illness, death, and reduced egg production following infection with pathogenic or disease causing strains. Prior to 1990, this disease had rarely been reported as a cause of mortality in the free-living native birds of the United States or Canada. Repeated large-scale losses of double-crested cormorants from ND in both countries has resulted in a need for enhanced awareness of ND as a disease of wild birds and, therefore, its inclusion within this Manual. Background information about ND in poultry is needed to provide a perspective for understanding the complexity of the disease agent, Newcastle disease virus (NDV). Some general information about ND in other avian species is also provided, but the primary focus for this chapter is the effect of NDV on double-crested cormorants.

This information on Newcastle disease is taken from the USGS Field Manual of Wildlife Diseases: Birds. Chapter 21: Newcastle Disease. It is accessible at http://www.nwhc.usgs.gov/publications/field_manual/chapter_21.pdf (last accessed on August 25, 2008). Only part of the information is presented here. For a more complete description of the disease, including information on seasonality and diagnosis, please visit the website. The article on page 5 of this issue of the Indiana Wildlife Disease News describes this Field Manual in more detail.

Newcastle Disease (ND) in domestic poultry is a focus for concern throughout much of the world's agricultural community because of severe economic losses that have occurred from widespread throughout the US, Canada, and select countries across the world. Significant die-offs of birds and fish have occurred regularly in Lakes Erie and Ontario. Since 2002, in excess of 52,000 avian botulism mortalities have been reported in the Great Lakes region.

The most basic form of this disease is held in a spore that will stay dormant in sediments of rivers, lakes, wetlands and streams until the right growing conditions are met. Outbreaks of the disease typically occur in the summer months, between July and September. This is a sensitive time for waterfowl, which are actively raising young and beginning to assemble flocks for migration. Low water levels...

Continued on pg. 2
Newcastle disease (continued from pg 1)

Cause

Newcastle disease is caused by infection with an RNA virus within the avian paramyxovirus-1 group. NDV is highly contagious and there is great variation in the severity of disease caused by different strains of this virus. A classification system for the severity of disease has been established to guide disease control efforts in poultry because of the economic damage of ND.

The most virulent ND form causes an acute, lethal infection of chickens of all ages with mortality in affected flocks often reaching 100 percent. These strains produce hemorrhagic lesions of the digestive tract, thus resulting in the dis-ease being referred to as viscerotropic or having an affinity for abdominal tissue, and velogenic or highly virulent Newcastle disease or VVND. This form of ND is rare in the United States, and it is primarily introduced when exotic species of birds are trafficked in the pet bird industry. Another acute, generally lethal infection of chickens of all ages affects respiratory and neurotropic tissues and is referred to as neurotropic velogenic Newcastle disease or NVND. Morbidity or illness from NVND may affect 100 percent of a flock, but mortality is generally far less with extremes of 50 percent in adult birds and 90 percent in young chickens. The NVND form of ND was essentially eradicated from the United States in about 1970, but it has occasionally been reintroduced via pet birds and by other means. A less pathogenic form of ND causes neurologic signs, but usually only young birds die and, except for very young susceptible chicks, mortality is low. These strains are classified as mesogenic or moderately virulent. NDV strains that cause mild or inapparent respiratory infections in chickens are classified as lentogenic or low virulence. Lentogenic strains do not usually cause disease in adult chickens, but these forms can cause serious respiratory disease in young birds. Some strains of lentogenic NDV cause asymptomatic enteric infections without visible disease.

The virus classification standard applies to ND in poultry and the standard is not directly transferrable to wild birds. Experimental studies have demonstrated differences in bird response to the same strain of NDV. Thus, a highly pathogenic strain isolated from wild birds may be less hazardous for poultry and vice-versa. ND may be transmitted among birds by either inhalation of contaminated particulate matter or ingestion of contaminated material.

Species affected

NDV is capable of infecting a wide variety of avian species. In addition to poultry, more than 230 species from more than one-half of the 50 orders of birds have been found to be susceptible to natural or experimental infections with avian paramyxoviruses. Experimental infections in mallard ducks exposed to large amounts of a highly virulent form of NDV for chickens disclosed that ducklings were more susceptible than adults, and that mortality of 6-day-old ducklings was higher than in 1-day-old and 3-day-old ducklings. Captive-reared gamebirds, such as pheasants and Hungarian partridge, have died of ND. However, large-scale illness and death from NDV in free-ranging wild birds has only occurred in double-crested cormorants in Canada and the United States. White pelicans, ring-billed gulls, and California gulls were also reported to have died from NDV in association with cormorant mortalities in Canada.

The 1990 epizootic of ND in Canada killed more than 10,000 birds, mostly double-crested cormorants. Mostly subadult cormorants died in these cormorant colonies. Losses in the United States have been primarily in nestlings and other young of the year. The total mortality attributed to ND during 1992 exceeded 20,000 birds. Mortality in Great Lakes cormorant colonies ranged from 2 to 30 percent, while that in Midwestern colonies was estimated to be 80 to 90 percent. In 1997, nesting failure of a cormorant colony at the Salton Sea in California was attributed to NDV. The total mortality in 1997 was about 2,000 cormorants. During the 1992 epizootic, a domestic turkey flock in the Midwestern United States was infected at the same time NDV occurred in cormorants near that poultry flock.

Distribution

Different strains of NDV exist as infections of domestic poultry and within other species of birds throughout much of the world. Highly pathogenic strains of NDV have spread throughout the world via three panzootics or global epizootics since ND first appeared in 1926. The first of these highly pathogenic strains appears to have arisen in Southeast Asia; it took more than 30 years to spread to chickens worldwide, and it was primarily spread through infected poultry, domestic birds, and products from these species. The virus responsible for the second panzootic involving poultry appears to have arisen in the Middle East in the late 1960s; it reached most countries by 1973, and it was associated with the importation and movement of caged psittacine species. The most recent panzootic also appears to have its origin in the Middle East, and it began in the late 1970s. This panzootic differs in that pigeons and doves kept by bird fanciers and raised for food are the primary species involved. NDV spread worldwide primarily through contact between birds at pigeon races, bird shows, and through international trade in these species. It has spread to chickens in some countries. A current question is whether or not the ND outbreaks that have occurred in double-crested cormorants are the beginning of a fourth panzootic.
Newcastle Disease (Continued from pg. 2)

In North American, NDV has caused disease in double-crested cormorants from Quebec to the West Coast. Most cormorant mortality has occurred in the upper Midwest and the Canadian prairie provinces, although smaller outbreaks have occurred at the Great Salt Lake, in southern California, and on the Columbia River between Washington and Oregon. Cormorants, the closely related shag, and gannets, which are another species of marine bird that has close associations with cormorants, were believed to be an important source of NDV for the poultry outbreaks along the coast of Britain during the 1949–51 epizootic in that country.

Field Signs

Clinical signs, observed only in sick juvenile double-crested cormorants, include torticollis or twisting of the head and neck, ataxia or lack of muscular coordination, tremors, paresis or incomplete paralysis including unilateral or bilateral weakness of the legs and wings, and clenched toes. Paralysis of one wing is commonly observed in birds surviving NVD infection at the Salton Sea in southern California.

Experimental inoculations in adult mallard ducks with a highly virulent form of NDV from chickens resulted in onset of clinical signs 2 days after inoculation. Initially, mallards would lie on their sternum with their legs slightly extended to the side. As the disease progressed, they were unable to rise when approached and they lay on their sides and exhibited a swimming motion with both legs in vain attempts to escape. Breathing in these birds was both rapid and deep. Other mallards were unable to hold their heads erect. By day 4, torticollis and wing droop began to appear, followed by paralysis of one or both legs. Muscular tremors also became increasingly noticeable at this time.

Human Health Considerations

NDV is capable of causing a self-limiting conjunctivitis or inflammation of the membrane covering the eyeball and a mild flu-like disease in humans. Most reported cases in humans have occurred among poultry slaughterhouse workers, laboratory personnel, and vaccinators applying live virus vaccines. Aerosols, rather than direct contact, are most often involved as the route for transmission to humans.

In North American, NDV has caused disease in double-crested cormorants from Quebec to the West Coast. Most cormorant mortality has occurred in the upper Midwest and the Canadian prairie provinces, although smaller outbreaks have occurred at the Great Salt Lake, in southern California, and on the Columbia River between Washington and Oregon. Cormorants, the closely related shag, and gannets, which are another species of marine bird that has close associations with cormorants, were believed to be an important source of NDV for the poultry outbreaks along the coast of Britain during the 1949–51 epizootic in that country.

Field Signs

Clinical signs, observed only in sick juvenile double-crested cormorants, include torticollis or twisting of the head and neck, ataxia or lack of muscular coordination, tremors, paresis or incomplete paralysis including unilateral or bilateral weakness of the legs and wings, and clenched toes. Paralysis of one wing is commonly observed in birds surviving NVD infection at the Salton Sea in southern California.

Experimental inoculations in adult mallard ducks with a highly virulent form of NDV from chickens resulted in onset of clinical signs 2 days after inoculation. Initially, mallards would lie on their sternum with their legs slightly extended to the side. As the disease progressed, they were unable to rise when approached and they lay on their sides and exhibited a swimming motion with both legs in vain attempts to escape. Breathing in these birds was both rapid and deep. Other mallards were unable to hold their heads erect. By day 4, torticollis and wing droop began to appear, followed by paralysis of one or both legs. Muscular tremors also became increasingly noticeable at this time.

Human Health Considerations

NDV is capable of causing a self-limiting conjunctivitis or inflammation of the membrane covering the eyeball and a mild flu-like disease in humans. Most reported cases in humans have occurred among poultry slaughterhouse workers, laboratory personnel, and vaccinators applying live virus vaccines. Aerosols, rather than direct contact, are most often involved as the route for transmission to humans.


Recent Wildlife Disease Activity

Veterinarian Advises Caution, Not Panic,

INDIANAPOLIS (22 July 2008)—Whitley County residents should not be alarmed at the recent local discovery of a rabid bat in the region. While the diagnosis indicates the presence of the disease in the area, all Hoosiers should take a few common sense precautions every day to protect their families and pets against the disease, regardless of where they live.

Rabies is a viral disease spread primarily through the bite of an infected animal. Dr. Sandra Norman, a veterinarian with the Indiana State Board of Animal Health, recommends the following guidelines to reduce the risk of rabies exposure to people and animals:

• Avoid contact with wild animals (not just bats) at all times. Do not feed or handle wild animals, and secure any trash and pet food in animal-proof containers. Cover attic and chimney openings and other entry points in the home which may invite unwanted visitors.

• Wild animals are generally active at night and avoid contact with people. Daytime contact with humans is unusual, and should be viewed suspiciously.

• Indiana law requires all dogs, cats and ferrets 3 months of age or older be vaccinated against rabies by a licensed, accredited veterinarian. Pets should be kept close to home, as free-roaming animals are at higher risk of exposure to the disease.

• If your pet is bitten or attacked by a wild animal, contact your veterinarian and local animal control. Your pet will need a booster if the animal is determined to be rabid.

• If you or someone in your family is bitten or scratched by a wild or stray animal or a pet, attempt to confine or kill the animal or determine the owner of the pet (if this can be done without risk of further injury). Wear leather or latex gloves when handling all animals with possible exposure (dead or alive).

• Immediately wash the wound thoroughly with soap and water. Call your physician at once to determine treatment and make sure the bite is reported to the local department of health and animal control.

For more information about rabies prevention and safety, visit the Indiana State Board of Animal Health online at www.boah.in.gov.

Source: BOAH
Wildlife Disease Resources in Focus

The USGS Field Manual of Wildlife Disease: General Field Procedures and Diseases of Birds

The USGS Field Manual of Wildlife Diseases is an excellent reference for biologists, property managers, and others who manage birds or are involved with bird diseases. Even better, the entire manual can be found on-line at http://www.nwhc.usgs.gov/publications/field_manual/ and can be downloaded in PDF format in its entirety. Below is the Foreword for the Field Manual explaining the importance of this manual.

DO WILDLIFE DISEASES REALLY MATTER? The waterfowl manager who wakes up one morning to find ten thousand dead and dying birds in the marsh would think so. Yet virtually every wild bird and mammal harbors at least a few parasites seemingly without obvious adverse consequences. Parasites, viruses, bacteria, and fungi are component parts of the ecosystems in which wildlife are found, but do not necessarily cause disease. Millennia of coevolution have engendered a modus vivendi that assures the survival of both host and parasite populations.

Then why the ten thousand sick and dying birds? Ecosystems are changing. Waterfowl are concentrated on shrinking wetlands and remain there for longer periods of time, facilitating bird-to-bird spread of the bacteria that cause avian cholera. Or permitting the buildup of parasites in their hosts from a small, relatively benign number to massive numbers that cause disease and death. Water quality of wetlands changes, favoring the production of deadly botulism toxin by bacteria and its mobilization up the food chain to waterfowl. New, totally artificial habitats are created with unpredictable results. The extreme temperature, salinity, and other conditions of the Salton Sea have created an unusual ecosystem in which botulism occurs in fish and in birds through biological cycles that are not yet understood.

Wetland loss in southern California leaves few alternative places for water-birds to go, so they are attracted to the Salton Sea. Behavior changes. Mallard ducks take up residence on the ponds and lakes of city parks and lose their migratory habits. They share these bodies of water with exotic species, such as Muscovy ducks that have also taken up residence there after introduction by people, setting the scene for outbreaks of duck plague, and creating the risk of spread to migratory waterfowl that also use these areas. Raccoons and skunks become well adapted to urban life, bringing rabies and canine distemper with them into the city.

The environment changes the physiology of wild animals. Human activity introduces into wildlife habitats chemical compounds that adversely affect physiological processes such as reproduction and immune responsiveness. These compounds become incorporated into the ecosystems, often becoming more concentrated as they move up food chains. Their effects can influence wildlife populations. Some of these endocrine-disrupting chemicals, such as chlorinated hydrocarbons (DDE, PCBs), interfere with normal endocrine function by mimicking natural hormones, with resulting eggshell thinning and breakage. Effects of these chemical compounds on immune-system responses to infectious and parasitic agents are less well understood.

What to do? Incorporating disease-prevention measures into wildlife management practices requires more information than is usually available. The information-gathering process must begin in the field. Field biologists must monitor disease occurrence. This Field Manual is a valuable aid in identifying the diseases that are likely to be present, and in giving guidance on the gathering and treatment of specimens needed to establish the diagnosis in the laboratory.

But the wildlife field biologist is in a position to provide valuable information that goes beyond the collection of samples from sick and dead individuals. Although diseased individuals are the basic unit of surveillance, the occurrence of disease must be put into an ecological perspective. A careful description of the ecological setting in which the disease is occurring, and any changes that have occurred over time, are ultimately as important as a careful description of the lesions observed in the individual, if the epidemiology of that disease is to be understood, and the disease prevented through sound wildlife management practices.

It is my hope that the awareness of diseases affecting wildlife and the good disease-surveillance practices promoted by this manual will spread throughout the range of the species we are trying to manage and protect. We must know more than we do currently about disease occurrence throughout the ranges that the wildlife occupy. Many migratory species know nothing of international boundaries. Neither do their diseases. Until we have a much more complete picture of the disease-environment relationships of the blue-winged teal from its nesting ground in Canada, its migration route through the United States and overwintering areas in Central America or the Cienaga Grande de Santa Marta in Colombia, sound disease-prevention management of that species will not be possible. Similar considerations exist for other species.

Thomas M. Yuill
Madison, Wisconsin May, 1999

Virulent Newcastle Disease Confirmed in Minnesota

September 5, 2008

The USGS National Wildlife Health Center (NWHC) is investigating double-crested cormorant mortalities from multiple locations in Minnesota. Biologists from the Minnesota Department of Natural Resources (MNDNR) contacted the NWHC after discovering dead double-crested cormorants in mid-July 2008. To date, combined cormorant mortalities are estimated to be over 1,200 in the counties of Meeker, Faribault, Mille Lacs, Cass, St. Louis (Voyageurs National Park), Lake of the Woods, and Lac Qui Parle. After preliminary testing at NWHC, samples were sent to the USDA National Veterinary Services Laboratory where virulent Newcastle disease was confirmed in samples from four of the counties. Final results from samples from the other counties are pending.

Newcastle disease (ND) is highly contagious and capable of infecting a variety of avian species. Double-crested cormorants are the wild birds most commonly affected with virulent ND virus. Prior to 1990, virulent ND had not been reported as a cause of mortality in free-living native birds of the U.S. or Canada. In 1992, multiple mortality events occurred in double-crested cormorant colonies across the Great Lakes, upper Midwest, and Canada with over 35,000 birds estimated dead. Sporadic outbreaks in cormorants have occurred in California, Utah, Nevada, and Oregon since 1990.

For the domestic poultry industry, ND is a concern because of severe economic losses from illness, death, reduced egg production, and potential trade restrictions. ND is transmitted through direct contact, feces, and excretions from infected birds. Quarantine and disinfection are necessary for any areas, clothing, or equipment that may have come in contact with infected poultry. ND is not a major concern for human health although it may cause a mild conjunctivitis and influenza-like symptoms. Further investigations are in progress to assess the extent of the outbreak. NWHC staff are working with officials from the National Park Service, MNDNR, USDA Wildlife and Veterinary Services, the MN State Veterinarian’s office, and the Canadian Cooperative Wildlife Health Centre to manage the outbreak by reducing possible impacts to wild birds and preventing spread to domestic poultry.

Wildlife Services working to investigate cormorant deaths (Photo: USDA)

Source: NWHC

69th Midwest Fish and Wildlife Conference

The 69th Midwest Fish & Wildlife Conference is coming soon! This year’s conference will be held December 14 – 17, 2008 at the Hyatt Regency in downtown Columbus, Ohio. We hope that you will find the heart of the Buckeye State a great venue for exploring the Future of Fish and Wildlife Management. From great hotel accommodations and a variety of casual dining opportunities to excellent meeting facilities at the Hyatt Regency, our planning committee hopes to provide you with an enjoyable stay and an excellent technical program.

Workshops will include:
- Abundance Estimation and Detection Probability - State of the Art
- Advancing Natural Resource Management Through Meeting Facilitation
- Introduction to Spatial Modeling Using ArcGIS and Geostatistical Analyst
- Making Human Dimensions Research Part of Your Research Agenda

Symposia will include:
- Wind Energy/Wildlife interactions: Assessing Risk and Minimizing Impacts
- Invasive Species Spread By Recreational Boaters: Impacts and Prevention Strategies
- Noninvasive Survey Techniques for Carnivores
- Long-Term Analyses and Ecological Thresholds
- Understanding Midwestern Reservoir Function By Building on Existing Conceptual Models
- Squeezing the Most Out of the Farm Bill Conservation Title
- Uses of Otolith Chemistry for Midwest Fisheries Management
- Nocturnal Bird Research and Monitoring
- National Estuarine Research Reserve

Abstracts for papers and posters are due September 2, 2008. Registration is due December 8, 2008. More details about the conference can be found at the conference website at www.2008mwfwc.com

Recent Wildlife Disease News

Michigan's First Case of Chronic Wasting Disease Detected at Kent County Deer Breeding Facility

August 25, 2008

LANSING - The Michigan departments of Agriculture (MDA) and Natural Resources (DNR) today confirmed the state’s first case of Chronic Wasting Disease (CWD) in a three-year old white-tailed deer from a privately owned cervid (POC) facility in Kent County.

The state has quarantined all POC facilities, prohibiting the movement of all - dead or alive - privately-owned deer, elk or moose. Officials do not yet know how the deer may have contracted the disease. To date, there is no evidence that CWD presents a risk to humans.

DNR and MDA staff are currently reviewing records from the Kent County facility and five others to trace that deer which has been purchased in the last five years of deer and the last seven years for elk. Any deer that may have come in contact with the CWD-positive herd have been traced to their current location and those facilities have been quarantined.

"Michigan’s veterinarians and wildlife experts have been working throughout the weekend to complete their investigation," said Don Koivisto, MDA director. "We take this disease very seriously, and are using every resource available to us to implement response measures to stop the spread of this disease."

CWD is a fatal neurological disease that affects deer, elk and moose. Most cases of the disease have been in western states, but in the past several years, it has spread to some midwestern and eastern states. Infected animals display abnormal behaviors, progressive weight loss and physical debilitation.

Current evidence suggests that the disease is transmitted through infectious, self-multiplying proteins (prions) contained in saliva and other fluids of infected animals. Susceptible animals can acquire CWD by direct exposure to these fluids or also from contaminated environments. Once contaminated, research suggests that soil can remain a source of infection for long periods of time, making CWD a particularly difficult disease to eradicate.

Michigan’s First Case of Chronic Wasting Disease Detected at Kent County Deer Breeding Facility: "Currently, one of our top concerns is to confirm that the disease is not in free-ranging deer," said DNR Director Rebecca Humphries. "We are asking hunters this fall to assist us by visiting check stations to allow us to take biological samples from the deer they harvest, so we can perform adequate surveillance of the free-ranging white-tailed deer herd in the area."

Deer hunters this fall who take deer from Tyrone, Solon, Nelson, Sparta, Algoma, Courtland, Alpine, Plainfield, and Cannon townships will be required to bring their deer to a DNR check station. Deer taken in these townships are subject to mandatory deer check.

The DNR is also asking hunters who are participating in the private land five-day antlerless hunt in September in other parts of Kent County to visit DNR check stations in Kent County so further biological samples can be taken from free-ranging deer for testing. The DNR is in the process of finding additional locations for check stations in Kent County to make it more convenient for hunters.

The deer that tested positive at the Kent County facility was a doe that had been recently culled by the owner of the facility. Michigan law requires sick deer or culled deer on a POC facility be tested for disease. The samples from the Kent County deer tested “suspect positive” last week at Michigan State University Diagnostic Center for Population and Animal Health, and were sent to the National Veterinary Services Laboratory in Ames, Iowa last Thursday for confirmatory testing. The positive results of those tests were communicated to the state of Michigan today.

Audits of the facility by the DNR in 2004 and 2007 showed no escapes of animals from the Kent County facility were reported by the owner. Also, there were no violations of regulations recorded during the audits.

Since 2002, the DNR has tested 248 wild deer in Kent County for CWD. In summer 2005, a number of those deer had displayed neurological symptoms similar to CWD; however, after testing it was determined the deer had contracted Eastern Equine Encephalitis.

More information on CWD is available on Michigan’s Emerging Diseases Web site at www.michigan.gov/chronicwastingdisease.

Botulism (Continued from pg. 1)

and warm waters (18-25 °C), low in oxygen are good sites for harboring botulism spores. In Lake Erie, benthic organisms like the amphipod and zebra mussels, have tested positive for the bacteria. Many scientists believe that zebra and quagga mussels have the potential for filtering the bacteria and passing it up the food chain. Invasive mussels may be responsible for the increase in the growth of the algae Cladophora, which may be tied to botulism outbreaks. Benthic fishes such as the freshwater drum, smallmouth bass, and round gobies were found to harbor the bacteria in their livers and intestines. Channel catfish and lake sturgeon can also be affected, as well as amphibians like mudpuppies.

Infected fish usually die quickly and most likely wash up on shore. However, prior to death they may be easy prey for fish-eating birds which is one way the toxin may pass up the food chain. Round goby lack swim bladders and they will normally remain on the bottom after death but they may become entangled in mats of algae which may harbor the toxins also. Water movement such as in a stream or wetland can increase the oxygen content, decrease water temperature and decrease the potential for a botulism outbreak.

While botulism affects birds, migratory waterfowl, and aquatic organisms, such as fish and mussels, the threat to humans is minimal. When birds contract botulism, they can potentially be treated and survive. Those that do not survive are left to die and decompose, quickly becoming hosts to maggots and fly larvae who become instant carriers of the disease. Red-breasted mergansers, ring-billed gulls, common loon, long-tailed ducks, and mallards are just a few known to be affected. Because of the conditions causing botulism, death of one bird from botulism in a “hot spot” can in turn cause the death of more birds and aquatic species.

In the heat of the summer district wildlife biologists often hear of mallard and Canada goose deaths on ponds or in urban water bodies around the state. These deaths can often be attributed to Type E botulism. Mallards and geese are thought to pick up the toxin by eating insect larvae that have been feeding on dead ducks or fish.

Once infected by the neurotoxin, birds lose voluntary movement like the ability to move their wings for flight, as well as the loss of motion in their legs. Paralysis takes place in the inner eyelid and then the neck muscles. Loss of movement in the neck ultimately results in drowning because the birds can no longer keep their head upright. It is important to note that death of infected waterfowl can also occur from water deprivation, electrolyte imbalance, respiratory failure or even predation. The infected waterfowl never show signs of any lesions or damage to skin and feathers.

While enjoying the outdoors a person is not at risk from botulism poisoning while swimming, or enjoying recreational activities in the region. Botulism is only contracted by ingesting fish or birds contaminated with the toxin. As a precaution, any fish or waterfowl that are sick or act abnormal should not be harvested or eaten. Pets should not be allowed to eat dead wildlife, as they may contain the toxin. If cleaning up along a Great Lakes beach, it is important to wear rubber gloves when handling dead or decaying wildlife. The infected waterfowl never show signs of any lesions or damage to skin and feathers.

While enjoying the outdoors a person is not at risk from botulism poisoning while swimming, or enjoying recreational activities in the region. Botulism is only contracted by ingesting fish or birds contaminated with the toxin. As a precaution, any fish or waterfowl that are sick or act abnormal should not be harvested or eaten. Pets should not be allowed to eat dead wildlife, as they may contain the toxin. If cleaning up along a Great Lakes beach, it is important to wear rubber gloves when handling dead or decaying wildlife. The infected waterfowl never show signs of any lesions or damage to skin and feathers.

While enjoying the outdoors a person is not at risk from botulism poisoning while swimming, or enjoying recreational activities in the region. Botulism is only contracted by ingesting fish or birds contaminated with the toxin. As a precaution, any fish or waterfowl that are sick or act abnormal should not be harvested or eaten. Pets should not be allowed to eat dead wildlife, as they may contain the toxin. If cleaning up along a Great Lakes beach, it is important to wear rubber gloves when handling dead or decaying wildlife. The infected waterfowl never show signs of any lesions or damage to skin and feathers.

Botulism is only contracted by ingesting fish or birds contaminated with the toxin. As a precaution, any fish or waterfowl that are sick or act abnormal should not be harvested or eaten. Pets should not be allowed to eat dead wildlife, as they may contain the toxin. If cleaning up along a Great Lakes beach, it is important to wear rubber gloves when handling dead or decaying wildlife. The infected waterfowl never show signs of any lesions or damage to skin and feathers.
Midwest Wildlife Disease Update (Continued from pg. 7)

in a social group of females, other females in the group continue to test negative. She says this suggests that CWD is not spreading rapidly among females within social groups. One alternate hypothesis is that areas where deer congregate - mineral licks, for example - may become hotspots for the disease. In those areas deer frequently lick the soil. They leave behind saliva that may also contain prions. Whether that behavior and the consumption of contaminated soil is at all associated with transmission is speculative, Mathews emphasizes, "but we can't rule out deer congregating around hot spots as another means of transmission." (Source- Science Daily, edited)

West Nile Virus (WNV) Still An Issue- Dr. Michael Sinsko, an entomologist with the Indiana Board of Health, reported that WNV transmission in Indiana continues to increase and spread with 102,237 mosquitoes tested from 91 counties. Of these, 32 positive pools of mosquitoes were discovered in 12 counties as of mid-summer. The positive counties may be viewed at http://diseasemaps.usgs.gov. The first human case, classified as a probable case, has been reported from Perry County. As of September 1, only two birds submitted for testing have been positive for WNV. Under normal summer conditions, transmission is likely to continue through the end of September. The public should take precautions to prevent mosquito bites, including use of repellents, wearing long sleeved clothing, and avoiding outdoor activity during prime mosquito activity. In addition, breeding sites for WNV mosquitoes should be eliminated when possible, such as clogged rain gutters, buckets, tires, unused wading pools and ornamental pools. (Source- DNR Internal Memo, 08/21/08)

Mysterious Bat Deaths Continue- Researchers now think that a fuzzy white fungus found on thousands of dead and dying bats in New England and New York last winter might be the primary cause of the illness. Scientists have learned that the unidentified fungus seems to thrive in cold temperatures found in caves and mines in winter when bats are hibernating there. Bats have continued to die this spring. Hundreds of the animals discovered in Vermont, Massachusetts, and New Hampshire through June have scarred wings believed caused by the fungus, and researchers say the wing damage can kill the bats as well. (Source: Boston Globe, 07/28/08)

West Virginia CWD Update- An additional eleven white-tail deer collected this spring have tested positive for CWD in West Virginia. That brings the total to 31 deer testing positive in Hampshire County. West Virginia’s first case of CWD came in September 2005. The spring samples were taken by DNR Wildlife Resources Section sharpshooter teams with the cooperation of private landowners. (Source- Cumberland Times-News, edited)

Minnesota DNR Reduces Deer Herd To Stop TB Spread- Sharpshooters have finished for now trying to dramatically thin the deer herd in part of northwestern Minnesota plagued by bovine tuberculosis. Shooting from the ground and a helicopter, federal and private sharpshooters killed 962 deer - more than had been estimated to inhabit the 164-square-mile core area in January. The sharpshooting began in February 2008 and ended in mid-May.

The total deer killed since September, including those shot by sharpshooters, hunters and landowners in the hunting season and after it is 2,656, the Department of Natural Resources reported Tuesday. "We’ve reduced numbers dramatically in the core area," said Paul Telander, DNR regional wildlife manager in Bemidji. He said officials hope that the unprecedented action will curb the spread of the disease to cattle herds.

Eight of the 962 deer killed by sharpshooters are suspected of having bovine TB. Two have tested positive; results are pending on the others. Since 2005, 20 deer have tested positive. Officials say the TB originated with infected cattle and has been found in 11 cattle herds in Roseau and Beltrami counties since 2005, and spread to wild deer. (Source- Minneapolis-St. Paul Star-Tribune)

LaCrosse Encephalitis Appears In Wisconsin- A local health department has confirmed two cases of LaCrosse encephalitis in Polk County, Wisconsin. Bonnie Leonard, Public Health Supervisor, says one of the patients will make a full recovery, the other patient’s status is unknown. This encephalitis is a mosquito-borne disease, and cannot be transmitted from person to person. Continued on pg. 9
The virus is transmitted by *Aedes triseriatus* and more recently the virus has been isolated from the Asian tiger mosquito, *Aedes albopictus*. In the USA, particularly in the Upper Midwest, an average of 70 clinical cases are reported annually, the vast majority in preadolescent children. (Source: ProMed, 08/21/08)

Lake Perch Tumor- On July 14, 2008 an eleven inch lake perch (Perca flavescens) weighing 350 grams was pulled from Lake Michigan near the Indiana/Illinois border. Because the fish had an unusually large abdomen it was submitted to the Purdue Animal Disease Diagnostic Lab (ADDL) for necropsy. Dr. T.L. Lin of the ADDL reported the abdomen contained a mass measuring 8 cm (length) by 7 cm (width) by 5 cm (height), and weighed 95 grams. The mass was diagnosed as a testicular tumor with fibrosis and adhesion to the peritoneum. The liver, stomach and intestine adjacent to the mass were compressed by the mass. Gonadal tumors in the testicle of yellow perch in the Great Lakes are thought to originate from the smooth muscle components of the testicle. (Source: Dean Zimmerman and ADDL report)

Tumor in lake perch from Indiana. Notice swollen abdomen. (Photo: D. Zimmerman)