Aspergillosis

Causes and Susceptible Species

Aspergillosis is a disease affecting the respiratory tract of birds and mammals. In regards to wildlife, aspergillosis is almost entirely confined to birds. It is caused by fungi in the genus *Aspergillus*, most commonly *A. fumigatus*. These fungi are ubiquitous in the environment and are especially common in soil and decaying plant matter. Birds are constantly exposed to the spores of *Aspergillus*. Under normal circumstances a bird's immune system will prevent infections from developing. If the immune system has been compromised, or if the bird has been exposed to an overwhelming number of spores, chronic or acute forms of the disease may develop.

The chronic form of the aspergillosis generally occurs in birds that have been weakened and stressed by malnutrition, injury, other disease, or exposure to toxicants. In the wild in New York, chronic aspergillosis is most often seen in gulls, common loons and raptors. It is a fairly common complication in wild birds that are held captive for rehabilitation.

Symptoms and Diagnosis

Chronic aspergillosis typically starts as small plaques on air sac walls. Plaques may grow, coalesce and completely cover the interior lining of air sacs. They may also form large rubbery masses that envelope blood vessels, particularly in the vicinity of the heart. Mature lesions often include sites of spore production, manifested by a dusty-looking, grey-blue/green surface; i.e. looks like fruiting mold on spoiled food. Despite, the fairly long-term growth in parts of the respiratory tract, actual lung involvement seems to occur only in the terminal phase of disease progression. Clinically, birds with chronic aspergillosis are thin (especially breast muscle), and are often reluctant to fly or are incapable of sustained flight.

The acute form of the disease is triggered by inhalation exposure to massive numbers of spores. In New York outbreaks are occasionally seen in potentially granivorous waterfowl (mallard ducks, Canada geese) feeding on moldy silage. Acute aspergillosis directly affects the lungs and is characterized by the development of small (1-3 mm diameter), yellow-white nodules throughout the lungs. The disease progresses rapidly over a period of several days and the birds typically die without noticeable weight loss.

Preventing the Spread of Aspergillosis

People who feed birds have been traditionally warned about feeding birds moldy birdseed or bakery products. This directive has a sound basis and may in part account for the fact that we rarely see aspergillosis in songbirds, including those found sick or dead near bird feeders (see Salmonellosis and House Finch Conjunctivitis). Live or dead birds with aspergillosis pose no significant threat to human health.

Bird Flu (Avian Influenza)

Frequently Asked Questions About Bird Flu

Q) What is bird flu?

A) Avian influenza, also called bird flu, is a disease of birds that is found primarily in wild waterfowl such as ducks, geese and swans. Sometimes, this disease can also spread from wild birds into domestic poultry. There are many strains or types of bird flu. Right now there is a type of bird flu called Highly Pathogenic Avian Influenza (HPAI) type H5N1 (also called H5N1 bird flu) that has made many birds and a few people sick elsewhere in the world. For more information, you can view the U.S. Fish and Wildlife Service publication, "Avian Influenza: Be on the Lookout" (pdf, 574 kb).

Q) Is the highly pathogenic avian influenza, H5N1 bird flu present in the United States?

A) No, this type of bird flu has not been found in the United States. Other strains of bird flu are commonly found in wild waterfowl in the United States, but usually affect small numbers of birds and generally do not cause obvious illness. These other types of bird flu are not considered a human health risk. The H5N1 bird flu is now circulating in Asia, Europe and Africa.

Information About Bird Flu and Pets

Q) Can pets get bird flu?

A) Yes. In some places where H5N1 bird flu has occurred, cats and other mammals have gotten sick and died after eating infected birds.

Q) Can my pets get vaccinated against bird flu?

A) No. But there are things you can do to protect your pet.

Q) What can I do to protect my pets from bird flu?

A) If you are worried about your pets, do not let them roam outside where they could be exposed to, or eat the remains of sick or dead wildlife. Many diseases can cause wild birds and other animals to get sick and die, and some diseases could be spread to pets that run free.

Q) My pet has been exposed to a dead bird, Can my pet be tested?

A) Routine testing of pets for bird flu is not necessary and currently unavailable. If you have concerns about your pet's health, it is best to contact a veterinarian.

Q) Can my pet give me bird flu?

A) There have been no confirmed cases of bird flu transmission between humans and pets. If H5N1 bird flu occurs in our country, it will be important to protect pets from possible exposure to sick birds and wildlife so that they will not get infected.

Q) How do I know that animals from a pet store don't have bird flu?

A) It's always best to make sure your pet has been checked by a veterinarian prior to purchase. There have been no confirmed cases of bird flu being transmitted from pets to humans.

Information about Bird Flu and Wild Birds

Q) Are you going to test dead wild birds for bird flu?

A) Currently, certain wild birds are being tested so that if H5N1 bird flu occurs in the United States we will recognize it right away. State and Federal agriculture and wildlife agencies have a list of birds that are of most concern and have begun testing these birds. Most birds do not need to be tested.

Q) What birds are being tested?

A) Waterfowl, such as ducks and geese, are a top priority to be tested. The vast majority of backyard birds-robins, sparrows, pigeons, cardinals, etc.-do not need to be reported or tested. Avian influenza usually involves migratory waterfowl, not backyard birds. If you are concerned about dead waterfowl in your area, contact your regional Department of Environmental Conservation office.

Q) If most birds are not being tested for disease, why am I still being asked to report dead crows?

A) Dead crow reports help us to know whether West Nile virus is present in an area. Just knowing where crows are dying tells us a lot. Dead crow reports are still important to help us track West Nile virus, but most crows will not be picked up or tested.

Q) I found a dead bird in my yard-what should I do?

A) First, there is no need to report a dead bird unless it is a crow. Dead crow reports help us to track West Nile virus, so please call 1-866-537-BIRD if you see a dead crow. Other types of birds do not have to be reported unless there are many dead birds in the same area. Call your regional Department of Environmental Conservation (DEC) office if you see a lot of dead birds in the same place. DEC will decide if testing is needed. To dispose of a dead bird, use a shovel and wear gloves to double-bag the dead bird and throw it in the trash, or bury it at least three feet deep, away from a stream or other water source. Always wash hands after disposing of a dead bird in this way.

Q) Should we stop feeding birds and not have bird feeders?

A) There is no need to change your normal practices for feeding backyard birds at this time. If the H5N1 bird flu does occur in our country, experts may have different advice, depending on what has been learned about the role of wild birds in spreading bird flu to humans.

Q) Should I feed ducks, geese and other waterfowl?

A) Unlike backyard birds, waterfowl are more likely to be infected when bird flu is present. But there are many other reasons that you should not feed ducks and geese. It increases the chance of spreading many diseases that are common among waterfowl. It makes them tame, and causes them to become a nuisance. They lose their natural behaviors. It is best to enjoy your local wildlife from a distance!

Q) Should I stop hunting waterfowl?

A) No. However, waterfowl hunters should always take simple precautions to protect themselves from exposure to disease, including:

- Do not handle obviously sick birds or birds found dead.
- Keep your game birds cool, clean and dry.
- Do not eat, drink or smoke while cleaning harvested waterfowl.
- Wear rubber gloves when cleaning waterfowl.
- Wash your hands with soap and water after cleaning waterfowl.
- Clean up tools and surfaces immediately with hot, soapy water and disinfect with a mixture of 10 percent household chlorine bleach in water.
- Thoroughly cook harvested waterfowl (165º Fahrenheit).

Human Exposure to Bird Flu

Q) Can I get bird flu from my neighbor's birds and animals?

A) No, you don't have to be concerned that neighbor's poultry and animals will expose you to bird flu at this time. If H5N1 bird flu does occur in our country, additional guidance from experts will be developed. Bird flu is primarily a disease among birds and rarely spreads to other animals and humans. Most people who got sick with bird flu had come into contact with infected chickens or domestic ducks and touched them with their bare hands.

Q) With the concern over bird flu, is it safe for my child to take part in projects that involve hatching eggs and raising chicks?

A) Yes. Chickens that get infected with bird flu become ill and often stop laying eggs so there is little risk of bird flu. However, chicks can carry other diseases such as salmonella. Projects involving hatching eggs and raising chicks should minimize hand contact and require thorough hand washing if contact does occur.

Q) With the concern over bird flu, is it safe to eat poultry and eggs?

A) Yes. There is no evidence that properly cooked poultry or eggs can be a source of infection from bird flu. Because other common diseases such as salmonella infection can be spread by eating undercooked poultry or eggs, always cook them thoroughly. Wash your hands with soap and hot water after touching any raw meat. Make sure to clean cutting boards and counters used for food preparation immediately after use to prevent cross contamination with other foods.

Q) Can I get bird flu from Canada goose droppings in parks, ballparks, reservoirs and other public places?

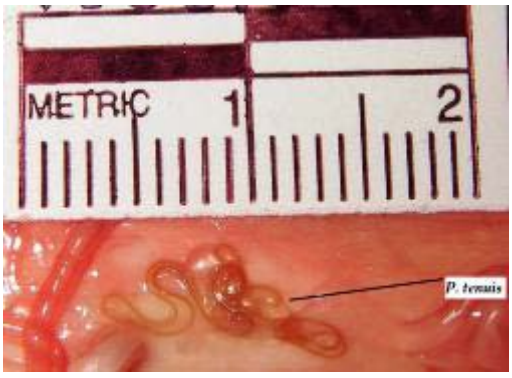
A) No. Right now, the H5N1 bird flu is not present in the United States. If and when it does occur in our country, we hope to have learned more about the role of wild birds in spreading bird flu to humans. Because many different bacteria, viruses and parasites can be present in bird droppings, it is best to avoid exposure to them at all times. The following precautions should be taken:

- Teach children to always wash their hands after playing outside.
- If you pick up droppings, use a shovel, "pooper scooper," or gloves-never your bare hands.
- If you are worried about exposure during swimming, swim at a regulated beach, where regular tests are conducted to make sure the water is not polluted from human, animal or farm waste.

Brain worm

Other Names: "moose sickness", *Parelaphostrongylus tenuis (P. tenuis*), meningeal worm, cerebrospinal nematodiasis, cerebrospinal Parelaphostrongylosis.

What Is It?

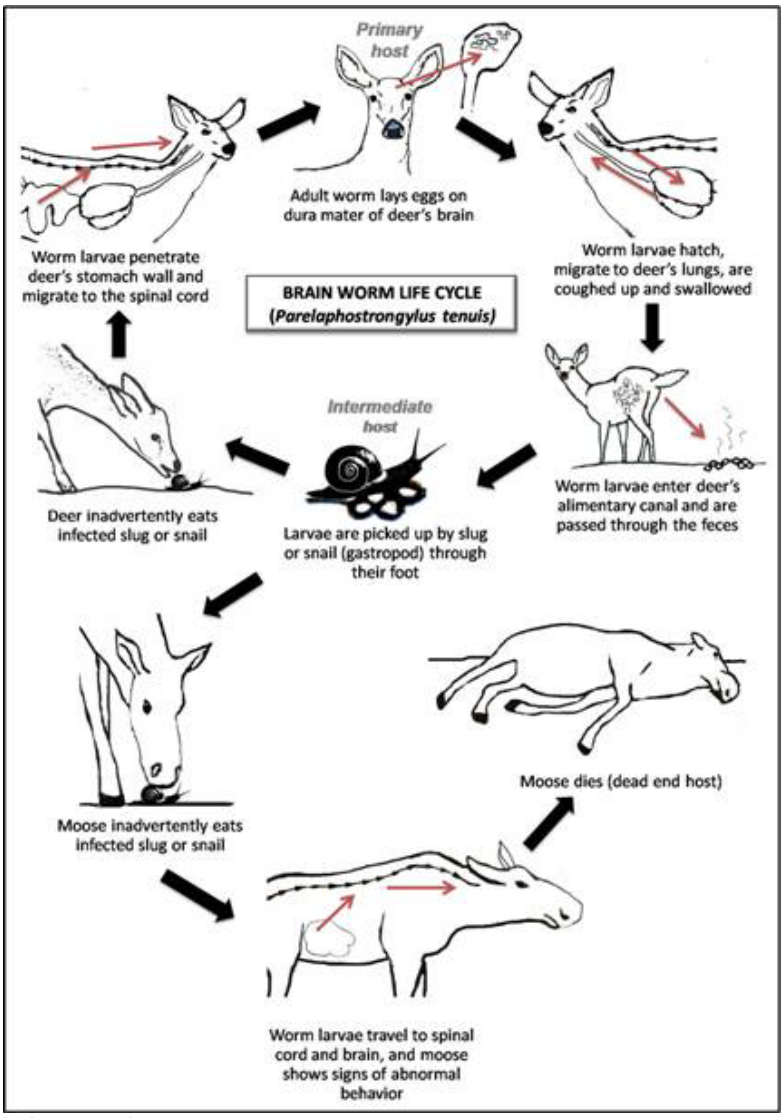


An adult brain worm (P. tenuis) on thespinal cord of a moose submittedfor diagnosis.
~Photo by DEC's Wildlife Pathology Unit~

Brain worm is the term commonly applied to the parasitic nematode (round worm), *Parelaphostrongylus tenuis (P. tenuis)*, which requires a living host to survive. The parasitic worm is frequently found in the subdural spaces (between the brain and the walls of the cranium) in white-tailed deer, which is its definitive (normal) host. The white-tailed deer is usually unaffected by the parasite; in fact, most adult deer in northern New York are parasitized by *P. tenuis*.

Although brain worm shows no apparent effect on white-tailed deer, it is often fatal to moose, mule deer, black-tailed deer, elk, caribou/reindeer, llama, alpaca, goats, and sheep.

Life Cycle



~Illustration by Natalie Sacco, NYSDEC~

Life Cycle Description in a Deer

- Parasitic adult worms lay eggs on the dura mater (the outermost of the three layers of the meninges surrounding the brain and spinal cord) or directly into the bloodstream of the deer (primary host).
- First stage larvae hatch and enter the deer's bloodstream, travel to the lungs, up the trachea (the deer's windpipe) and enter the mouth where they are swallowed.
- The larvae then pass through the alimentary canal and are excreted with the fecal pellets. The first stage larvae are now within the mucoid coating on the outside of the deer's fecal pellets.
- The larvae are picked up by a gastropod, such as a slug or snail, by penetrating the gastropod's foot (part of the snail or slug that is used for locomotion). Once in the gastropod, the nematode larvae mature into an infective second and third stage larvae.
- Deer inadvertently consume the infected gastropod by feeding on plants.
- The larvae penetrate the deer's stomach wall and travels along the nerves of the deer until it reaches the spinal cord and moves into the brain.
- In the brain, the third stage larvae mature into an adult, and the cycle continues without affecting the deer.

Life Cycle Description in a Moose

- The infected gastropod (slug or snail) is inadvertently consumed by a moose.
- The parasitic larvae travel to the spinal cord and brain of the moose, as it does in the deer.

- The nematode disrupts the nervous tissue through mechanical destruction, manipulation, and/or inflammation, resulting in neurologic signs and aberrant behavior of the infected animal. Upon infection, there may be periods where the moose seems to recover as the worm or worms move through different portions of the brain or cord; however, the moose typically shows neurologic signs again after several days. An adult *P.tenuis* within the brain or spinal cord of moose is often fatal. Death can be the result of paralysis, lack of fear/inappropriate behavior (resulting in motor vehicle strike or being shot by police or Environmental Conservation Officer), inability to feed (starvation) or feeding on inappropriate food items (malnutrition).

Signs

Typically there are no signs of infection in the host (white-tailed deer). When *P. tenuis* infects a moose, mule deer, black-tailed deer, elk, caribou/reindeer, llama, alpaca, goat, or sheep the following signs may be exhibited:

A cow moose (adult female) exhibiting neurologic signs of brain worm in Rensselaer County, NY ~Photo by NYSDEC Wildlife Pathology Unit~

- Ataxia (unsteady gait, loss of voluntary muscle control)
- Listlessness
- General weakness
- Fearlessness
- Apparent deafness and/or blindness
- Circling
- Unusual head tilt or neck position
- Inability to feed/forage
- Emaciation
- Paralysis
- Fatality

Diagnosis

Definitive diagnosis relies on the detection of adult *P. tenuis* in the spinal cord or brain during necropsy (animal autopsy).
Presumptive diagnoses are sometimes based on gross or microscopic evidence of worm damage in the spinal cord or brain, or by clinical signs (listed above) when a necropsy is not conducted.

Management Implications

Brain worm is prevalent in New York's white-tailed deer population, yet they apparently suffer little consequence. Brain worm is typically fatal for moose and captive deer/elk, llama, alpaca, goats and sheep. Affected moose cause public concern due to their unusual

behavior and Environmental Conservation Officers, DEC Biologists, or local Law Enforcement are frequently called to the scene to evaluate sick moose. They are usually shot and submitted to DEC's Wildlife Pathology Unit for testing of brain worm, Chronic Wasting Disease, rabies, and cause of death determination.

Over the years several biologists have speculated that New York's large white-tailed deer population with its attendant *P. tenuis* infections would limit the population growth of moose in New York, however, this has yet to be seen. As of 2010, New York moose populations are continuing to rise and are currently estimated at 800 animals. During 2009-2010 the Wildlife Pathology Unit confirmed brain worm infections in six of the 18 moose examined; the infected moose were found in Clinton, Essex, Oneida, Rensselaer, and Saratoga Counties.

Chronic Wasting Disease

Chronic Wasting Disease (CWD) is a rare, fatal, neurological disease found in cervids, members of the deer family. It is a transmissible disease that slowly attacks the brain of infected deer and elk, causing the animals to progressively become emaciated, display abnormal behavior and invariably results in the death of the infected animal. It has been known to occur in wild elk and deer populations in parts of some western states for decades and was recently documented in wild moose as well. The disease has also been confirmed in captive deer and elk herds in several western states and Canadian Provinces. Its discovery in wild deer in south-central Wisconsin in 2002 has generated unprecedented attention from wildlife managers, hunters, and others interested in deer. CWD poses a significant threat to the deer and elk of North America and, if unchecked, could dramatically alter the future management of wild deer and elk.

CWD is one of a group of transmissible spongiform encephalopathies (TSEs) that include scrapie in sheep and goats, transmissible mink encephalopathy of ranched mink, and bovine spongiform encephalopathy (BSE), known as "mad cow disease", in cattle. TSE's are thought to be caused by abnormal, proteinaceous, infectious particles called prions (pree-ons). CWD occurs naturally only in mule deer, white-tailed deer, moose and Rocky Mountain Elk and very likely other subspecies of elk. The mode of transmission of CWD has not yet been fully identified and research is ongoing to explore possibilities of transmission of CWD to other species. However, evidence has shown that the disease can pass from cervid to cervid by direct contact through saliva, urine, and feces, and by indirect contact through environmental contamination with infective substances. There is no known treatment for CWD and it is always fatal. Currently there is no evidence that CWD poses a risk for humans or domestic animals.

The state Departments of Environmental Conservation (DEC), Agriculture and Markets (DAM), and Health (DOH), together with the United States Department of Agriculture's Animal and Plant Health Inspection Service (USDA-APHIS) are cooperating to develop a comprehensive statewide response to the threat of CWD. Together we are actively participating with other agencies and organizations in nationwide efforts to learn more about this disease and to prevent its spread. New York has a vigilant wildlife disease monitoring program in place, comprised of three main components. The first is a regulatory component

designed to reduce the risk of bringing the disease into NY from other parts of the country and minimizing its spread if it is brought here. The second part is an ongoing field surveillance program to ensure the early detection of CWD and the third part is an agency response plan in the event that CWD is found. Through these active surveillance programs the cooperating state agencies first detected and verified positive cases of CWD in two captive deer herds in March 2005. A response plan was then initiated to remove future threats for these captive herds and implement a sampling strategy to determine if CWD had spread into wild New York deer herds.

Regulatory Action - When the potential CWD threat was identified in 2002, New York responded quickly with regulations restricting various activities that could introduce or spread CWD within the state. These regulations allowed DEC time to gather information about CWD, the impacts that various restrictions would have on constituents, and the level of protection they provided. The CWD regulation, which is part of DEC's comprehensive disease management effort, went into effect July 30, 2003. This regulation was later amended on July 14, 2004 to be consistent with the New York State Department of Agriculture and Markets regulations establishing a herd health certification program that covered all captive cervid herds in New York. The DEC CWD regulation includes:

- ***A Restriction on Importation of live Deer, Elk and Moose***
  The regulation prohibits the importation into New York State any wild or captive deer, elk or moose except under permit issued by the New York State Department of Agriculture and Markets.
- ***A Restriction on Importation and Possession of Certain Animal Parts and Carcasses***
  New York is also restricting the importation of deer, elk and moose carcasses and parts from western states and provinces, and from any captive herds, as a further preventative measure to ensure that infectious prions are not brought into the state. The regulation imposes a restriction on the importation and possession of high risks tissues where CWD has been shown to be concentrated including the brain, eyes, spinal cord, lymph nodes, tonsils or spleen.
- ***A Restriction on the Liberation of Wild or Captive Deer, Elk and Moose***
  The regulation imposes a restriction on the liberation of the specified species of deer, elk and moose. An exception is made for wild white-tailed deer temporarily held under department license such as those under the care of a licensed Wildlife Rehabilitator.
- ***Transportation of Deer, Elk and Moose Carcasses and Parts through New York***
  An exemption allows travelers passing through New York to transport carcasses, provided no parts are disposed of or remain in New York State.
- ***Reporting Requirements for Taxidermists that handle Deer, Elk or Moose***
  The regulation requires taxidermists to maintain and keep in their place of taxidermy, a log of all deer, elk, and moose specimens processed in the current year and previous two years.
- ***A Restriction on Deer and Moose Feeding***
  The regulation also prohibits feeding of wild deer and wild moose under most circumstances as a further protective measure. This prohibition includes the use of substances that serve as an edible attractant, such as powdered or crystallized minerals. This regulation does not restrict the planting of food plots for wildlife or cutting browse for deer in the winter.

Field Surveillance - Annually, thousands of wildlife specimens are examined to monitor the presence and distribution of various wildlife diseases. Following the discovery of CWD in Wisconsin, the Department implemented a statewide surveillance program in April 2002 to test wild white-tailed deer for the presence of CWD. This ongoing program uses a statistically valid sampling scheme based on New York's wild white-tailed deer population density to determine the appropriate number of samples needed throughout the State. Samples are collected and sent to an approved USDA laboratory for analysis.

Status of Chronic Wasting Disease in NY

New York State Department of Environmental Conservation (DEC) has received confirmation of Chronic Wasting Disease (CWD) from two wild white-tailed deer sampled in central New York.

The first positive result in a wild deer was announced on April 27, 2005 and came from a yearling white-tailed deer sampled from the Town of Verona, Oneida County. The second positive result is from a three year old doe, located within a mile of the location where the initial positive result was detected. These are the first known occurrences of CWD in wild deer in New York State.

Since CWD testing began in 2002, a total of just over 32,000 deer have been sampled statewide, including 7,300 deer directly from the Oneida/Madison County containment area. Despite intensive testing of wild white-tailed deer, no additional cases have been detected in the New York since 2005.

New York is now entering into the recovery phase, and DEC has taken regulatory action to lift the designation of the containment area. While there is no precedent for eradicating this disease, wildlife and animal health protocols typically scale back response efforts when adequate testing indicates that the threat level has diminished.

DEC will continue annual statewide surveillance sampling and testing of suspected clinical deer. Restrictions will be maintained on importation of risk materials in parts and carcasses from most states and provinces. Restrictions on the feeding of wild deer and moose will remain in effect. DEC will also continue to require all persons holding special licenses to possess captive white-tailed deer to comply with the cervid health program requirements administered by the New York State Department of Agriculture and Markets (DAM).

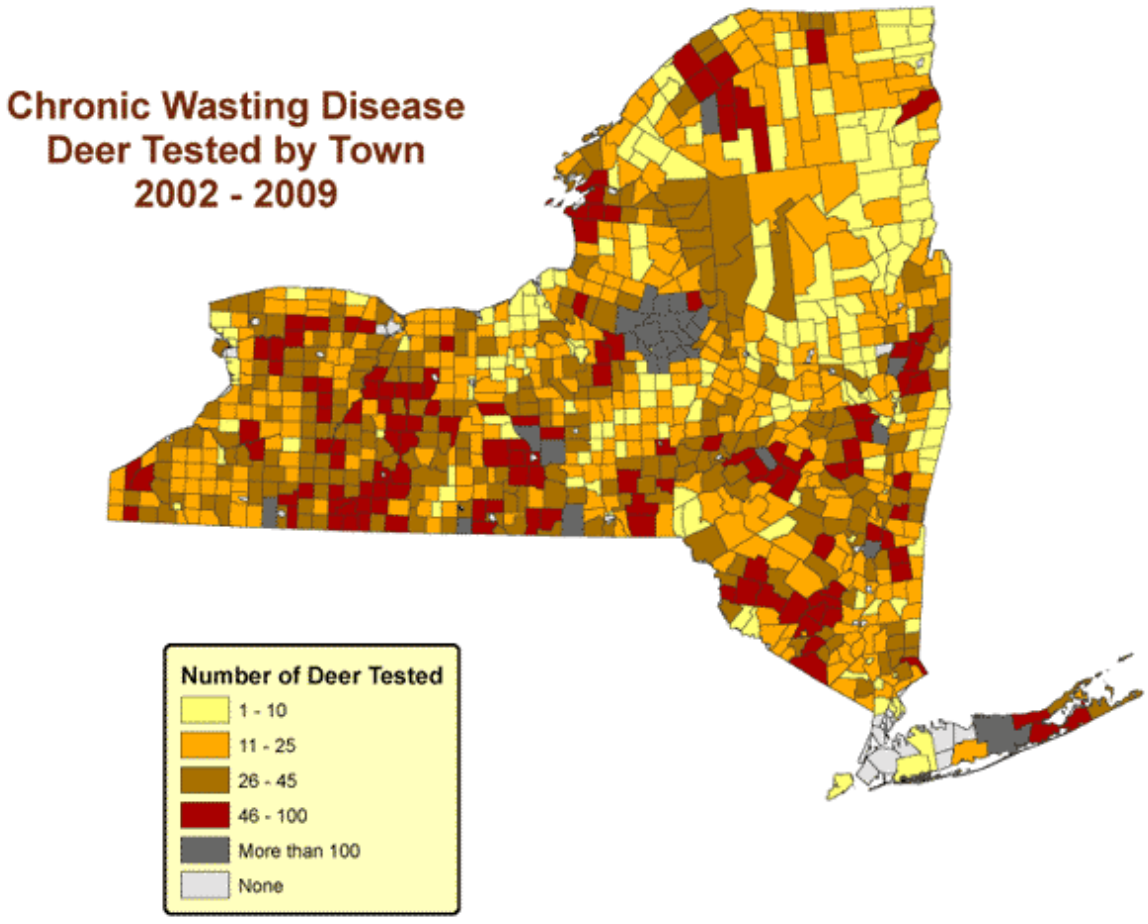
Background on CWD Surveillance in New York

In April of 2002, the DEC initiated a program to collect tissue samples to test for CWD in wild white-tailed deer populations throughout New York State. A sampling scheme was developed to determine the number of deer needed from each county in order to detect if CWD was present in the state. The sampling program required DEC to collect from 800 to 1,000 samples each year with more samples collected from counties with larger deer populations and fewer samples collected from counties with less deer.

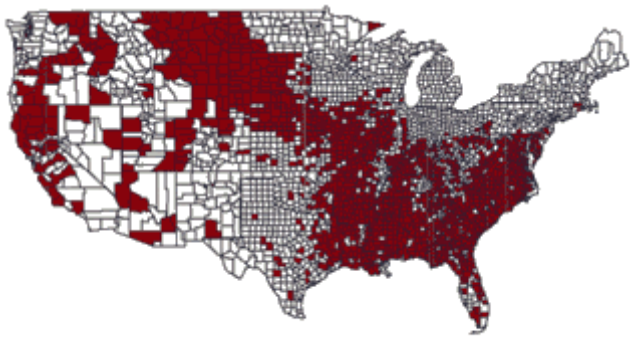
In early April of 2005, through DAM testing of the captive white-tailed deer herds in New York, CWD was detected in five deer from two captive herds in Oneida County. After CWD was discovered in these two captive herds, DEC established a Containment Area around the CWD positive samples and, along with the U.S. Department of Agriculture's Wildlife Services program, implemented an intensive monitoring program on the local wild deer population. This intensive monitoring was completed by the end of April, 2005, and resulted in 290 samples of wild deer from Oneida County, two from neighboring Madison County, and 25 wild deer from the Town of Arietta, Hamilton County (this was a possible transfer site of deer to one of the captive deer facilities). CWD was detected in two wild white-tailed deer collected during this intensive sampling effort.

In order to increase confidence in our understanding of CWD in the Oneida/Madison Containment Area, a targeted sample of deer was collected during February and March of 2009. Information developed through this targeted sampling effort better informed managers on decisions to be made concerning future CWD management and possible Containment Area regulation changes following the 2009 big game season. With still no new cases of CWD detected, the Containment Area was decommission in July of 2010.

The distribution of samples collected since 2002 are depicted on the map below. Each shaded polygon represents a town or city in New York State.



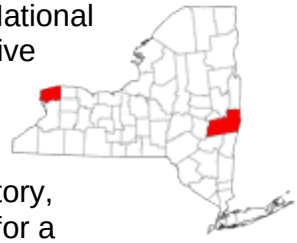
Epizootic Hemorrhagic Disease in New York State



Nationwide distribution of acute or chronic hemorrhagic disease in wild deer between 1980 and 2003.

U.S. Distribution of Epizootic Hemorrhagic Disease

Epizootic Hemorrhagic Disease (EHD) is the most important infectious viral disease among white-tailed deer. The landmark outbreak of the virus was identified and described in New Jersey in 1955. It occurs every year in many southeastern states and has been recently reported throughout the mid-Atlantic. In states where the disease has been detected, it has not had a significant negative impact on the long-term health of the deer herd. It tends to infect only localized pockets of animals within a geographic area.



In October 2007, the first case of EHD in New York was confirmed by the National Veterinary Services Laboratory in Ames, Iowa and the Southeast Cooperative Wildlife Disease Study Group. Several deer carcasses from parts of Albany and Rensselaer Counties were submitted to the DEC Wildlife Pathology Unit for examination and tissue analysis. In November, Niagara County was added to the confirmed list. A combination of case history, characteristic signs and lesions, and the isolation of the virus is necessary for a diagnosis of the disease. Typically, the virus deteriorates in about twenty four hours after death and cannot be spread from deer carcasses. Presently, there are no wildlife management tools or strategies to prevent or control EHD.

The virus is transmitted from animal to animal by bites of infected midges (*Culicoides*), commonly referred to as "no-see-ums" or "gnats". All documented outbreaks of EHD tend to occur during late summer and early fall due to an increase in midge numbers and cease with the onset of a hard frost killing the virus carrying midges and virus. Outbreaks can range from a few mild cases to high deer mortality.

There are several different forms of EHD, but usually in a new outbreak, the very rapid form occurs and kills the animal within one to three days of infection. There are several symptoms of EHD, all of which are not necessarily present in all infected deer. They include: swollen head, neck, tongue or eyelids with a bloody discharge from the nasal cavity; erosion of the dental pad or ulcers on the tongue; and hemorrhaging of the heart, lungs causing respiratory distress. Additionally, the virus creates high feverish conditions leading infected deer to sometimes be found near water sources. For more detailed information about EHD, visit the Southeastern Cooperative Wildlife Disease Study website in the right column of this page.

EHD does not infect humans and people are not at risk by handling infected deer, eating venison from infected deer, or being bitten by infected midges. It rarely causes illness in domestic animals, such as cattle, sheep, goats, horses, dogs and cats. As always, hunters

should observe normal precautions around any sick or strange-acting animals. Wearing gloves when field dressing game and washing their knife is recommended. Hunters should also wash any part of their body exposed to animal tissue, blood or urine. Simply use hot water and soap.

House Finch Conjunctivitis

Cause and Origins

Currently HFC remains largely a disease of House Finches and has been confirmed in most if not all of the species' eastern range. As this eastern population originated from a small release of birds on Long Island in 1940, it has been speculated that lack of genetic diversity may have contributed to the vulnerability of this population to disease challenge. Recently, however, HFC has been detected in House Finches in the Northwest United States and its spread will be closely monitored. HFC poses no threat to human health or non-avian pets.

House finch conjunctivitis (HFC) is an extremely important cause of morbidity in House Finches. It is caused by the bacterium Mycoplasma gallisepticum, well-known for causing chronic respiratory tract disease in domestic poultry and gamebirds. It was not recognized as a potentially important pathogen of wild birds until it began to decimate House Finch populations in the eastern United States in the mid-1990s. HFC has also been confirmed infrequently in other finches and allies, particularly goldfinches.

Symptoms and Diagnosis

The chief clinical signs in House Finches are swollen reddened eyelids usually accompanied by a clear discharge that often mats the feathers around the eye. One or both eyes may be involved. Sick birds may linger around bird feeders for extended periods, not following the comings and goings of other House Finches. Although mortality has been shown to be low in experimental infection of captive birds, mortality of free-living birds is apparently high no doubt in part due to predation and added stress of exposure to challenging weather.

The observed ocular signs are caused by infection of the conjunctiva (the transparent membrane comprising the inner lining of the eyelid and adjoining surfaces of the eyeball) by M. gallisepticum. Infection of the nasal sinuses is also common in House Finches. M. gallisepticum is spread by direct contact with infected birds at feeding stations and roosts. Transmission also probably occurs by contact with contaminated surfaces. Tube-type bird feeders are suspected as likely to facilitate this latter means of infection.

A tentative diagnosis of HFC can be readily made from the gross appearance of the eyes in most cases. Should more definitive confirmation be desired, M. gallisepticum DNA can be detected by polymerase chain reaction testing. Culture attempts, even from fresh specimens, are frequently unsuccessful.

Treatment

Sick birds are easily captured once vision is seriously impaired and can potentially be treated with ocular antibiotic ointments and tetracycline in drinking water. The value of such treatment is controversial as treated birds may still harbor the organism after overt disease is no longer apparent. Treated birds may therefore continue to be a source of infection on release.

The recommended course of action once an outbreak is detected is to suspend bird feeding operations for a minimum of two weeks. All feeders should be cleaned with a 10% solution of household bleach (1 part bleach: 9 parts water). Clean-up of seed hulls and spilled seed under feeders is also recommended.

Salmonellosis

Causes and Susceptible Species

Salmonellosis refers to disease caused by bacteria in the genus *Salmonella*. There are many species and strains of *Salmonella* and a large number of them are at least potentially pathogenic to a variety of vertebrates. In many instances, however, salmonellae may inhabit the alimentary canal without causing overt disease; note, for example, the frequent reports of *Salmonella* in pet turtles. The following discussion will focus on salmonellosis in songbirds caused by *Salmonella enterica serovar typhimurium*, by far the most frequently recognized problem with regard to *Salmonella* and wildlife in New York.

In recent times salmonellosis has generally emerged in mid-to-late winter in flocks of redpolls and pine siskins that have fled boreal forests to the north. During outbreaks, salmonellosis is sometimes confirmed in other species such as goldfinches and evening grosbeaks, but such so-called spillover has to date remained a very minor feature of these episodes. Note: all of the aforementioned species typically feed in long bouts at feeding stations (as opposed to the come and go habits of chickadees and nuthatches).

Transmission

The disease is transmitted through fecal contamination of food. How outbreaks begin is poorly known. Possible mechanisms include the presence of carrier birds in either affected or unaffected species, and the possibility of low levels of contamination in commercially packaged birdseed. In addition to outbreaks in redpolls and siskins, salmonellosis is sporadically confirmed in house sparrows without any noticeable seasonal component. Predators and scavengers of diseased songbirds may be susceptible to infection but there is little evidence of significant morbidity in those species beyond an occasional diagnosis in house cats.

Outbreaks of salmonellosis in redpolls and pine siskins tend to occur in those winters in which there are large winter movements of these species into the northern United States.

Many outbreaks may be recorded over broad geographic regions and the total mortality by winter's end may be large. It seems plausible that any benefit these species gain by access to bird feeders may be cancelled by losses to salmonellosis.

Diagnosis

Diagnosis is tentatively made from the characteristic esophageal lesions in the esophagus. If further confirmation is desired, the organism can be cultured and identified in alimentary canal samples (contents, lesion fragments).

S. enterica sv typhimurium principally infects parts of the alimentary canal. In the avian species mentioned above, the most severe lesions are usually in the esophagus. These lesions are sites of thickening and necrosis that appear as relatively firm yellowish masses that can often be palpated and visualized through the skin. Sick birds may appear weak, and they may tend to sit around feeding stations with fluffed-up plumage. At death most individuals are thin despite evidence of continued feeding.

Preventing the Spread of Salmonellosis

When an outbreak of salmonellosis is detected at a bird feeding station, the traditional recommendation is to halt feeding for a minimum of two weeks. Spilled seed and seed husk debris should be cleaned up, and the feeders disinfected with 10% household chlorine bleach (1 part bleach/ 9 parts water) before redeployment. This strategy will disburse the birds, separating uninfected susceptible birds from sick birds and the contaminated feeder environment.

Trichomoniasis

Causes and Susceptible Species

Trichomoniasis is caused by the flagellate protozoan Trichomonas gallinae. It is the most common disease finding in mourning doves, occasionally causing mortality over sizeable geographic areas. Other dove species are variably susceptible. It is thought that T. gallinae came to North America with Rock (Common) Pigeons accompanying European settlers. Trichomoniasis is occasionally diagnosed in raptors that consume infected doves. Falconers have historically referred to this disease in their birds as frounce.

Symptoms, Diagnosis and Transmission

T. gallinae is a parasite of the upper alimentary canal, most commonly the mouth and upper esophagus. Virulent strains produce cheesy necrotic lesions that may block passage of food and impair breathing. Sick doves appear depressed and oral lesions may show externally as bulges beneath the head.

Trichomoniasis is tentatively diagnosed from the gross lesions. Observation of the live flagellated trichomonad in saliva or lesion scrapings is confirmatory. Fresh unfrozen specimens are required for confirmation in this manner.

Adult doves may transmit the parasite directly to their young via crop milk (doves produce a milk-like substance in their crops which they feed to their young). Transmission via food and water contaminated by sick birds may be important.

Threats to Other Species or Humans

Trichomoniasis is by far the most important disease of mourning doves. Despite some initial concerns, trichomoniasis did not prove to be a significant problem during the reintroduction of Peregrine Falcons to large cities where Rock Doves were the principal prey. T. gallinae poses no threat to human health.

White-nose Syndrome

White-nose Syndrome Threatens New York's Bats

Many thousands of hibernating bats are dying in caves and abandoned mines in New York, Massachusetts and Vermont from unknown causes, prompting an investigation by the New York State Department of Environmental Conservation (DEC), as well as wildlife agencies and researchers around the nation. The most obvious symptom associated with the die-off is a white fungus encircling the noses of some, but not all, of the bats. This has led to the name "white-nose syndrome", which is actually a collection of related symptoms, including a fungus. It is not clear how this fungus alone can cause bats to die, however, impacted bats deplete their fat reserves months before their normal springtime emergence from hibernation, and starve to death as a result.

Bat biologists across the country are evaluating strategies to monitor the presence of the disease and collect specimens for laboratory analysis. Biologists are taking precautions (using sanitary clothing and respirators when entering caves) to avoid unintentionally spreading a disease in the process. Bat populations are particularly vulnerable during hibernation as they congregate in large numbers in caves, in clusters of 300 individuals per square foot in some locations, making them susceptible to disturbance or disease. The vast majority of the hundreds of thousands of bats known to hibernate in New York do so in just five caves and mines. Because bats migrate hundreds of miles to their summer range, the impacts of white-nose syndrome are expected to have significant implications for bats throughout the Northeast.

Indiana bats, a state and federally endangered species, are perhaps the most vulnerable. Half the estimated 52,000 Indiana bats that hibernate in New York are located in one former mine that is now affected with white-nose syndrome. Eastern pipistrelle, northern long-

eared and little brown bats are also dying. Little brown bats, the most common hibernating species in New York, have sustained the largest number of deaths.

DEC has been working with the Vermont Fish and Wildlife Department, the U.S. Fish and Wildlife Service, the Connecticut Department of Environmental Protection, the Northeast Cave Conservancy and the National Speleological Society, along with researchers from universities and other government agencies to study the problem.

Type E Botulism in Lakes Erie and Ontario

What is Type E botulism?

A common bacteria (Clostridium botulinum) produces a toxin under certain conditions, namely the anaerobic (oxygen-free) conditions that occur in dead organisms.

Animals, especially fish-eating birds, ingest the toxin and get sick and die. (In Lake Erie, some types of fish and mudpuppies (a type of salamander) also have died from eating food containing the toxin.)

What are symptoms that an animal might have botulism?

Birds can't hold their head up (thus water birds often drown).

Birds (especially gulls) can often walk, but not fly - they may drag one or both wings (poor posture) while standing.

Fish may flounder or swim erratically near the surface of the water.

Fish usually die quickly and are most likely to be seen washed up on shore.

ANY fish or waterfowl that seem sick should not be harvested or eaten.

Can I get Type E botulism?

You must ingest the toxin, usually by eating an infected fish or animal. Any fish or waterfowl that are sick or act abnormally should not be harvested or eaten because cooking may not destroy the Botulism E toxin.

Can I go swimming in water where wildlife affected by botulism have been found?

You're not at risk for botulism by swimming in Lake Erie or Lake Ontario waters.

If you have concerns about water quality, contact your county health department (phone numbers are listed at back of this sheet) or swim in a regulated beach area. Beaches sometimes close for other reasons (such as fecal contamination).

Can I still take my dog to the beach?

If you bring pets to the shore, keep them away from dead animals on the beach.

Will DEC come and pick up sick or dead animals from my beach?

DEC may collect animals that are still alive, but show symptoms, for testing. Call the DEC's Division of Fish and Wildlife at one of the numbers listed on the back of this fact sheet.

Can I remove dead fish, birds or other animals from my beach?

Wear disposable, rubber or plastic gloves or invert a plastic bag over your hand when handling sick, dead, or dying fish, birds or other animals.

Double bag them and dispose of them with your household trash or bury them away from shoreline areas. Bury them deep enough (two feet or deeper) to discourage other animals from digging them up.

You can bury or dispose of fish, birds or other animals you collect from your property only.

Can I fish and hunt on Lake Erie and Lake Ontario?

You should only harvest fish and waterfowl that act and look healthy.

Don't take any fish or game that show signs of illness at the time of taking.

Follow good sanitary practices when preparing them.

How should I prepare HEALTHY fish or game that I harvest?

Wear rubber or plastic protective gloves while filleting, field dressing, skinning or butchering.

Remove intestines soon after harvest, don't eat intestines and avoid direct contact with intestinal contents.

Hands, utensils and work surfaces should be washed before and after handling any raw food, including fish and game meat.

Fish and game should be kept cool (with ice or refrigerated below 45° F or 7° C) until filleted or butchered and then should be refrigerated or frozen.

Healthy fish and other seafood should be cooked to an internal temperature (in the thickest part) of 140° F (60° C); game birds should be cooked to an internal temperature (in the thickest part) of 165° F (74° C).

The New York State Department of Health issues advisories on eating sportfish and game because some of these foods contain chemicals at levels that may be harmful to your health.

Viral Hemorrhagic Septicemia (VHS) in New York

What is VHS?

Viral hemorrhagic septicemia (VHS) virus is a serious pathogen of fresh and saltwater fish that is causing a disease issue in the Great Lakes region of the United States and Canada. VHS virus is a rhabdovirus (rod shaped virus) that affects fish of all size and age ranges. It does not pose any threat to human health. VHS can cause hemorrhaging of fish tissue, including internal organs, and can cause the death of infected fish. Once a fish is infected with VHS, there is no known cure. Not all infected fish develop the disease, but they can carry and spread the disease to other fish. VHS has been blamed for fish kills in Lake Michigan, Lake Huron, Lake St. Clair (MI), Lake Erie, Lake Ontario, the St. Lawrence River, Skaneateles Lake, Seneca-Cayuga Canal, Conesus Lake, a private pond in Ronsomville and several inland lakes in Wisconsin and Michigan. The World Organization of Animal Health has categorized VHS as a transmissible disease with the potential for profound socio-economic consequences. Because of this, they list VHS as a disease that should be reported to the international community as an exceptional epidemiological (study of diseases in large populations) occurrence.

What is the history of VHS?

VHS was first discovered in the mid 20th Century in Europe where it was originally a significant and costly disease of cultured rainbow trout. Since its initial discovery in Europe, four strains of the VHS virus have been identified, including both freshwater and marine strains. In 1988, VHS was reported in spawning salmon in the Pacific Northwest and was determined to be a new strain of the virus (Type IV) that appears to be a North American strain. It is widespread in the Pacific herring and Pacific cod populations in the Pacific Northwest and has also been found in Atlantic herring and Greenland halibut in the Atlantic Ocean.

In 2005, a very large die-off of freshwater drum in Lake Ontario and a muskellunge kill in Lake St. Clair were linked to VHS, representing the first documentation of the disease in freshwater in the western hemisphere. A subsequent test of an archived muskellunge collected from Lake St. Clair in 2003 tested positive for the virus, indicating that the virus was present, but undetected in the Great Lakes system for at least two years. The drum and muskellunge virus isolates were determined to be different than those from infected fish from other regions and were categorized as a unique strain of the virus (Type IVb).

In 2006, additional fish kills in Lake Huron, Lake Erie, Lake Ontario, the St. Lawrence River and Conesus Lake were linked to VHS. Species involved in fish kills linked to VHS included

muskellunge, smallmouth bass, northern pike, freshwater drum, lake whitefish, gizzard shad, yellow perch, black crappie, bluegill, rock bass, white bass, redhorse sucker, round goby, burbot and walleye. Other freshwater fish species that have tested positive for VHS are chinook salmon, bluntnose minnows and emerald shiners.

In 2007, VHS continued to spread throughout the Great Lakes region. Lake Michigan became the fourth Great Lake with VHS-positive fish. Fish kills from inland waters in New York (Skaneateles Lake, Seneca-Cayuga Canal, and private pond in Ransomville), Wisconsin (Lake Butte des Mortes, Lake Winnebago), and Michigan (Budd Lake) were also found to be linked to VHS. In addition, the virus was detected in several new fish species including lake trout, rainbow trout, and common carp.

In 2008 VHS was found in muskellunge from Clearfork Reservoir in Ohio (Ohio River drainage). In 2009 it was found in bullheads from Baseline Lake in Michigan (Great Lakes drainage) and in ciscoe from the Apostle Islands, WI area of Lake Superior.

What are the clinical signs of VHS?

The clinical signs of VHS may include tissue hemorrhaging (bleeding), unusual behavior, anemia, bulging eyes, bloated abdomens, and the rapid onset of death; however, these symptoms could apply to many different fish diseases. There is no clear visual diagnostic to confirm VHS. Additionally, not all fish infected show any signs and may become carriers of the disease. The only way to confirm VHS is to test the fish in a lab.

How is VHS spread?

VHS can be spread from one waterbody to the next through a variety of means, not all of which are known at this time. One known method of spreading VHS is moving fish from one waterbody to another. This can be done by importation, stocking, or the use of bait fish. Other potential sources of VHS spreading are natural fish movements, recreational boating/angling, bird assistance, ballast water discharge, and sampling activities.

Where is VHS found in New York?

VHS has been found in the following waters in New York:

- Lake Ontario
- St. Lawrence River
- Lake Erie
- Niagara River
- Conesus Lake
- Skaneateles Lake
- Seneca-Cayuga Canal
- Private Pond in Ransomville

What can be done to prevent the spread of VHS?

To reduce the likelihood of spreading VHS in New York State, DEC encourages anglers and boaters to abide by the following guidelines:

- Do not transport fish from one body of water to another! Note that this practice is illegal without a DEC fish stocking permit;
- Only release bait fish into the waterbody it was taken from. Bait purchased commercially should not be released into any body of water;
- Do not dispose of fish carcasses or by-products in any body of water.
- Remove all mud, aquatic plants and animals from all gear, boats, motors and trailers before leaving a body of water;
- Drain your live well, bilge and bait tanks before leaving the water you are fishing or boating on. Anglers or boaters using any waterbody known to be infected with the VHS virus should disinfect their live wells and bait wells with a 10 percent chlorine/water solution. Rinse well to remove all residual chlorine;
- Follow all fish health regulations. Please note that due to the timing of this fish health emergency, the fish health emergency regulations were not in the printed 2006-2008 New York State Freshwater Fishing Regulations Guide; and
- Inform your friends about the fish health regulations. It will take the cooperation of ALL anglers to help prevent the spread of VHS.

USDA-APHIS Federal Order Prohibiting Importation of Certain Species of Live Fish

Due to the potential adverse effects of this disease to fish populations and the desire to prevent or delay its spread to other states, the Animal and Plant Health Inspection Service (APHIS) issued a Federal Order on October 24, 2006, that prohibits the importation of certain species of live fish from Ontario and Quebec and interstate movement of the same species from eight states bordering the Great Lakes, effective immediately. The states included are Illinois, Indiana, Michigan, Minnesota, New York, Ohio, Pennsylvania and Wisconsin.

Fish species included in the federal prohibition are: black crappie, bluegill, bluntnose minnow, brown bullhead, brown trout, burbot, channel catfish, chinook salmon, emerald shiner, freshwater drum, gizzard shad, lake whitefish, largemouth bass, muskellunge, northern pike, pumpkinseed, rainbow trout, rock bass, round goby, silver redhorse, smallmouth bass, trout perch, walleye, white bass, white perch, and yellow perch. Additional fish may be added to the order as they are confirmed to be carriers of this disease. Additional information on the Federal Order can be found on the APHIS website at www.aphis.usda.gov/animal_health/animal_dis_spec/aquaculture/.

What happens to the fish population in a body of water once VHS is present?

The impact of the Type IVb strain of the VHS virus on fish populations is uncertain. It has caused fish mortalities ranging from a few fish to thousands.

What steps are the DEC taking in response to VHS?

The DEC filed fish health regulations on June 6, 2007, in response to VHS. These regulations were filed to halt the spread of VSH and other fish diseased into un-infected waters in New York.

Additionally, the DEC, in cooperation with the College of Veterinary Medicine at Cornell University, is sampling fish from a number of waters across the state, including all waters used as sources of brood stock for DEC hatchery activities, to help determine how far the disease has spread in New York. The DEC is also working with the USFWS fish pathology lab in Lamar, PA and USDA APHIS to conduct surveillance for the presence of VHS in wild fish.

What to do if you find sick/dead fish

If you witness a large number of dead or dying fish (usually 100 or more), please contact the nearest DEC regional office and ask for the Bureau of Fisheries. Questions about VHS and potential DEC actions to prevent its spread can be e-mailed to fwfish@gw.dec.state.ny.us or by calling 518-402-8896.

Whirling Disease

A Resource Stewardship Challenge

Note: The following article first appeared in the June 1996 (Vol. 21, No. 6) issue of *Fisheries* magazine. Author- Philip J. Hulbert.

Whirling disease caused by the myxosporean *Myxobolus cerebralis* has recently emerged as an issue of controversy and concern in portions of North America. This occurred, at least in part, because whirling disease was detected in some prominent wild trout populations in the western United States and was implicated in adverse impacts occurring among those populations. Whirling disease has also been detected in wild trout and hatchery-reared trout in New York since October 1994. The incidence and severity of fish diseases are generally believed to be greater for fish reared in hatcheries than for wild populations (Herman 1970), and whirling disease in particular was regarded as a problem that could be surmounted with careful management of hatchery practices. Has something changed?

Whirling disease was native to salmonids in Europe but was inadvertently introduced to the eastern United States in 1955 via shipments of frozen trout that harbored spores of this myxosporean fish parasite (Markiw 1992). Earthen-bottomed rearing ponds used in many trout hatcheries provided conditions where *Tubifex* worms, the second host of the parasite, could flourish. Fish transfers probably spread whirling disease to many of the states in which it was eventually detected, including New York. Whirling disease was reportedly found in some private fish hatcheries in New York 10-20 years ago, but was not reported from any Department of Environmental Conservation (DEC) fish hatcheries (link below) (John Schachte, New York State Department of Environmental Conservation, personal communication). Comprehensive fish health inspections, including testing for whirling disease at facilities with earthen ponds containing rainbow trout (*Oncorhynchus mykiss*), have been conducted at DEC hatcheries annually since 1976. Despite these efforts, 1994 year-class rainbow trout exhibiting clinical signs of whirling disease, later confirmed with *Myxobolus cerebralis* spores, were detected at DEC's Rome Hatchery on October 19, 1994. This essay summarizes DEC response to this outbreak.

A review of hatchery records and testing for whirling disease indicated our Caledonia Hatchery was the probable source of infected rainbow trout. Wild brook trout (*Salvelinus fontinalis*) in Spring Brook, the water supply for Caledonia Hatchery, tested positive for whirling disease, as did the 1994 year-class of rainbow trout in the hatchery. Other transfers from Caledonia Hatchery occurred earlier in 1994; thus, two more DEC hatcheries, Chateaugay and Salmon River, contained domestic rainbow trout and Skamania strain steelhead, respectively, that tested positive for whirling disease. No fish except the original suspect lot at Rome Hatchery showed clinical signs of whirling disease (whirling, black tail, cranial or skeletal deformity). We had no basis on which to determine how or when trout at Spring Brook and Caledonia Hatchery were first exposed to whirling disease.

Most infected rainbow trout and Skamania steelhead were scheduled to be stocked in spring 1995. Since no therapeutic treatments eliminate this disease from infected fish, continued rearing or stocking carried a risk of introducing the disease into potentially uninfected hatchery lots or other waters, respectively. Thus, we destroyed all 570,000 infected domestic rainbow trout and Skamania steelhead by November 1, 1994. This eliminated approximately 75 percent of the statewide domestic rainbow trout inventory and all production fish of the Skamania strain of steelhead.

We then developed a comprehensive plan to more fully deal with the outbreak. Major elements of the plan were to gather information on risks of whirling disease to wild salmonids; to test all lots of salmonids in DEC hatcheries for whirling disease; to test all lots of salmonids from private and other governmental aquaculture operations in New York; to survey wild salmonids for whirling disease in selected rivers and lakes across the state; to communicate our findings to technical and executive staff within the agency and to interested parties outside the agency; and to develop recommendations for future actions. Our goal was to minimize the effects of whirling disease on important wild trout and salmon stocks and on overall recreational fishing opportunities in New York.

Technical literature focused on the life history and geographic distribution of whirling disease but provided little information about impacts to wild salmonids. We sent questionnaires to states in which whirling disease was reported (Markiw 1992) to learn of

their experiences and whether or not impacts to wild fish had been detected. The resulting input indicated that some states believed whirling disease had harmed wild salmonids but some, such as Pennsylvania, indicated robust wild trout populations were maintained in whirling-disease-positive waters. Others had not investigated this aspect. By fall 1994, some nontechnical periodicals indicated that whirling disease was becoming a controversial issue in Colorado. After initial contacts, DEC staff were invited to meet with fisheries staff of the Colorado Division of Wildlife to review the history of the whirling disease outbreak in that state and their analysis of possible impacts to wild populations in several waters. We concluded that whirling disease had the potential to harm wild salmonids in New York, at lease in combination with other stresses.

Information cited by Markiw (1992) clearly indicated that the disease can be spread by stocking infected fish into suitable uninfected waters. To avoid doing so, DEC adopted an interim policy of not stocking fish from infected lots and began testing all lots at our nine salmonid hatcheries. Lots were either tested one or two times before spring 1995 at a 95 percent detection capability or three to four times before spring at a 99 percent detection capability. The more rigorous testing was required to satisfy concerns stemming from the Great Lakes Fish Disease Control Policy and Model Program, which prohibits stocking any fish from a hatchery where whirling disease has been detected in Great Lakes Basin waters for at least two years after testing negative for *M. cerebralis*. Since major components of DEC's salmonid stocking program for the Lake Erie and Lake Ontario watersheds are reared at the Salmon River Hatchery, the multimillion-dollar recreational fisheries occurring in those waters would have been harmed if uninfected lots of trout and salmon were not stocked. After meeting with members of the Great Lakes Fish Health Committee, we received concurrence to do so.

On March 17, 1995, the DEC implemented testing and adopting a whirling disease inspection requirement for privately stocked fish to help control the disease's spread. The whirling disease inspection requirement, DEC's first fish health inspection requirement ever, was applied to salmonids imported or reared within the state for stocking. Private aquaculturists were generally unhappy with the whirling disease inspection requirement adopted in Mach 1995 believing it was unnecessary and burdensome. However, testing results were negative for 75 percent and positive for 25 percent of the private hatcheries DEC tested in New York. At least five out-of-state facilities were certified to export trout for stocking into New York in 1995.

The DEC tested salmonids in selected lakes and rivers to help define the geographic extent of the whirling disease infestation. Sampling priorities included wild rainbow trout populations, waters that had been stocked with rainbow trout from Caledonia Hatchery, waters near fish culture sites, and waters supporting wild brook trout or brown trout (*Salmo trutta*) populations. Analyses from nearly 100 locations yielded positive findings for fish from nine waters. All positive samples were from waters near a fish culture facility or from waters known to be stocked. We detected whirling disease spores in rainbow, brook and brown trout, but not in lake trout (*Salvelinus namaycush*), Atlantic salmon (*Salmo salar*), coho salmon (*Oncorhynchus kisutch*), chinook salmon (*Oncorhynchus tshawytscha*), or kokanee (*Oncorhynchus nerka*). Overall, the infection appeared to be present in scattered locations across the state but was not widespread. None of the wild fish collected was reported to exhibit any clinical signs of whirling disease.

Since fall 1994, we have communicated our actions and findings within and outside the agency to provide accurate information, minimize hysteria, and promote cooperation. We have sent press releases, conducted numerous interviews with the media, and given presentations to technical and nontechnical audiences. As a result, most public reaction to DEC's action has been favorable and supportive.

Continued monitoring and testing of salmonids and sampling from additional wild populations are under way. The DEC is providing technical assistance to private aquaculturists to help minimize the occurrence and spread of whirling disease. Also, we are working to increase the availability of whirling-disease-testing services in New York and nearby states because private aquaculturists told us they were concerned that testing services were not widely available. We have also launched research into susceptibility of selected strains of wild trout to whirling disease and surveys to establish the presence or absence of population-level impacts in selected waters.

The preceding actions were taken to protect public resources from possible harm. The recent discoveries of whirling disease in several western states and New York have polarized constituent groups and stakeholders. Some predict dire consequences to wild trout populations and fisheries, while others insist whirling disease poses little or no threat to wild fish or even to aquaculture. At lease some information can be found to support either view. Fisheries professionals do not know all they need to about whirling disease. For example, in New York, the apparent absence of whirling disease from most of the waters sampled in 1994 was surprising because infected trout could have been legally stocked for decades. Wild salmonids are common or abundant in most coldwater streams, and *Tubifex* worms should be present near sites of organic material enrichment. Since even resistant species, such as brown trout, can be carriers of the parasite, it is possible that New York waters do not provide conditions that favor either a virulent or widespread expression of whirling disease. We simply do not know. A coordinated approach in investigating whirling disease across the country would shorten the learning curve, thereby benefitting public resource stewards and the private sector.