January 1994

Canadian Cooperative Wildlife Health Centre, Volume 3-1, Winter 1994/1995

Canadian Cooperative Wildlife Health Centre

Centre Canadien Coopératif de la Santé de la Faune

Newsletter 3 - 1, Winter 1994/1995

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Feature Articles

Ecology of Waterfowl Botulism

Botulism, one of the most important diseases of wild waterfowl, has a complex life history. Botulism is a form of food poisoning caused by a potent toxin produced by the bacterium Clostridium botulinum. Birds are poisoned by eating material containing this toxin. There are several strains of C. botulinum; the one responsible for most waterfowl deaths is called Type C. Another strain, Type E, causes botulism in fish-eating birds. Clostridium botulinum occurs as a resistant spore or resting stage and as a vegetative or growing stage. Only the vegetative stage produces toxin.

In many ways, the ecology of waterfowl botulism is analogous to fire in a forest. Both require certain critical factors to begin, while the magnitude of the resulting occurrence is determined by other factors. Both occur in many forms: small, short-lived flare-ups that end spontaneously; smoldering episodes; or massive outbreaks. The exact "spark" that starts both fires and botulism outbreaks is often unknown. Factors needed to start a fire are a spark and fuel; the size of the resulting blaze is determined by the amount and type of fuel, as well as wind, humidity and other environmental features. Factors needed for botulism to occur are: (1) the bacterium, (2) substrate in which it can grow and produce toxin, and (3) birds to eat the toxic material. Spores of C. botulinum are common in marsh soil, particularly where outbreaks have occurred in the past, so the bacterium is available in many marshes. The ideal substrate for growth of the toxin-producing vegetative form of the bacterium is decomposing organic matter. Decaying plant material is not a good substrate, but animal carcasses (invertebrate or vertebrate) are ideal.

Animals that live in botulism-prone marshes consume spores regularly; however, spores "germinate" to the vegetative stage only in an anaerobic (devoid of oxygen) environment, so spores do not germinate in living animals. When an animal dies, its tissues become anaerobic, and spores within the digestive tract may grow and produce toxin. Thus, any animal dying in a marsh where C. botulinum spores occur is a potential source of toxin. Invertebrates, killed by factors such as desiccation, high salinity or water temperature, might act as a microenvironment for toxin formation; these dead invertebrates could be consumed by birds. However, it is unlikely that dead invertebrates are the "spark" that starts most outbreaks. Evidence is much stronger that vertebrate (bird, mammal, fish) carcasses are the initial source of toxin. In addition to abundant nutrients and an anaerobic environment, vertebrate carcasses also provide the optimal temperature for toxin production. Toxin production by C. botulinum type C is maximal at about 37C; temperatures this high occur in decomposing carcasses as a result of the metabolic activity of maggots and bacteria. Large amounts of toxin may form in carcasses within 3 to 5 days, about the same time required for maggot growth.

Ducks do not scavenge carcasses but they do eat invertebrates, including fly maggots, and as few as 4 or 5 maggots floating from a carcass, or small numbers of other

scavenging and toxin-bearing invertebrates, may contain sufficient toxin to poison an adult duck. The carcass of each bird that dies of botulism then can become another incubator for production of toxin and maggots, potentially perpetuating and expanding the outbreak. Animals die continuously in nature and one might expect botulism outbreaks to occur each year in marshes where spores are present. However, in the same way that not every cigarette butt results in a forest fire, conditions are not always appropriate for botulism. A critical factor seems to be the amount of substrate available for toxin production. Under normal circumstances, most animals that die in a marsh are consumed rapidly by scavengers, so that little substrate remains for toxin production. (Studies in areas as diverse as Texas and France suggest that the average life-span of a duck carcass is about 2 days, while toxin and maggots are not available for 3 to 5 days). Some carcasses may persist until toxin forms but the toxin may never reach other birds, and if only a few birds consume toxin and die, the outbreak may not continue to expand. It is likely that in some marshes a few birds die of botulism in most years. For example, occasional birds sick or dead of botulism can be found almost every year in one marsh in Saskatchewan, but major outbreaks have occurred there only about once or twice per decade. If many carcasses are present, the scavengers may be "overloaded" so that most carcasses persist and many toxin-laden maggots are available to birds. The initial factor responsible for death of the animals seems unimportant; outbreaks have started from birds dying after collision with overhead wires, blue-green algae poisoning, and hail storms, all of which provided abundant dead birds to act as substrate. Outbreaks have also been associated with fish kills and even with the presence of a cow carcass in a marsh! Weather conditions favourable for blowflies and, hence, maggots, and dense aggregations of susceptible birds in the area all favour occurrence of an outbreak.

<u>How Botulism Outbreaks may be Regulated</u>

The following simple **model** illustrates how these factors might interact. All of the numerical values used in this example are made-up and are used simply to show how disease outbreaks may be regulated. Let us assume that in a hypothetical marsh: - 40 % of animals have C. botulinum spores within their body (probability [p] = .40). - 90 % of carcasses become infested by blowfly maggots (p = .90) - 10% of carcasses remain unscavenged until toxin forms and toxin-bearing maggots emerge (p = .10) In this marsh, the probability that a single carcass will produce toxin-bearing maggots = .40 × .90 × .10 = 0.036 or, in other words, <4% of animals that die in the marsh are likely to produce toxin-bearing maggots. For the disease to be perpetuated, it is necessary also to factor in: - the probability that sufficient maggots will be found and consumed by a bird to result in death (p = .10), - the probability that this new bird's carcass will persist and result in toxic maggots (p = 0.036 - from the above paragraph). Thus, to reach the point at which maggots are available for a second round of poisoning , the probability = .40 × .90 × .10 × .10 × .036 = 0.00013, or about 1 chance in 10,000! (Note: none of these probabilities is known exactly and each will be highly variable among real marshes).

This model suggests that the chance that a single carcass will start a major outbreak is likely very small. However, despite the odds, massive outbreaks, as in Alberta and Saskatchewan during 1994, do occur (CCWHC Newsletter Vol.2, No.3). The model allows us to predict features that can change the odds in favour of an outbreak. For

example, a worst case scenario would include: - abundant C. botulinum spores in the soil (so that most animals that die contain spores); - abundant substrate in the form of animal carcasses for toxin production; - inefficient scavenging (so that many carcasses persist); - hot, still weather ideal for fly activity (so that all carcasses contain maggots); - a large, dense population of susceptible birds (so that toxin-bearing maggots are likely to be found and consumed).

Factors, such as islands, loafing areas or receding water levels that concentrate birds would also increase the likelihood that birds will find toxic maggots and become poisoned. Botulism usually occurs in hot weather of summer and early autumn; in some marshes the disease may recur very early the following spring among diving ducks. Botulinum toxin is very stable at cold temperatures and these birds may become poisoned by consuming toxic material (perhaps invertebrates) associated with sunken carcasses of birds that died the previous autumn. These spring outbreaks end spontaneously, probably because the weather is too cold for fly activity and perpetuation of the outbreak. (Gary Wobeser - CCWHC Western/Northern Regional Centre)

Responses to the Questionnaire to Readers

In the Summer 1994 issue (Vol. 2, No.2), readers were asked to return a questionnaire about their perceptions of the CCWHC Newsletter. As of November 1994, 156 questionnaires had been returned: 78 from wildlife managers, wardens, biologists, and similar wildlife professionals, 28 from research scientists, 22 from veterinarians and 28 from persons of other professional backgrounds. We thank all of the readers who took the time to respond. The responses have provided us with both feedback and guidance for the continued evolution of the Newsletter and of other publications that may better fill the needs expressed than can the Newsletter itself. In general, there was enthusiasm for the feature articles and the regular regional wildlife disease updates. A large number of topics were suggested for future feature articles and use of more graphics in these features was a common suggestion. Under-representation of fish diseases was identified as a bias. This reflects the current activities of the CCWHC and is something we hope, over time, to correct. Calls for more detailed treatment of a variety of diseases support a decision taken by the CCWHC to assess the feasibility of publishing a series of information pages on specific diseases in a format other than the Newsletter itself. The readership will be kept fully informed of the evolution of this project. Because of budgetary uncertainties, readers were asked whether or not they would be willing to pay an annual subscription fee for the Newsletter; 76% indicated that they would. However, we will institute such a fee only if it becomes absolutely necessary. Some alternative ways of supporting the Newsletter currently are being explored.

Mycoplasma gallisepticum in House Finches in the Eastern United States

Mycoplasma gallisepticum is a species of bacteria that causes chronic respiratory disease in chickens and infectious sinusitis in both wild and domestic turkeys. Most meat-producing commercial poultry flocks in North America are free of this bacterium due to a campaign of eradication and exclusion, and eradication from egg-producing flocks is currently being attempted. In February 1994, house finches (Carpodacus mexicanus) with mild to severe conjunctivitis were reported at bird feeding stations in Maryland and Virginia. Mycoplasma gallisepticum was identified in lesions of affected eyes of these birds. This disease has since been reported in coastal states from Massachusetts to North Carolina, and in West Virginia.

Affected birds have watery or thick and opaque exudate on the conjunctiva which can partially or completely obscure vision. Microscopically, there is chronic inflammation of the conjunctiva, sinuses and nasal cavity. Hundreds of affected house finches have been reported. There are anecdotal reports of similar clinical signs in one or more individual cardinals, chickadees and nuthatches, but infection has not been confirmed in these species. This is the first reported occurrence of infection with Mycoplasma gallisepticum in passerine birds. The house finch is not native to eastern North America but was introduced from its normal range (from the Rocky Mountains westward) to the New York City area in 1940. Since then, it has expanded its range southward to Georgia, westward to the Mississippi River and northward to include southern Ontario, Quebec and Nova Scotia. Discovery of infection in these passerines raises concerns for the poultry industry as well as for affected wild bird populations. (This information was supplied by the Southeastern Cooperative Wildlife Disease Study and its newsletter: SCWDS Briefs)

Disease Updates

Atlantic Region

Unusual observations of harp and hooded seals

Annual migrations of harp seals (Phoca groenlandica) and hooded seals (Cystophora cristata) along the northwest Atlantic are closely associated with the movement of ice. These animals whelp and reproduce on ice floes in the Gulf of St. Lawrence and east of Newfoundland in February and March, and afterwards gradually move back toward the waters around Greenland where they spend the summer months. During late summer and early fall of 1994, a few adult harp seals and hooded seals, either very weak or dead, were reported along the shores of PEI and Nova Scotia. Two harp seals and one male hooded seal found on the north shore of PEI were examined by necropsy. All three were in poor body condition and were thought to have died from emaciation/starvation. The stomach of one of the harp seals contained more than 15,000 nematodes of the genus Contracaecum. Although no precise record is available on these types of observations, the

latter may represent a trend toward an increasing number of stray animals of these two species during the summer and fall, possibly reflecting increasing populations of these species. (John Parsons and Sylvia Craig, Dalhousie University, Halifax, Nova Scotia, and Pierre-Yves Daoust, CCWHC - Atlantic Regional Centre)

Morbillivirus infection (canine distemper) in a bobcat

Retrospective examination, by immunohistochemical techniques, of the brain of a juvenile bobcat (Lynx rufus) submitted in November 1993 revealed the presence of very large amounts of morbillivirus antigen. This bobcat had been found by a hunter in the woods in central New Brunswick in mid-November 1993 and had been presented to a local veterinary clinic, two days later, because of nervous signs. These signs had progressed over the following week, at which time the animal was euthanatized. The bobcat had severe chronic encephalomyelitis (inflammation of the brain and spinal cord). Morbilliviruses are associated with several diseases in humans (measles) and animals, including distemper in canids and raccoons. However, disease caused by these viruses in cats has only recently been reported, one case involving captive leopards, tigers, lions and a jaguar in California, and another associated with substantial mortality among lions in the Serengeti region of Tanzania.

Cerebral nematodiasis in a bobcat

In the spring of 1994, conservation officers in Nova Scotia and New Brunswick received several reports of bobcats (Lynx rufus) in close proximity to human habitation during daylight hours. The animals often were quite thin and appeared to be foraging. Between April and June, three of these animals were submitted to our laboratory. The first bobcat, an immature male, was observed convulsing before it was humanely killed. The animal was in good body condition and no lesions were identified grossly. Microscopically, there were areas of inflammation randomly scattered throughout the brain. In two areas, the inflammation was centred around nematode parasites, and a single aggregate of inflammatory cells surrounded protozoan organisms. The nematodes were 30-40 m in diameter. Unfortunately, other morphological features typically used to precisely identify these parasites could not be distinguished. Several nematodes are associated with this type of lesion, including, in the Atlantic region, Baylisascaris procyonis (raccoon roundworm) and Halicephalobus (Micronema) deletrix (a soil nematode that is an opportunistic pathogen of horses and humans). The protozoan organism was tentatively identified as Toxoplasma gondii, since wild and domestic cats are commonly infected with this parasite. The other two bobcats examined (an adult female and an immature male) were in very poor body condition, and severe emaciation was the final diagnosis. The brain of the female had a few areas suggestive of inflammation, but marked post-mortem decomposition precluded a more detailed examination. The male was also badly decomposed and had been shot in the head, thus preventing examination of the brain.

Therefore, in both of these cases, the possibility of emaciation having been secondary to a pre-existing neurological disease could not be ruled out. (Drs. Pierre-Yves Daoust and Scott McBurney - CCWHC Atlantic Regional Centre)

Québec Region

Stranding of two bottlenose whales in the St. Lawrence estuary

On November 9th, a team from the CCWHC Quebec Regional Centre went to Montmagny to perform a post mortem examination on a stranded adult female northern bottlenose whale (Hyperoodon ampullatus). The examination did not reveal any abnormalities which could explain the death of this 7.40 m cetacean. Three days later, an immature male of the same species was found stranded 45 km downstream at Saint-Roch-des-Aulnaies. This specimen was smaller (3.90 m), and consequently could be brought to the veterinary college in Saint-Hyacinthe. The presence of milk in the adult's mammary gland leads us to believe that she was lactating and that this second cetacean may have been her calf. Bottlenose whale calves are not weaned until they are at least one year old and a calf this size could therefore still be dependent on its mother and be unable to survive without her. Bottlenose whales usually live in deep water off shore where they feed on squid, pelagic fishes and sea stars. The presence of these two specimens in the St. Lawrence Estuary and their death are unexplained, but may be related to the fact that there were strong northwest winds during the previous days. This is only the second time that this species has been reported in the St. Lawrence Estuary. Another stranded bottlenose whale was observed under similar wind conditions in the 1940's.

Prevalence of parasitic cataracts in St. Lawrence River fish

The prevalence of parasitic cataracts in 13 species of fish was evaluated during a study of the health status of the fish communities of the St. Lawrence River in the Quebec city region. The eyes of 4741 fish were examined for the presence of lens opacities. A direct microscopic examination was also performed on crushed lenses of 153 of these fish (randomly selected). These results indicate that ocular trematodes are very common in numerous species of fish in the St. Lawrence River. Since it is the larval form of the parasite (metacercaria) that is found in the lens of the eye, it is not possible to identify the parasite species though these trematodes are probably of the genus Diplostomum. The adult form of these trematodes is found in the digestive tracts of piscivorous birds which are infected upon ingestion of fish eye lenses containing metacercaria. Snails act as initial intermediate hosts after infection by larvae present in avian feces. These larvae subsequently invade the lens of the second intermediate host, the fish, and produce a cataract as they develop. Blindness can be complete if a large number of parasites are

present and the blind fish are consequently more vulnerable to avian predators. The impact of these parasites on fish populations is difficult to evaluate.

Rupture of a dissecting aneurysm of the pulmonary trunk in a beluga whale from the St. Lawrence Estuary

An adult male beluga whale found stranded on June 13, 1994 in Ste-Flavie (Québec) was brought to the Quebec Regional Centre for post mortem examination. The left pleural cavity and pericardial sac were filled with several litres of partially coagulated blood. The pulmonary trunk was markedly dilated and its wall was thin, haemorrhagic and split into two sheets. These sheets formed a sac filled with coagulated blood. The cause of death was internal haemorrhage caused by the rupture of a dissecting aneurysm (a pathological thinning and bulging of a blood vessel) of the pulmonary trunk. This vessel conducts blood from the right ventricle of the heart to the lungs. Interestingly, an identical case was diagnosed in 1983 in another beluga from the same population (Martineau et al., Journal of Wildlife Diseases, 22, 1984, 289-294). Dissecting aneurysms of the pulmonary trunk are rarely reported in mammals. In humans and other mammals, excessively high pressure in the pulmonary artery has been identified as a potential cause. Pneumonia due to lung worms was present in both whales and may have contributed to the development of pulmonary hypertension. (Stéphane Lair and Daniel Martineau - CCWHC Quebec Regional Centre)

Ontario Region

Tularemia in beavers

In October, a beaver found dead by a trapper at Pickle Lake was submitted through the Ministry of Natural Resources (MNR) office in Sioux Lookout to the CCWHC laboratory in Guelph. At necropsy, the beaver was found to have hepatic, splenic and lymphoid necrosis. Francisella tularensis, the agent of tularemia, was isolated from the liver. Since then, there have been reports of other dead beavers in the area, and of reduced beaver populations in the Atikokan area. However, no additional carcasses have yet been submitted for necropsy, and the connection between these reports and tularemia remains speculative. MNR has issued an advisory to the trappers in the area, outlining some of the important aspects of the disease and describing precautions that should be taken when handling the carcasses of potentially infected animals (tularemia is a contagious disease that can affect humans). In the past, there have been numerous outbreaks of tularemia on the Winisk and Severn River systems, the most recent being in 1981. (Doug Campbell, CCWHC - Ontario Region and E.M. Addison, Ministry of Natural Resources)

Ring-billed gulls - False Duck Island

In early October the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA) veterinary diagnostic laboratory in Brighton was advised by a local poultry farmer that many bird carcasses had been seen on False Duck Island at the eastern end of Lake Ontario. Personnel from the Canadian Wildlife Service (CWS) and the CCWHC visited the island and found 100-150 carcasses of ring-billed gulls and smaller numbers of carcasses of double-crested cormorants, herring gulls and greater black-backed gulls. All carcasses were desiccated, indicating passage of time since death. The CWS has monitored this colony of ring-billed gulls for several years; this level of mortality in juvenile birds is not unusual and may be attributed to starvation. The carcasses were unsuitable for investigation of other causes of death such as infectious disease and exposure to toxins. This incident provides a good example of the importance of early detection and investigation of mortality incidents. (Doug Campbell, CCWHC - Ontario Region, and Chip Weseloh, Canadian Wildlife Service)

Lead poisoning in loons

Lead poisoning from the ingestion of fishing weights continues to be diagnosed in loons submitted for necropsy. Since the publication of the last Newsletter, 6 more adult loons submitted by the Ministry of Natural Resources have been examined; 4 had ingested fishing weights and had lesions compatible with lead poisoning.

***Salmonella* and *Campylobacter* in gulls**

There is some concern regarding the carriage of zoonotic pathogens such as Salmonella and Campylobacter by gulls, particularly those that forage at sewage lagoons and garbage dumps. This year, samples of liver and small intestine from all gulls submitted for post mortem examination at the CCWHC - Ontario Regional Centre were submitted to the bacteriology laboratory for detection of Salmonella and Campylobacter species. Of 32 ring-billed gulls examined, 5 were positive for Campylobacter (16%) and 6 were positive for Salmonella (19%). In 8 gulls of other species (7 herring gulls and 1 greater black-backed gull), 1 was positive for each of Campylobacter and Salmonella. The carriage rate for Campylobacter is similar to that reported elsewhere. The rate of Salmonella carriage is higher; 5% is a commonly quoted figure for inapparent carriage of this pathogen. The fact that many of these birds had concurrent disease or traumatic injury may have increased the rate of Salmonella excretion.

Other observations

In general, neoplasms are rare in wild animals but occasionally they are observed either as the cause of death or as an incidental finding at necropsy. Lymphosarcoma (a malignant neoplasm of lymphocytes) involving spleen, liver and kidney was diagnosed in an emaciated great horned owl brought initially to a rehabilitation clinic. Emaciation and death were attributed to the effects of the neoplasm. In contrast, a renal adenoma (a benign neoplasm of the kidney) was considered an incidental finding at necropsy in a red tailed hawk which had extensive traumatic injuries.

Lead poisoning due to the ingestion of lead shot was diagnosed in a Canada Goose. The unusual aspect of this case was that the shot was of very small calibre, typical of that used in target shooting. The suspicion is that this animal, and others in the flock found dead but not examined, were grazing on a skeet range. A similar incident involving Canada geese occurred several years ago. In this recent instance, the concentration of lead in the liver was 487 parts per million (dry weight) which is extraordinarily high. (Doug Campbell and Ian Barker, CCWHC - Ontario Region)

Western and Northern Region

Mortality patterns in orphaned harbour seals

For each of the last several years, the Animal Health Centre in Abbotsford, B.C. has been assessing the causes of mortality in orphaned harbour seals undergoing rehabilitation at the Vancouver Public Aquarium. In the summer of 1994, we examined 55 animals in 45 submissions; 37 of these submissions were made in July and August. Pneumonia was the most prominent diagnosis and occurred in 26 animals; the bacterium Pseudomonas aeruginosa, in association with hemorrhagic pneumonia, was isolated from 16 of these seals. Five seals were diagnosed with meningitis and Ps. aeruginosa was cultured from three of these. Starvation and emaciation were significant findings in 12 orphaned seals. Recent unpublished observations suggest that adult seals may leave their offspring for periods of up to 48 hours. In some cases, well-meaning individuals may have assumed that such pups were orphaned and "rescued" them for rehabilitation. It is likely that some of these animals become malnourished and that their immune systems are weakened. They are, thus, susceptible to opportunistic pathogens such as Pseudomonas sp.. (Ron Lewis - Animal Health Centre, B.C Ministry of Agriculture and Fisheries, Abbotsford)

Effects of vomitoxin on ducks

Some fungi produce poisonous substances termed mycotoxins. During the cool, wet summer of 1993, the fungus Fusarium graminearum infected many grain crops in Manitoba causing head-blight or Tombstone Disease (reported in CCWHC Newsletter

Vol.2, No.2). There was concern that the toxin deoxynivalenol, also called vomitoxin, produced by this fungus might harm field-feeding waterfowl. This toxin causes food refusal, growth depression and vomition in domestic livestock; hence its common name. A study was done at the Western College of Veterinary Medicine during the summer of 1994 with support from The Wildlife Health Fund, Ducks Unlimited Canada and Manitoba Natural Resources to determine: (a) if mallard ducks would willingly consume wheat contaminated with vomitoxin when given a choice between contaminated and uncontaminated wheat; and (b) if consumption of a diet consisting entirely of contaminated wheat for 14 days would have any clinical, hematological or pathological effects on mallards. Wheat used in the trials had 10% infected kernels and 5.8 parts per million of vomitoxin (a moderate to high level of contamination). Ducks did not avoid eating the infected and contaminated wheat. The amount of contaminated wheat consumed was greater than that of uncontaminated wheat in each of four experimental groups. Consumption of contaminated grain as the sole diet for 2 weeks had no apparent adverse clinical, hematological or pathological effects on either male or female mallards. The results suggest that wild ducks will consume grain containing vomitoxin but that a moderate level of this mycotoxin is unlikely to have serious adverse effects. (Sarah Boston and Gary Wobeser - CCWHC Western/Northern Region)

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