emaciation, or wasting away, that occurs in affected animals. Abnormal behaviour is the other central feature of the disease. Affected animals are weak and have an odd gait, reduced fear of humans, drooped heads and ears, and excess salivation. There are no gross abnormalities except emaciation. Diagnosis is based on microscopic changes in the brain. Nerve cells and the brain substance around them develop large, clear vacuoles that give the tissue the appearance of a sponge with its many holes - hence the name "spongiform" encephalopathy - literally a spongy abnormality of the brain. These vacuoles develop in association with accumulations of a special protein found only in TSE-affected brains. This protein can be identified by special techniques, and its presence together with the vacuoles is generally accepted as the ultimate criterion for making a diagnosis.

The causal agents of the TSE's are transmissible from infected to uninfected individuals. In general, there is a very long interval, usually a large proportion of the normal lifespan of the animal species involved, between exposure to the causal agent and development of disease. Thus, it is very difficult to study transmission of these diseases, and relatively little is known about it. Eating TSE-infected tissue is one way to acquire the disease. The most famous form of CJD, the human version of these diseases, was studied in the Fore people in New Guinea, among whom the disease, called Kuru, was transmitted by ritual cannibalism. The sudden emergence of BSE in Europe has been linked to feeding cattle

with meat meal, presumably contaminated by a TSE-causing agent from an animal source. Various of the TSE's have been transmitted to species in which they do not regularly occur by feeding the brains of affected animals. Thus, quite aside from the loss of the animals themselves, the TSE's, and particularly BSE, have become a major issue of public health and food safety. Has CJD been caused in people from eating parts of cattle affected with BSE? Is it safe to eat the flesh of deer that might carry the agent of CWD?

Unfortunately, the answers to these questions are not clear at all. CJD occurs spontaneously in people world-wide at the rate of about one case per million people per year. A considerable increase in this low rate of occurrence would be necessary in order to be certain, statistically, that the incidence is indeed rising. And, because there can be decades between exposure to the causal agent and development of the disease, exposure of people to the agent in the 1980's might not result in disease until the 1990's or 2000's. How should society respond to this situation? Some feel the health risk to people who eat TSE-affected animals is high and that drastic and immediate actions are called for to prevent any possibility of this occurring. Others consider this an alarmist view based on unfounded assumptions and insufficient information.

What causes CWD and the other TSE's? The current answer to this question is in the grey zone between knowledge and theory. It appears too early to be sure whether we now have the entire story or only a partial answer. Current understanding of the cause of the TSE's rests on the concept of the prion ("prèe-on") protein. As disease-causing agents, prion proteins fall somewhere between our usual notions of infectious agents and of toxins. They are abnormal versions of a normal body protein that accumulate in cells because of two properties: they propagate themselves by changing the normal proteins into abnormal forms whenever they encounter them, and they can not be broken down and removed by the body. Accumulation of these abnormal proteins in nerve cells appears to account for the brain dysfunction that characterizes the TSE's. These abnormal prion proteins are remarkably hardy. They are not destroyed by cooking, formaldehyde, alcohol or ultraviolet light. They can be inactivated by strong sodium hydroxide (lye) , household chlorine bleach (undiluted) and autoclaving at 132C for 4.5 hours. The spontaneous cases of CJD that affect one in a million people each year are thought to arise by chance spontaneous conversion of some normal prion protein to the abnormal form, which then propagates itself and accumulates. Ingestion of a quantity of abnormal prion protein, as found in the brains of affected animals, is thought to result in absorption of a sufficient amount to initiate the slow process of self-propagation and accumulation, culminating in disease.

Wildlife personnel must respond to the legitimate public concerns about CWD without the benefit of much firm information. No one knows whether or not CWD poses any risk to human health or to domestic animal health, or if it is a significant threat to wild deer populations. Its mode and rate of transmission are unknown, as is its relationship to other TSE's such as Scrapie and CJD (BSE does not occur in North America). In Colorado and Wyoming, major surveillance programs for CWD are underway; the results may help define the geographic extent of the disease and other aspects of its biology. Even in the

affected area of north-central Colorado, the prevalence of affected animals appears relatively low, being highest among mature male Mule Deer with about 6% of animals affected. Both Saskatchewan and Alberta have initiated limited surveys for CWD based on available specimens, but quite large samples will be needed to determine the prevalence of a disease that probably does not occur or at least is very rare. Dr. Beth Williams, University of Wyoming, has established a battery of tests for detection of CWD and the application of these tests to large samples of animals will help determine the best methods for detection and their limitations. We are at a very early stage in our understanding of this disease. We must communicate this honestly to the public and enlist its support for the research necessary to learn enough about this disease to respond to it wisely. (For more detailed information, readers are directed to the following publication and references cited therein: Spraker, T.R. et al. 1997. Spongiform encephalopathy in mule deer, white-tailed deer and Rocky Mountain elk in north central Colorado. Journal of Wildlife Diseases 33: 1-6.) (Ted Leighton, CCWHC Headquarters Office).

Disease Updates

Atlantic Region

Salmonellosis in Herons, Gannet

In early fall 1996, salmonellosis was diagnosed in three immature great blue herons (Ardea herodias) and one immature northern gannet (Sula bassanus) from widely dispersed locations on PEI. In all these birds, there was severe necrosis of the intestinal lining, and Salmonella species was isolated from several internal organs. Although we have been following causes of mortality in these two species for several years, these were the first cases of salmonellosis identified in either species at this centre. (P-Y Daoust and S. McBurney, CCWHC - Atlantic region)

Probable Newcastle Disease in a Cormorant

An immature double-crested cormorant (Phalacrocorax auritus) exhibiting neurological signs was found close to Shediac, on the north shore of New Brunswick, in October 1996. It had microscopic evidence of a nonsuppurative encephalitis, and an immunohistochemical test on its brain demonstrated the presence of viral antigens belonging to paramyxovirus type 1, the Newcastle Disease (ND) group. Unfortunately, no virus could be isolated from this bird, so the strain of virus could not be determined. In 1995, ND virus (i.e. with moderate pathogenicity for poultry) was isolated from another immature double-crested cormorant found unable to fly on the north shore of PEI. These and a few other cases over the past several years suggest a pattern of sporadic occurrence of the disease in the Atlantic region. (P-Y Daoust and S. McBurney, CCWHC - Atlantic Region)

Neoplasm in Eiders

In January 1997, an immature common eider (Somateria mollissima) shot on the southern coast of Labrador was submitted for necropsy because the hunter had noticed white lumps on its wings and breast. The lumps were multiple, pale yellow masses that ranged in greatest width from 0.5 to 4.0 cm. They were most numerous within breast and wing muscle, but also were found in the lungs, heart, ribs, kidneys, sciatic nerves, and testes. The bird was otherwise in good body condition. Microscopically, the masses were tumors comprised of a mixture of fibrous and fatty tissues. A similar condition has been described previously as "multicentric lipomatosis/fibromatosis" in wild geese. The cause is unknown. This is the first known occurrence of this condition in a duck of any species. Reference: (Multicentric intramuscular lipomatosis/fibromatosis in free-flying white-fronted and Canada geese. P.-Y. Daoust, G. Wobeser, D.J. Rainnie, and F.A. Leighton, J.Wildl.Dis. 27(1), 1991, pp 135-139).

Birthing Death in a Harbour Porpoise

In June 1996, an adult female harbour porpoise (Phocoena phocoena) in good body condition, bearing a full-term fetus, was found dead on the south shore of PEI. Various observations, including a dilated vulva, the very decomposed state of the fetus, and microscopic evidence of inflammation in the wall of the uterus, suggested that this female had died as a result of failure to deliver her calf (dystocia). This is the second case of dystocia diagnosed at this centre out of a total of 16 harbour porpoises of various ages and of both sexes examined since 1988. (P-Y Daoust and S. McBurney, CCWHC-Atlantic region)

Québec Region

Low Prevalence of Neoplasia in St. Lawrence River Eels

American eels (Anguilla rostrata) spend their adult lives in the rivers and lakes of the North Atlantic basin. After many years in this habitat, they migrate downstream in late summer or early fall and travel to the Sargasso Sea, an area of the Atlantic east of the Caribbean, to spawn. Eels from Ontario lakes and other tributaries have high levels of chemical pollutants in their tissues. Despite this high exposure to contaminants that can cause cancer in other fish species and in experimental rodents, cancer does not appear to be common in these eels.

Although there has been a sharp decline in eel populations throughout the range of the species, there remains a substantial commercial fishery in the St. Lawrence. Thus, many fish are handled and observed annually. Fish with abnormalities occasionally are submitted to diagnostic laboratories, and others are examined in the field by scientists. No external tumours were seen in 343 eels thus examined in 1994 and 1995. In 1996, two eels were submitted for diagnosis. One, submitted by a commercial fisherman, had a

cauliflower-like mass, 1.5 cm in diameter, on the lip of the lower jaw which turned out to be a true cancer (squamous cell carcinoma). The second, from a group of 1081 eels examined during a population study, had a gray mass on its stomach wall that tuned out to be a benign tumour derived from muscle (leiomyoma). High prevalences of lip tumours have occurred in brown bullheads (Ictalurus nebulosus) and white suckers (Catostomus commersoni) in association with chemical contaminants in sediments, but eels in the same habitats have not been similarly affected. Our observation of a low prevalence of tumours in St. Lawrence eels in consistent with these previous observations at other locations. (Igor Mikaelian, Daniel Martineau - CCWHC Quebec Region)

Mortality at a Bird Feeder

An ornithologist in the Hudson region who kept many bird feeders in the garden noted mortality among house sparrows at the rate of 1-2 per week during the winter of 1996-97. Eight of the birds were frozen and submitted to the laboratory at the end of the winter. Six of the birds had died of salmonellosis, one of avian pox and one of trauma. These results show that mortality at bird feeders can have various causes. The predominance of salmonellosis underscores the need regularly to disinfect bird feeders and to remove feed remnants contaminated with feces in order to reduce transmission of the disease among birds at the feeders. Salmonellosis can affect humans and the public should be aware of this health hazard in order to take reasonable sanitary precautions to prevent acquiring infection.[Igor Mikaelian, Daniel Martineau (CCWHC) and Lyse Sylvestre (Le Nichoire)]

Strychnine Poisoning of a Peregrine Falcon

A peregrine falcon that had been releasedfrom a captive breeding and reintroduction program was found dead on the roof of a building in the city of Hull. A freshly dead and half-eaten pigeon (rock dove) was found beside the falcon. At necropsy, the falcon was found tobe in good nutritional condition and its crop was filled with pigeon flesh. Toxicologic evaluation of the crop content detected strychnine. Thus, the falcon died of secondary strychnine poisoning from eating a pigeon that had been poisoned. In Canada, use of strychnine to kill pigeons is permitted. A search of the CCWHC (Québec Region) records found three previous cases of strychnine poisoning: one case involving 31 common grackles and two involving one pigeon each. All these poisonings occurred in urban areas. It is our view that the use of pesticides that pose a high risk to non-target species should be discontinued. In Europe, strychnine is recognized as a highly dangerous product and its use is prohibited. [Igor Mikaelian, Daniel Martineau (CCWHC) and Daniel Saint Hilaire (MEF-Hull)].

Ontario Region

Suspected Teflon Toxicosis in Songbirds

In August of 1996, the Ontario regional office of the CCWHC was contacted by the City of Scarborough Department of Public Health with a request for help in responding to a perceived problem of emissions from an industrial operation in which cookware is coated with teflon (polytetrafluoroethylene or PTFE). Residents of an adjacent neighbourhood were concerned about affects on air quality and health, and cited as evidence an unusual number of birds found dead in backyards located close to the plant. A number of birds found dead in this neighbourhood from July to October were retrieved and submitted to the CCWHC for necropsy. These included house sparrows, yellow-bellied sapsuckers, starlings and mourning doves. All birds had been frozen prior to submission. Some weeks later, a second submission, consisting entirely of house sparrows, the carcasses of which had been opened and placed in formalin on site, was received. In all instances, freezing artifact or autolysis limited pathological interpretations. However, a consistent pattern of pulmonary congestion, edema and occasional hemorrhage was present in these birds, in the absence of lesions of trauma or infectious disease. These lesions were consistent with death due to inhalation of PTFE fumes. It was not possible to be more definitive.

Following an evaluation of the available evidence, including stack emissions, wind direction, weather conditions, and circumstantial evidence such as the death of these birds, the company, Scarborough Department of Public Health, Ontario Ministry of the Environment, and the residents negotiated a course of action aimed at reducing emissions and their potential effects on the neighbourhood.

PTFE toxicosis is often suspected in sudden deaths of caged birds, particularly where the circumstantial evidence includes a possible source of combustion products. However, a definitive diagnosis is rarely reached because of the relatively non-specific nature of the gross and light microscopic lesions. In experimental exposures of budgerigars to the products of PTFE combustion. (American Journal of Veterinary Research, 1982, 43: 1238-1242, 1243-1248), there was extensive pulmonary congestion and hemorrhage. Microscopically, there was necrosis and hemorrhage in the lung, as well as changes in the airways. PTFE combustion products are very toxic to rodents, causing pulmonary edema and hemorrhage. In humans, exposure to PTFE fumes can result in an illness known as polymer fume fever. (Fundamental and Applied Toxicology, 1991, 17: 254-269). (D. Campbell and I. Barker, Ontario region - CCWHC).

Suspected Avitrol Poisoning of Non-target Species

Avitrol (4-aminopyridine) is an agent used in the control of nuisance species of birds, particularly in urban areas. The compound is delivered in bait, usually kernels of corn. It affects the nervous system in a manner similar to organophosphate and carbamate insecticides. In sufficiently high doses, it is lethal, while at lower doses, it causes disorientation and the emission of distress calls, which lead to the dispersal of other birds of the same species. Concerns have been raised over the possibility of poisoning of non-

target species that consume either the bait or the carcasses of birds that have died as the result of avitrol poisoning. Of particular concern is the possibility of inadvertent poisoning of raptors such as peregrine falcons that have been reintroduced to urban areas.

The CCWHC Ontario Regional Centre received a submission that consisted of a single dead pigeon and the crop contents of a second bird from a person whose dog had found a number of dead pigeons, and was thought to have consumed some of the birds. Within hours of this event, the dog began to salivate excessively, developed diarrhea and eventually entered respiratory distress. It was taken to an emergency clinic, but died in spite of treatment. A necropsy was conducted on-site. No significant lesions were noted, and no tissues were saved for further examination and analysis. Concerned that the dog may have been poisoned by consuming the pigeons, the owner brought the two samples from the birds to the CCWHC. Avitrol was detected in the crop contents, which consisted of corn kernels.

There is, of course, no way of determining whether the dog died as the result of avitrol poisoning, but the circumstances suggest that possibility. Mammals are as susceptible to avitrol poisoning as are birds; a potentially lethal dose is generally less than 10 mg/kg (Toxicology and Applied Pharmacology 1973, 26:532-538). The crop contents of the pigeons contained 34-43 μg/g (ppm) of avitrol. A review of cases of avitrol poisoning previously diagnosed at this laboratory found a range of avitrol concentrations in crop contents of 12-480 ppm. Liver concentrations are much lower, and never exceeded 10 ppm. At these concentrations, it would be difficult for a large dog to consume sufficient amounts of bait or poisoned birds to be fatally poisoned. The dog would have to consume undiluted or poorly mixed avitrol-laced bait in order to consume a lethal dose. Similar considerations apply to raptors consuming pigeons. A raptor would need to eat the crop contents of a poisoned pigeon in order to encounter a lethal quantity of avitrol. Tissue concentrations are sufficiently low that poisoning from this source is unlikely.

Avitrol is a compound that is capable of affecting a wide range of species in addition to those against which its use is directed. This increases the need for those using it to apply it in strict conformity to the manufacturer's directions with regard to dilution and placement of baits. (D. Campbell and I. Barker, Ontario region - CCWHC).

Northern and Western Region

Forensic (Medico-legal) Examinations

Examination of dead animals to collect information that can be used as evidence in court has become a service requested with increased frequency at the Western/Northern Regional Centre. Prior to 1996, only a few such cases were submitted each year. During 1996 and the first five months of 1997, 33 forensic submissions were received, consisting of 56 white-tailed and 9 mule deer, 30 coyotes, 3 American coots, 3 white pelicans, 3 red foxes, and one each of moose, raven, 1 raccoon, red-winged blackbird, snowy owl, great

grey owl, Swainson's hawk, eared grebe, and a bovine calf (the latter was a poison bait). Most cases involved animals shot illegally, run down illegally with snowmobiles (coyotes, foxes), or suspected to have been intentionally poisoned. All the cases were submitted by wildlife enforcement officers.

Although the basic techniques used are the same, forensic examinations require much greater input than the average non-legal case involving the same species. We estimate that an "ordinary" necropsy requires about 20% of the time required for a comparable forensic case. Much of this extra input is in establishing and maintaining a continuous chain of custody of the animals and specimens that result from the examination, recording observations in great detail both photographically and in writing, and in collecting, preparing and returning evidence to the submitting official, or sending it to other laboratories for specialized testing.

The role of the pathologist in all forensic cases is to provide an objective, unbiased appraisal of the evidence. In many cases, the service is useful to the submitting enforcement officer. Evidence, such as bullets recovered from a carcass, demonstration of a poison in both a bait and a dead animal, or positive linkage of specimens by DNA technology, often helps to convince violators to plead guilty. In other instances, the information gathered has exonerated suspects. Most cases have not required that a CCWHC pathologist appear in court, which is fortunate, because appearance as an expert witness is very time-consuming.

Mortality of Porpoises in British Columbia

During May 1997, 18 porpoises were found dead on beaches or adjacent waters of southern Vancouver Island. Affected animals included both Dall's and Harbour Porpoises, estimated at 2-4 years of age and equally represented by males and females. Routine stranded marine mammal surveillance has detected only 3-4 dead porpoises in the same area and time frame each year over the past 10 years, with the exception of a mortality event involving some 30 animals over a six week period in 1993. Four animals were examined at necropsy by a veterinarian and eight others were examined grossly by stranding network personnel. There were no consistent pathological findings among the animals to indicate a single cause of mortality. Two had pancreatitis, possibly associated with trematodes (flukes) in the pancreatic duct. Two had significant inflammation of the chest cavity (fibrinous pleuritis) and one had a large thrombus (pathological blood clot) in the pulmonary vein. There was no evidence or subsequent reports of mortality of other marine animals in the area and no algal blooms, contaminant spills or other potentially causal events are known to have occurred in the area during or preceding the mortality event. (Craig Stephen - Centre for Coastal Health; Ron Lewis - Animal Health Centre, Ministry of Agriculture, Fisheries and Food).

Predator Attacks in British Columbia

There were 12 reported attacks on humans by bears and cougars in British Columbia during 1996.

Predator Attacks on Humans in British Columbia, 1996		
Predator Species	Human Injuries	
	Injured	**Killed**
Cougar	4	1
Grizzly Bear	4	0
Black Bear	2	1

The wildlife branch is very concerned by these statistics. Response plans include a continued refinement of waste disposal systems, and ongoing research on animal inventory and census techniques, relocation success and population dynamics of large carnivores. In addition, an improved incident investigation and necropsy protocol has been developed to standardize procedures and maximize data collection. (Helen M. Schwantje, Wildlife Veterinarian, Wildlife Branch, BC Ministry of Environment, Lands and Parks).

[Wildlife personnel often are called upon to participate in investigations of predator attacks. A key element often is linkage of the attack to an individual predator animal. The forensic techniques required for this are highly specialized: for example matching blood from the claws of the animal with that of the victim. Crucial information can be lost when the animal is improperly handled. Wildlife personnel are advised to seek full advice and participation from appropriate police, medical and veterinary personnel before proceeding with such investigations. Ed.]

Spring Botulism

The traditional picture of botulism in waterfowl is a large scale die-off of dabbling ducks that begins during the hot "dog-days" of late summer and continues until the weather cools in autumn. Another form of botulism, that is recognized less frequently, occurs early the following spring on some of these wetlands. The unusual features of these spring outbreaks are that they begin very early while the weather is still very cool (some occur immediately after the ice thaws), the birds involved are diving species rather than dabbling ducks, and the outbreaks usually are short-lived and end spontaneously. It has been suspected that diving birds were finding toxin, formed during the previous summer outbreak, that persisted over the winter on the bottom of wetlands. Two important pieces of research have confirmed that type C botulinum toxin is stable for extended periods of time under cool conditions (Hubalek and Halouzka, 1988) and that toxin-bearing maggots could remain intact, although dead, in mud at the bottom of wetlands for several months

under winter conditions and still contain sufficient toxin so that about 10 such maggots would kill a duck (Hubalek and Halouzka, 1991). The reason that these spring occurrences end is likely related to the generally cool conditions so that fly activity is limited and most carcasses are removed by scavengers before the carcass-maggot cycle has a chance to become established. Spring outbreaks have been recognized on several wetlands in Saskatchewan (Wobeser et al., 1983) and diving ducks (primarily Lesser Scaup) and American coots were found dead of botulism on Whitewater Lake in Manitoba during early May of this year. (Whitewater Lake had a massive outbreak during the summer of 1996). For further information: Hubalek, Z. and J. Halouzka, 1988. Thermal stability of Clostridium botulinum type C toxin. Epidemiol. Infect. 101:321; Hubalek, Z. and J. Halouzka, 1991. Persistence of Clostridium botulinum type C toxin in blow fly (Calliphoridae) larvae as a possible cause of avian botulism in spring. J. Wildl. Dis. 27:81; Wobeser, G. et al., 1983. Avian botulism during late autumn and early spring in Saskatchewan. J. Wildl. Dis. 19:90.

Summer Botulism:

Botulism in small numbers of dabbling ducks and other species has been detected at Whitewater Lake (Manitoba) and Old Wives Lake (Saskatchewan) in late June. These lakes and Pakowki Lake (Alberta) are being closely monitored in a cooperative effort involving many agencies interested in waterfowl. All have been sites of large-scale outbreaks in recent years.

Online @ http://wildlife1.usask.ca/newsletters/newsletter4-3.htm