

**Canadian  
Cooperative Wildlife  
Health Centre**



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

Newsletter 2-1, Winter 1994

**In this issue:**

**[Work Underway at the CCWHC](#)**

**[CCWHC Headquarters Secretary](#)**

**[Feature Articles](#)**

[Newcastle disease in cormorants](#)

[Rabies update: First case in PEI, Rabies in Bats](#)

[New viral disease of lagomorphs in Europe](#)

[Investigation of pesticide poisoning](#)

[Hantavirus in North America](#)

**[Disease Updates](#)**

**[Atlantic Region](#)**

[Mortality in Leach's storm petrels](#)

[Common loon mortality](#)

[Verminous pneumonia in red foxes](#)

**[Québec Region](#)**

[Strychnine poisoning in grackles](#)

[Leptospirosis in a raccoon](#)

[Great horned owls and porcupines](#)

**[Ontario Region](#)**

[Tick species near Thunder Bay](#)

[Mercury poisoning: loons, eagle](#)

[Parvovirus in raccoons](#)

**[Western/Northern Region](#)**

[Phorate poisoning in Yukon wildlife](#)

[Avian cholera in Manitoba](#)

**[Announcements](#)**

**Work Underway at the CCWHC**

The CCWHC **Disease Investigation Manual** will be available in February. This is a comprehensive manual on what to do when confronted with dead and diseased wildlife in the

field. Topics include: who to contact for assistance in each province, collecting appropriate background information, when to investigate, when and how to perform a necropsy, samples to collect, shipping specimens, protecting yourself, and disposal of carcasses.

A review of **Surveillance of Wild Animal Diseases in Europe** is being undertaken by Dr. Ted Leighton, co-director of CCWHC. During the 1993-94 academic year, Dr. Leighton is a visiting scientist at the Laboratory for Studies of Rabies and Wild Animal Pathology near Nancy, France. The aim is to identify systems to detect and monitor diseases in European wildlife and to catalogue key institutions and individuals responsible for this surveillance. The benefits of this will be a list of contacts and sources of information for CCWHC when asked to provide information on topics such as importation of wild animals from Europe, and to allow CCWHC to profit from the experience of European colleagues who have established national systems for wild animal disease surveillance. Funds have been contributed by the governments of Canada and France.

### **CCWHC Headquarters Secretary**

**Jacqui Brown** is secretary for the CCWHC in Saskatoon. She is responsible for most routine correspondence for Headquarters and Western/Northern Region, as well as the distribution of the Wildlife Health Centre Newsletter and periodic wildlife disease updates. In addition, Jacqui maintains the CCWHC directory of wildlife disease expertise and has assisted in production of the Wildlife Disease Investigation Manual. She is a graduate of Robertson's Secretarial School and has considerable experience as a medical secretary, having worked in both the College of Medicine and the Department of Veterinary Pathology prior to joining the CCWHC. She is two-time winner of the "Smo-Kin Fingers" speed typing competition. She currently works afternoons and can be reached at 1-800-567-2033 or (306) 966-5099.

## **Feature Articles**

### **Newcastle Disease in Double-Crested Cormorants**

Two outbreaks of Newcastle disease (ND) occurred recently in aquatic birds in North America. The first involved double-crested cormorants, white pelicans and gulls in Alberta, Saskatchewan and Manitoba during late summer of 1990. Although it is difficult to make accurate estimates, mortality is thought to have approached 10,000 cormorants and 2,000 white pelicans and gulls. Disease was first detected in early August and continued through September in both adults and juveniles.

Newcastle disease was not detected in 1991, although surveillance of cormorant colonies was increased. However, in 1992 another epizootic of ND occurred, this time involving a larger geographic area than in 1990. Newcastle disease resulted in cormorant deaths on the Great Lakes, in the American Midwest and again in Alberta, Saskatchewan and Manitoba. Mortality was first recognized in juvenile cormorants and pelicans during late June and July on Canadian and American islands in the Great Lakes and on lakes in Minnesota, North Dakota, South Dakota and Nebraska. On the Canadian prairies the disease was not observed until later in July and August. Young-of-the-year were most severely affected and, depending on the colony, mortality

varied from 6 to 90% of the hatch. Newcastle disease virus (NDV) was isolated from double-crested cormorants, but not from pelicans or gulls, although some showed typical symptoms of inability to fly, abnormal behaviour, depression, and unilateral wing or leg paralysis.

Newcastle disease was not diagnosed in cormorants in Canada or the United States in 1993, although approximately 50% mortality in young-of-the-year was detected on a large cormorant colony in northern Saskatchewan. Unfortunately, the colony was not visited until after the fall migration and carcasses were too autolysed for any diagnostic follow-up.

Newcastle disease has been reported previously in cormorants but mortality to the extent seen in the epizootics of 1990 and 1992 appears unprecedented. Other reports of ND in cormorants include: Scotland between 1949 and 1951; the St. Lawrence River, near Rivière du Loup and Trois Pistoles, Québec, during the summer of 1975; and on the Volga River delta of the former Soviet Union.

### **Epidemiology of Newcastle Disease**

Newcastle disease is considered infectious to most birds and is an important disease of captive-wild and domesticated birds. It may also be significant in the ecology of some free-living birds, such as cormorants. Several strains of NDV have been identified and the type and severity of disease dependent on strain of virus and species of bird infected. Newcastle disease virus has been isolated from numerous wild birds, but epizootics in free-living birds are rarely observed.

Three panzootics of ND have been recognized. The disease was first described in chickens near Newcastle-upon-Tyne, England in 1926, hence its name. This was part of an epizootic thought to have originated in southeast Asia, spreading world-wide over 25 years. The second panzootic emerged in the Middle East in the late 1960's and spread rapidly to most countries by 1973. The virus was associated with parrot or psittacine species and the rapid spread is thought to have been due to international trade in these birds. The most recent, and ongoing, panzootic involves pigeons. This disease initially spread across Europe in the early 1980's and became established in feral pigeons in several European countries, and Canada. Transmission from feral pigeons to susceptible domestic poultry flocks has occurred in England.

Strains of NDV are characterized by the severity of disease produced in chickens and by the body system most severely affected. "Velogenic" describes strains that are highly pathogenic to chickens; lentogenic strains produce mild disease; mesogenic strains are intermediate in severity. These terms do not necessarily reflect the pathogenicity of a viral isolate in hosts other than chickens. Neurotropic and viscerotropic strains produce neurological and intestinal signs, respectively. These groupings are useful in understanding the epidemiology of ND and in predicting the potential risk of a viral isolate to poultry.

Monoclonal antibodies (Mab) are now used to better characterize strains of ND viruses. Based on Mab analysis, the viruses from the 1990 and 1992 outbreaks in cormorants appear closely related, or are the same virus, and are very similar to the viscerotropic velogenic virus responsible for the 1970-73 panzootic. In recent years, virus isolates of this type have only been recognized in poultry and caged birds in, or suspected of originating from, South America.

Whether this virus is endemic in cormorant populations or whether cormorants become reinfected with this strain on southern wintering grounds remains to be determined.

### **Antibodies to NDV in Cormorant Colonies**

In 1993, CCWHC, Canadian Wildlife Service, and several provincial wildlife departments surveyed cormorant colonies across Canada. Between May and June, approximately 30 cormorant eggs were collected from 20 colonies located in coastal British Columbia, Alberta, Saskatchewan, Manitoba, the Great Lakes in Ontario, the St. Lawrence River in Québec, and coastal Prince Edward Island.

During production of an egg, the hen transfers antibody to the yolk. At hatch, the transferred antibody protects the chick against the specific disease agent to which the antibody responds. The amount of antibody transferred to the egg depends on the level of antibody in the hen at the time of laying; high levels result from recent and/or repeated exposure to the disease agent. Antibody levels drop over days or weeks after hatch and, until their own immune system produces antibody to NDV, chicks are susceptible. Outbreaks of ND in juvenile cormorants may be a result of waning maternal antibody levels in the developing chick.

Interpretation of yolk titres is difficult because we cannot differentiate between different strains of NDV, and the significance of various levels is unproven. Despite the limitations, yolk titres may be useful for obtaining information on differences in prevalence of exposure to NDV among years, and relating effects of flock immunity on the development of epizootics. The result of the 1993 cormorant colony survey are shown in Figure 2. Individuals with positive titres (> 1:20) were present in 19 of 20 colonies and the prevalence of positive titres and levels of titres appeared to be greater in western Canada than in the Great Lakes, St. Lawrence River and Prince Edward Island. A more restricted survey in 1991, performed by many of the same agencies as in 1993, indicated that the prevalence and titres were higher in the Great Lakes than in the west. These apparent trends must be interpreted with caution because of the small sample size and the lack of statistical analysis to this point. Unfortunately, surveys were not done in 1990 and 1992, years in which ND epizootics occurred. *Dr. Trent Bollinger - CCWHC Western/Northern Region.*

### **Rabies Update**

**(addendum to previous CCWHC newsletter)**

The first case of rabies on Prince Edward Island was diagnosed in November of 1993 in an adult red fox. The isolate was identified as being of bat origin, but differed from strains isolated from bats in other regions of Canada and from terrestrial mammals in the Atlantic region.

The following article (excerpted from Provincial Museum of Alberta natural history occasional paper no. 19) was provided by the author, *Dr. Margo Pybus - Alberta Fish and Wildlife.*

### **Rabies in insectivorous bats in the prairies**

Rabies was first identified in insectivorous bats in 1953, although it is likely the virus was overlooked for many years. Since then, it has been identified in 30 of 39 bat species found in North America and in most provinces and states.

The pattern of infection and transmission of virus in bats appears to be distinctly separate from that in terrestrial species (such as skunks and foxes). While rabies in terrestrial species tends to sweep through susceptible populations in successive waves, rabies in insectivorous bats is much more localized and often is restricted to individual solitary bats or a few infected individuals in a colony. Transmission may occur more often during normal aggressive or grooming behaviours than by an induced hyperaggressiveness as seen in carnivores. Monoclonal antibody tests can distinguish bat sources of virus from terrestrial sources of virus and further support the suggestion that rabies cycles independently in bats, with very little overlap with the virus in other species.

Rabies has been identified in bats in each of the three prairie provinces. Similarly, it is enzootic (occurs naturally) in bats throughout Canada and in many states in the USA. The pattern of infection appears similar throughout North America: a few infected individuals are reported each year in most areas; infected individuals often are distributed widely; the majority of cases are reported June through September; and a variety of colonial and solitary species are involved.

In the prairie provinces, about 10-12 rabid bats are recorded each year. Big brown bats make up the highest proportion of rabid bats (50-60%); however, the rate of infection is relatively low (5-10%). This species often uses maternity roosts (summer colonies) in buildings shared with people and therefore is likely to be seen and collected. Big brown bats also are the only species to hibernate in buildings in the prairie region. Normally, hibernating bats are hidden from public view; however, in periods of rapid temperature change (either increasing or decreasing), conditions within buildings may become unsuitable and big brown bats become active and seek other sites to sleep. At this time they may be seen and submitted for rabies testing.

Little brown bats are by far the most common bat species throughout the prairie regions. This species uses maternal colonies in a wide variety of buildings and they are the bat that people are most likely to see in houses, in barns, along rivers, and streams, and around most lakes and sloughs. They also are the species most likely to be submitted for rabies testing, and yet they are the species least likely to be rabid.

We know very little about rabies in solitary forest-dwelling species of bats. Hoary bats, silver-haired bats, and red bats migrate north across the prairies in late spring/early summer, spend the summer in deciduous and conifer cover in the aspen parkland and boreal ecoregions, and then cross the prairies again in August and September. As such, they are rarely seen by people and thus, infrequently submitted for rabies testing. All we can say is that rabid individuals of these species are recorded.

People should be warned not to handle bats, similar to the warnings against handling any wild animal. Bats found on the ground should be removed (without handling them directly) in order to prevent them from being picked up by a child or a pet. This is accomplished easily by wearing gloves or by using a stick to push the bat into a container. Once collected, such bats should be

submitted to a veterinarian or wildlife officer along with a request to have the animal tested for rabies. (Dr. Pybus has provided a comprehensive list of references that is available on request.)

## **New Viral Diseases of Rabbits and Hares in Europe**

### **Hare today and gone tomorrow?**

During the past decade, two previously-unknown devastating diseases, one of rabbits and one of hares, have swept across Europe, with severe consequences for wild populations and agricultural enterprises alike. These diseases illustrate the constant evolution of new pathogens and infectious diseases, and they also represent new potential threats to wildlife populations in North America.

The two diseases are now known as Rabbit Viral Hemorrhagic Disease (RVHD) and European Brown Hare Syndrome (EBHS). Their similarities are more striking than their differences. They both were first recognized in the early 1980's. They are caused by very similar, but slightly different, viruses and are characterized by very high mortality, rapid death, and liver necrosis. In rabbits, and apparently also in hares, very young animals are resistant to infection and suffer no disease. The viruses that cause these diseases are caliciviruses. They are readily identifiable by immunological methods but neither has been isolated in cell culture despite numerous attempts. They are hardy, able to remain infective in the environment for weeks, and highly contagious among members of each species of susceptible host. There have been several experimental studies of the infectivity of the virus of RVHD for the European Brown Hare (*Lepus europaeus*) and of EBHS for the European Rabbit (*Oryctolagus cuniculus*). The majority of studies did not report disease after such cross-infections, but a few did. Thus, the cross-infectivity of the two viruses remains uncertain. There is, as yet, no evidence of cross-infection between wild populations of hares and rabbits.

What is now known to be EBHS was recognized in Sweden, Denmark, Germany, and Italy in the early 1980's as unusually high mortality of the European Brown Hare. The cause was not apparent and was sought among a wide range of poisons, including mycotoxins, herbicides and type 00 rape (a new variety of oil-seed, newly and widely planted in Europe at that time), and also among a wide range of possible viruses, none of which could be isolated from the dead hares. In 1988-89 it was finally established that EBHS was caused by a virus. The disease occurs widely in western and eastern Europe, including Great Britain and affects both the European Brown Hare and, to a lesser extent, the Varying Hare (*Lepus timidus*). There are no accurate measures of the effect of EBHS on wild hare populations, but the scale of mortality has been very large. European Brown Hares are raised commercially in Europe, and mortality rates on infected farms have been as high as 100%. Hares that survive the disease produce antibodies against the virus and appear to be immune to further disease. In two wild populations studied in the late 1980's, 90% of hares had antibodies to EBHS virus. The disease appears now to be permanently established over much or all of the range of the European Brown Hare in Europe.

Rabbit Viral Hemorrhagic Disease is a disease of the European wild rabbit (this species is also the domestic rabbit now distributed world-wide). It was first recognized in domestic rabbits imported into China from Germany in 1984. It is not known whether the virus originated in Europe or Asia. The disease spread quickly among commercial rabbit farms in China and Korea,

was first recognized in European domestic rabbits in 1986 and reached Mexico in imported frozen rabbit meat in 1988. RVHD is now permanently established in wild rabbits over most or all of their range in Europe. It has been a huge economic cost to commercial rabbit production. In Germany, the disease had cost approximately \$105 million by 1989; in Italy, approximately 32 million rabbits died in the three years following the first occurrence of the disease; in Mexico, 65,000 rabbits died of the disease and 100,000 others were killed in a successful eradication program. Rabbit colonies in research laboratories also have been devastated. The effect of the disease on wild rabbit populations has not been determined but, as with EBHS, the scale of mortality of wild rabbits has been very high. Sudden disappearance of nearly all rabbits in a colony has been a common observation. However, re-population also occurs fairly rapidly, perhaps in part due to the fact that very young rabbits are not affected by the disease.

What risk do these diseases pose to Canadian populations of wild rabbits and hares? Experimental infections of Cottontail Rabbits (*Sylvilagus* sp) with the virus of RVHD have been carried out. No disease was produced but infection apparently did occur and the cottontails shed enough virus to infect and kill domestic rabbits in contact with them. The susceptibility of our wild hares (Jack Rabbits, Snowshoe Hares, Arctic Hares - all members of the genus *Lepus*) to the viruses of either EBHS or RVHD has not been tested. This should be done, since the virus of EBHS can cause severe disease in two other species of the same genus. The possibility that these diseases might be imported to Canada certainly exists. A chronic form of EBHS appears to occur, with persistence of virus and potential shedding of virus over long periods. The same may occur in rabbits that survive RVHD. Importation of such rabbits or hares that have the potential to transmit these viruses to other animals could have grave consequences.

Thus, sometime in the early 1980's, two new viruses appeared. Their origin is uncertain, but serological evidence suggests the existence of a non-disease-producing form of one or both of these viruses in eastern Europe 12 years before the first occurrence of RVHD. Origin of RVHD and EBHS viruses by mutation of this non-virulent strain is a reasonable suggestion. The viruses spread throughout Europe and around the world through commercial trade that was unregulated with respect to these new diseases. Both diseases are now well established in wild populations, which now also are permanent reservoirs of the diseases for domestic populations. Early detection of these diseases in wild populations was possible because of the systems for wild animal disease surveillance in place in some of the affected countries at that time. This story argues for vigorous surveillance of wild animal diseases, and a regulatory apparatus responsive to the possible emergence of new diseases and the threats they pose to both wild and domestic animal populations. (Dr. Ted Leighton - on sabbatic leave in France)

### **Investigation of a Wildlife Pesticide Mortality Incident**

When pesticide poisoning is suspected, properly collected and preserved samples and an accurate description of circumstances increase the likelihood of a confident final diagnosis. *Dr. Pierre Mineau, Pesticide Evaluation Section, Canadian Wildlife Service, Hull PQ*, provided information used for the following outline.

## Collection of Information

- **Species and numbers affected.** Count or estimate the number, age, sex, and condition of carcasses for each species. How fresh do they appear to be? Were carcasses scavenged? Do they bear obvious marks? Is there evidence that carcasses were disposed of before you arrived? Landowners can often provide this information! Record the area searched for carcasses.
- **Clinical signs.** If there are sick individuals, describe their behaviour. Were they easy to approach or capture? How were the carcasses distributed?
- **Describe the area.** Record the size of field, type of crop, stage of growth, and condition of the field (flooded or not). Estimate the distance from suitable habitat for the species of concern. It may be useful to photograph the site and some of the carcasses.
- **Environmental factors.** Recent rains, storms, high winds, or temperature extremes should be noted.
- **Chronology of events and contacts.** Who found the kill and when? When were you notified? How much time elapsed before discovery of the kill and your visit? Record names, addresses, and phone numbers of all persons involved.
- **Suspected Agent.** Record details of recent pesticide applications in the area, including when they occurred, formulation, rate, method of application, and if they occurred with other treatments, e.g. irrigation. Describe how this information was obtained and if the information was confirmed by presence of empty containers or other means.

## Collection and preservation of samples

Most recent wildlife poisonings have been caused by organophosphate or carbamate insecticides. A biochemical test (cholinesterase enzyme inhibition) is used for diagnosis. Samples collected for analysis must be collected and stored properly.

Whenever you investigate a potential poisoning, **avoid contaminating yourself - wear gloves, boots, and coveralls.**

- **Collect samples from the most fresh carcasses.** The most important part for diagnosis are the head and the alimentary tract. With small animals collect the whole carcass. With larger wildlife, take the head and whatever samples you can get from the esophagus and stomach. Also consider taking



samples of liver and kidney. For large kills, a sample of 10 individuals should be ample.

- **Collect samples of vegetation** from the site as well as 5cm deep cores of soil. The latter are useful for identifying granular insecticides. Look for any evidence of surface granules.
- **Freeze samples as soon as possible.** The head especially should be treated properly - the best assay will be of brain tissue. If it cannot be frozen immediately, pack carcasses in ice and transfer to a freezer ASAP. If you have access to dry ice or liquid nitrogen) this is a good way to freeze and hold the samples temporarily. Companies that supply hospitals with oxygen usually sell dry ice. Wear insulated gloves when handling dry ice or liquid nitrogen.
- **Store samples in a freezer that achieves the lowest possible temperature.** A good chest freezer set to minimum temperature should be adequate; it will maintain temperatures of about -30°C.
- **Do not allow head or stomach contents to thaw.** If carcasses are to be necropsied, tell the pathologist these items must be removed and returned to a freezer before they thaw.
- **Collect blood samples from sick individuals.** Blood can be drawn into heparinized capillary tubes. (for a large bird a small scratch or puncture to the brachial vein on the inside of the wing is an easy way to obtain blood.) Two or three tubes provide a sufficient sample. They should be sealed at one end only and kept cool, but not frozen, until they can be spun to separate the plasma which is then frozen as cold as possible.

## Hantavirus: A New Zoonotic Disease in the United States

A rodent-associated virus is responsible for a newly recognized disease that has caused the death of a number of people in the United States. The virus is a member of the **hantavirus** group, which cause persistent subclinical infection in small mammals and may cause severe, sometimes fatal, disease in humans. Transmission to humans is thought to occur primarily by inhalation of aerosolized virus from rodent's saliva and excrement. The deer mouse, *Peromyscus maniculatus*, a species found throughout most of North America, is thought to be the natural host and source of this virus.

The disease was first recognized in the four-corners region of New Mexico, Arizona, Utah, and Colorado in May, 1993. Affected individuals initially complained of flu-like symptoms, such as fever and muscle pain, then rapidly developed severe pulmonary disease resulting in respiratory failure and, often, death. The disease, called hantavirus pulmonary syndrome (HPS), appears to have affected primarily young healthy adults and the case fatality rate is in excess of 60%. Since those initial cases, others have been confirmed in several states including Idaho, Montana, and

North Dakota. Although HPS was recognized only recently, evidence suggests the virus has been present in deer mice, at least in the southwest United States, for some time.

The Centers for Disease Control and Prevention (CDC), U.S. Dept. of Health and Human Services, have published comprehensive recommendations to reduce the risk of infection with hantavirus, based on minimizing exposure to the rodent and its excrement. Some of their recommendations include: exterminating mice and other rodents from homes and other buildings; handling rodents and their excrement with gloves under well-ventilated conditions; and soaking carcasses and excreta in household disinfectant before handling.

Hantavirus has not been recognized in Canada however, individuals exposed to rodents and their excrement should be aware of the potential risk. Health and Welfare Canada is currently collecting sera and tissues from *Peromyscus* sp. to test for the presence of hantavirus in Canada. Until the geographic distribution of this virus and the risk factors for human infection are defined, precautions should be taken when dealing with rodents. *Dr. Trent Bollinger - CCWHC Western/Northern Region*

## **Disease Updates**

### **Atlantic Region**

#### **Mortality in Leach's storm-petrels**

In mid-July, several thousand dead or moribund Leach's storm-petrels (*Oceanodroma leucorhoa*) were found in Notre Dame Bay, northern Newfoundland, prompting an investigation by members of the Canadian Wildlife Service in St. John's. Thirty-one birds were submitted to CCWHC at the Atlantic Veterinary College for further examination. The majority were adults. The only significant finding in these birds was poor body condition, as indicated by atrophy of pectoral muscles, absence of fat reserves, and average body weight (38.6g) at least 6 grams less than that of adult storm-petrels collected from their burrows in eastern Newfoundland in an earlier study. The weather before and during this die-off was characterized by onshore winds and fog. Leach's storm-petrels are pelagic plankton feeders, and it is possible that strong onshore winds kept these birds from their normal feeding grounds long enough for them to deplete their energy reserves. *Dr. John Chardine - Canadian Wildlife Service, St. John's, Newfoundland; Dr. Pierre-Yves Daoust - CCWHC Atlantic Region*

#### **Common loon mortality**

Three female adult common loons were submitted by the Nova Scotia Department of Natural Resources, during the summer of 1993. All three birds were emaciated, and, in one, the gizzard contained a portion of a fishing line, two fishing hooks, a swivel and a nail, the latter having worked its way almost entirely into the liver. The levels of lead in the kidneys of these three birds were 167 ppm (the bird with the fishing material in its gizzard), 124 ppm and 15.5 ppm (wet weight). The first two values are remarkably high (background residues of lead in the kidneys of normal waterfowl are in the order of 5.0 ppm). The levels of mercury in the kidneys of these birds were 17.99 ppm, 26.46 ppm, and 19.71 ppm (wet weight). Although these levels

seem high, they are difficult to interpret since the effects of mercury intoxication can be influenced by several factors, including the rate of intoxication and the proportion of inorganic and organic forms of mercury involved. Moreover, these high levels of mercury in the kidneys may have been reached when stored fat was mobilized in response to inadequate food intake. *Dr. Lyn Ferns - Provincial Veterinary Laboratory, Truro, Nova Scotia*

### **Verminous pneumonia in red foxes**

*Angiostrongylus vasorum* is a nematode parasite of the pulmonary arteries and right cardiac ventricle of dogs and foxes. It is widely distributed in western Europe but is rarely reported in North America. In early 1993 a severe chronic verminous pneumonia, likely caused by the larval stages of *A. vasorum*, was found in an adult female red fox trapped in the eastern region of Avalon Peninsula, Newfoundland. A few small areas of necrosis and an intravascular parasite compatible with *Angiostrongylus* sp. were also found in this animal's heart. In early September, another adult female red fox was captured in the same general area. Although *Angiostrongylus* was not found in this fox, its lungs had marked interstitial fibrosis and vascular lesions strongly reminiscent of the pulmonary lesions described in dogs with heartworm.

In September, one of two red fox pups submitted by Park wardens from the Fortress of Louisbourg, Nova Scotia, had severe pneumonia associated with the presence of large numbers of adult and larval stages of the nematode *Crenosoma vulpis* and a lesser number of adults and larvae of *Capillaria aerophila*. *Dr. Hugh Whitney - Animal Health Division, St. John's, Newfoundland; Dr. Gary Conboy - Atlantic Veterinary College; Dr. Pierre-Yves Daoust and Dr. Scott McBurney - CCWHC Atlantic Region*

### **Quebec Region**

#### **Strychnine poisoning in two common grackles**

From July 20-27, 1993, several dozen birds of various species were found dead in Hull. Two common grackles were submitted for necropsy. They were in excellent body condition and their gizzards contained only a moderate amount of crushed corn. No significant lesions were seen grossly or microscopically. Toxicological tests on the gizzard contents revealed the presence of strychnine. The source of strychnine in this case is unknown, but the method of administration (coarsely ground corn) leads us to suspect intentional poisoning. Cases of this nature raise questions regarding the safeguards necessary to prevent inadvertent poisoning of non-pest bird species and of small mammals, including children.

#### **Leptospirosis in a raccoon**

An adult female raccoon in good body condition, but having difficulty climbing, was found in October 1993. The animal was euthanized due to suspicion of rabies. Microscopically, the kidney had severe multifocal lymphocytic inflammation with large numbers of spirochete bacteria. Immunoperoxidase assay with polyclonal antibody for *Leptospira* serovar *pomona* was strongly positive. Identification of the serovar was not performed.

Leptospirosis has been reported frequently in raccoons from many areas of North America, but little is known of its morbidity and mortality in wild animals. *Leptospira* spp. are large, finely-coiled bacteria. A wide range of wild and domestic animals serve as animal reservoirs and shed the organism via their urine. Human infection may occur directly, via contact with urine or tissue of an infected animal, or indirectly, via contact with water, soil, or vegetation contaminated by the urine of an infected animal.

Initially, rabies was suspected in this animal; however, we found no evidence of that disease. Rather, we observed a small area in the brain cortex where the nervous tissue was destroyed and infiltrated by white blood cells, mainly eosinophils. The presence of eosinophils in an area of tissue destruction is highly suggestive of the passage of a parasite, most likely a nematode larva.

This raccoon was affected by a disease which is potentially zoonotic, meaning that it can be transmitted from animals to man. This disease, along with other raccoon zoonoses (such as rabies and *Baylisascaris procynosis*) should be of concern to people who keep raccoons as pets.

### **Great horned owl death following predation on a porcupine**

An emaciated female great horned owl was found near Lotbiniere in September 1993. Many porcupine quills were observed in the plumage of the head, breast, and ventral surfaces of the wings and feet. These quills penetrated deeply into the subcutaneous tissue and muscle and several quills were also visible in the internal cavity, particularly in and surrounding the gizzard. The damage caused to the various organs (liver, spleen, eye, muscle and gizzard) was considered to be the cause of the bird's emaciation.

Great horned owls are known to prey upon a large variety of animal and bird species, including porcupines. Nevertheless, our experience has demonstrated this predation to be rare (out of 185 great horned owls admitted to the Raptor Rehabilitation Clinic of the Veterinary College here since 1987, only 5 had evidence of porcupine predation). *Dr. Daniel Martineau and Dr. Stéphane Lair - CCWHC Quebec Region*

## **Ontario Region**

### **Tick species found in the Thunder Bay area**

This past summer at Lakehead University we investigated the prevalence and species of ticks on pets in Thunder Bay, to determine if the ticks *Dermacentor variabilis* (the American dog tick or wood tick) and *Ixodes scapularis* (the black-legged tick, formerly *I. dammini*) were present locally. These ticks are important as vectors of zoonotic disease. The former is capable of transmitting Rocky Mountain Spotted Fever and tularaemia, while the latter is the vector of Lyme borreliosis. Both ticks are prevalent in Minnesota, and the wood tick is reported from northwestern Ontario to 40-50 km south and west of Thunder Bay.

Ticks were collected during weekly visits by two students to local veterinarians and grooming parlours. Most of the 50 ticks identified were *D. variabilis* from dogs that had recently returned to Thunder Bay from Minnesota, or from the Fort Frances, Atikokan or Dryden areas, west of

Thunder Bay. However, nine cases may have been contracted locally. While it is not yet clear that *D. variabilis* is established in Thunder Bay, the rate at which it is being imported on traveling domestic pets increases the risk of establishment.

A single *I. scapularis* female was obtained (through OMNR) from a person at Crow Rock Inlet on Rainy Lake near Mine Centre. A small mammal survey (n=45) failed to reveal any animals in the locality infested with *I. scapularis*, suggesting with a high degree of confidence, that this tick was not established in that locality, and that the tick found on the person was probably translocated to the area as an immature stage on a bird, mammal, or person. There are scattered records of this tick in the Fort Frances/Rainy River area, and from Thunder Bay; in one case in Fort Frances a male and female were found on the same dog, suggesting that a population might be established in that area, though this is unconfirmed to date. Since *I. scapularis* is present in Minnesota, to the south, migrating birds, and humans or their pets traveling from the south may carry ticks into this part of Ontario. Temperature requirements for tick development suggest that it is unlikely to establish north of Kenora, or in the Thunder Bay area.

Although scattered individual *I. scapularis* adults have been found in southern Ontario, small mammal surveys, including at sites where adult ticks have been found (such as Huntsville, this year), have failed to reveal established populations other than at Long Point, Lake Erie. The scattered pattern of human cases of Lyme Disease in Ontario may be related to the unpredictable distribution and low density of ticks translocated to the province by birds or other means. Climatic conditions would permit establishment of viable populations of this tick in Ontario south of North Bay, so other factors may be influencing its distribution. *Dr. Murray Lankester - Lakehead University, Dr. Ian Barker - CCWHC Ontario Region, Robbin Lindsay - University of Guelph*

### **Mercury poisoning in loons**

Seventeen loons from widely scattered locations, collected by employees of the Ontario Ministry of Natural Resources, were submitted to the CCWHC for post-mortem examination

As reported in Wildlife Health Centre Newsletter Vol 1.3, a variety of pathological conditions were recognized at necropsy in these birds. Samples of liver and kidney from all birds were submitted to the Ontario Ministry of Agriculture and Food toxicology laboratory for determination of mercury and lead levels.

One bird, which has a lead fishing sinker present in its stomach, had lead levels of 290 parts per million dry weight (ppm dw) in kidney and 76 ppm in liver. Lead was not detected in any of the other birds.

Six birds had tissue mercury levels interpreted to be significantly elevated. Concentrations in liver ranged from 76 - 278 ppm dw in these birds; kidney concentrations ranged from 63 - 212 ppm dw. Liver mercury levels of  $29.73 \pm 12.97$  ppm wet weight (approx. 90 ppm dw) have been previously reported in loons from mercury-contaminated waters. Four of the six birds with high mercury levels were in poor body condition. The average body weight of the contaminated birds was 3.1 kg, while a published estimate of a normal average weight for loons is 4.4 kg. One bird

had been shot; two were entangled with fishing lines; two had high levels of parasitic infestation with acanthocephalans and trematodes, and one bird had a hemorrhagic enteritis of unknown etiology. No consistent pattern of lesions could be associated with elevated levels of mercury in tissue, and it is uncertain what the exact role of mercury was in causing debilitation and death in these birds. The remaining 11 birds had mercury concentrations in liver ranging from 5-70 ppm dw, and kidney concentrations of 8-81 ppm dw.

### **Mercury poisoning in a bald eagle**

A mature bald eagle from Gogama in the Timmins district was submitted by the Ontario Ministry of Natural Resources to the OVC wild Bird Clinic in a state of emaciation and weakness. Cardiac and musculoskeletal abnormalities were detected, and the bird was euthanised because of a poor prognosis. Post-mortem examination revealed multifocal areas of skeletal and cardiac myofibre degeneration and hyalinization of arteries in the lungs and throughout both central and peripheral nervous systems. Liver and kidney were submitted for heavy metal analysis: mercury levels (parts per million, dry weight) were 260 (kidney) 31 (liver), and 60 (brain). Lead was not detected in any tissues.

### **Parovirus infection in raccoons**

In August, two groups of raccoons were submitted to the CCWHC from wildlife rehabilitation centres in southern Ontario. One submission consisted of 50 animals from a group of 72 which had died over a one week period; the other consisted of 6 animals. All were juveniles, 3-5 months of age, that had been raised as orphans. A sample of the first group and all of the second group were necropsied. In all animals there was a severe diffuse, fibrinous enteritis. Microscopically, there was extensive crypt necrosis and epithelial loss, consistent with Parovirus infection. Animals from both groups harboured large numbers of the raccoon roundworm, *Baylisascaris procyonis*. The majority of the animals were unvaccinated.

These cases demonstrate the importance of vaccination programs in rehabilitation facilities in preventing catastrophic mortalities in captive animals. An additional recommended prophylactic measure, of importance to public health, is a proper deworming program. *Baylisascaris* larvae in the environment constitute a serious zoonotic hazard, as a potential cause of visceral larva migrans. *Dr. Doug Campbell - CCWHC Ontario Region*

## **Western/Northern Region**

### **Phorate poisoning in Yukon wildlife**

Fourteen birds and mammals including: 2 wolves, 2 coyotes, 2 red fox, 2 golden eagles, 1 gull, 1 magpie, 1 northern harrier, and 2 ravens were found dead in the Kusawa - Dezadeash Lake area of the Yukon Territory during the spring of 1993. Necropsies determined that Phorate was responsible for the death of one wolf, one eagle, one red fox, one marsh hawk, one raven, and one gull. Others were likely killed by the pesticide as they were found within the "ring of death" associated with those that tested positive. Phorate, an organophosphate insecticide licensed for control of soil insects was confirmed to have been introduced illegally to the Yukon by Edwin

Peter Wiens of Tofield, Alberta. Wiens was convicted on April 20, 1993 in Territorial Court after pleading guilty to Wildlife Act charges of counseling two guides in his employ to place lard baits laced with Phorate to kill wolves. He was fined \$3,500 and prohibited from holding any licenses for a period of three years. Investigators are confident that all of the Phorate introduced into the Territory has been accounted for. The Department of Renewable Resources is now proposing new regulations under the Environment Act which would make possession of such pesticides illegal without a permit. Penalties would be a maximum of \$20,000 and up to 6 months imprisonment. *Tony Grabowski - Yukon Department of Renewable Resources*

### **Avian cholera outbreak in waterfowl in Manitoba**

Avian cholera, due to infection with the bacterium *Pasteurella multocida*, caused the death of several thousand waterfowl in Oak Hammock Marsh and over a large area approximately 20 km southwest of Selkirk, Manitoba. Dead waterfowl were first reported on October 24 and carcasses continued to be found into mid-November. Snow geese (429), Canada geese (500), mallard ducks (400+), 1 white pelican, 3 snowy owls, and 10 gulls were retrieved during clean-up. At least 20 eagles were seen in the vicinity of the die-off and eagles were observed feeding on waterfowl carcasses, but none were found dead. Avian cholera was confirmed in all 3 snowy owls.

Investigation and clean-up of this outbreak involved members of the Manitoba Department of Natural Resources, Canadian Wildlife Service and Ducks Unlimited. All retrieved birds were buried and covered with lime, scare cannons were used to disperse birds from affected areas, and road-killed deer and elk were placed in areas of high eagle concentrations to provide disease-free food to scavengers. *Information provided by Murray Gillespie - Manitoba Department of Natural Resources*

### **Announcements**

The Ontario Ministry of Natural Resources is holding the fifth Annual Meeting on "Rabies in the Americas" to be held in Niagara Falls, Ontario, Canada. The tentative dates are November 16-19, 1994. This Conference will be attended by delegates from North, South & Central America, as well as Europe and other parts of the world. If you would like to receive further information, send your name, address, and institution affiliation to the name below as soon as possible. If you would like to give a 20 minute oral presentation, please indicate this, along with your subject title.

Sarah Crosgrey  
Rabies Animal Facility  
Ministry of Natural Resources  
Midhurst District Office  
Midhurst, Ontario L0L 1P0  
Bus#: (705) 722 3663  
Fax#: (705) 722 5720

**Copyright © 1994, CCWHC**