Novel Virus Killing Common Eiders

The common eider is a large sea duck found along the coasts of Scandinavia, Greenland, Canada, Alaska, and the Northeastern United States as far south as Massachusetts. There are six subspecies of common eider, with four occurring in North America and two in the United States. Common eiders are the largest duck in the northern hemisphere and represent an important species for many hunters and indigenous tribes. This species also fills another, slightly more unique, role as an important source of down feathers. Eiderdown is traditionally collected from the nests of these birds, cleaned, and used as filler for clothing and blankets. The economic, sociologic, and ecologic value of eiders has been a driving force for their careful management since the early twentieth century. Management measures include monitoring for disease and other factors affecting their health.

Between 1998 and 2011, eleven common eider mortality events involving from 30-2,800 individuals were observed along the coast of Massachusetts near Cape Cod. The cause of the mortality was unknown, and the affected eider colonies and the potential population impacts related to these mortalities remain unknown. From 2009-2011, SCWDS received a total of 17 birds from three of these events for diagnostic evaluation. Gross and microscopic findings revealed necrosis of the liver, kidneys, and spleen consistent with multi-systemic disease. In late 2009, SCWDS diagnosticians isolated a previously undescribed orthomyxovirus, which was tentatively named Wellfleet Bay Virus (WFBV), from three of these birds. Working in collaboration with multiple institutions, including the University of Pittsburgh and the University of Texas Medical Branch, efforts are currently underway to further characterize the virus, including comprehensive antigenic characterization and genomic sequencing. Meanwhile, the USGS National Wildlife Health Center (NWHC) has been collaborating with SCWDS and the U.S. Fish and Wildlife Service (USFWS) by providing diagnostic support and conducting experimental inoculation trials in captive eiders in order to further characterize this disease.

Orthomyxoviruses are a group of RNA viruses that affect a wide range of species. Other viruses in this group include influenza viruses, Dhori virus, and Thogoto virus. The exact relationship of this new common eider virus to previously described orthomyxoviruses will not be known until the genetic sequencing is complete. While the genetic data will provide a better understanding of WFBV, much about WFBV remains unknown, including its host range, its temporal and geographic distribution, its epidemiology, and its pathogenesis in common eiders.

To provide answers to some of these important questions, SCWDS has initiated a large collaborative study in cooperation with the USFWS, NWHC, Tufts University, Environment Canada, and other institutions and agencies across North America. In addition to characterizing the virus, we are validating a serologic test for WFBV that will enable us to screen eiders, sea ducks, and other wild avian species for evidence of prior virus exposure. These data will help focus additional research efforts on specific geographic areas and avian species and hopefully will provide insights into the extent of exposure and impacts on common eider populations in the Northeast. (Prepared by Jennifer Ballard and Justin Brown in consultation with Chris Dwyer of USFWS and Andrew Allison of Cornell University)
Rinderpest Eradicated Globally

The Global Rinderpest Eradication Programme (GREP) announced in June 2011 that rinderpest, a devastating animal disease, had been eliminated worldwide, making it the second disease eradicated globally (after smallpox in 1980). This followed the recognition of all 178 member countries of the World Organisation for Animal Health (OIE) as free of rinderpest in May 2011. Rinderpest sporadically decimated cattle populations throughout Europe, Asia, and Africa during the 18th, 19th, and 20th centuries. In 1889, the introduction of rinderpest into Africa killed millions of head of livestock and wildlife triggering widespread famine. Devastated populations of some affected wildlife species took decades to recover. The last known outbreak of rinderpest occurred in 2001 in Kenya.

In 1989, the OIE launched a three-stage “OIE Rinderpest Pathway” for countries to become officially recognized as free from the disease. In 1994, the “OIE Pathway” was implemented in parallel with the GREP managed by the United Nation’s Food and Agriculture Organization (FAO) in collaboration with the UN’s International Atomic Energy Agency. According to the FAO, “Initially, GREP put much effort into determining the geographical distribution and epidemiology of the disease. Later, it promoted actions to contain rinderpest within infected eco-systems and to eliminate reservoirs of infection through epidemiologically and intelligence-based control programmes. Once experts had accumulated evidence that the virus had likely been eliminated, GREP's activities progressively focused on establishing surveillance systems to verify the absence of the disease.” The final remaining reservoirs of rinderpest virus were eliminated through targeted vaccination strategies, serologic surveillance, and the concerted efforts of numerous organizations.

Rinderpest occurred in epidemics with morbidity and mortality rates approaching 100%. Belonging to the Morbillivirus genus within the family Paramyxoviridae, the virus was transmitted via direct and indirect contact between infected and susceptible animals, particularly young animals with waning maternal immunity. The pathogenesis involves a primary focus of infection in lymph nodes draining the upper respiratory tract with subsequent spread throughout the lymph and blood system. Rinderpest virus then would infect epithelial cells, particularly of the gastrointestinal tract. Virus has been isolated from all secretions and excretions including nasal and ocular discharge, saliva, feces, milk, semen, and urine. In addition, virus may spread indirectly via contamination of the environment and aerosolization of particles allowing spread over distances up to 100 meters. Rinderpest viral particles were readily inactivated in the environment by drying for six hours in the sunlight, or by common disinfectants, burning, and autolysis of the animal’s carcass.

In the field, all species in the order Artiodactyla are susceptible to rinderpest with the highest mortality rates in domestic cattle, water buffalo, yak, wild bush pigs, warthog, African buffalo, giraffe, eland, kudu, wildebeest, and antelope. Although North America never experienced the devastation caused by rinderpest, potential introduction of this disease was of great concern because of the potential impacts on our livestock and native, cloven-hoofed wildlife.

The susceptibility of white-tailed deer was demonstrated through experimental inoculation with virulent rinderpest virus at the USDA’s Plum Island Animal Disease Center in the 1970s. Within two days, inoculated deer developed clinical signs typical of natural infection including conjunctivitis, nasal discharge, bloody diarrhea, and death within 5-6 days. It is clear that an epidemic of rinderpest in the United States could have caused significant mortality in our wildlife and livestock populations, and the eradication of this devastating disease is good news for all of us. (Prepared by Danielle Yugo of the University of Tennessee and John Fischer)

Hemorrhagic Disease in 2011---So Far

As part of our mission to support diagnostics related to epizootic hemorrhagic disease viruses (EHDV) and bluetongue viruses (BTV) in the United States, we processed more than 100 samples during the fall of 2011. To date, 36 viruses have been isolated and with one exception (a BTV-11 isolated in North Carolina) all were identified as EHDV-2. Positive deer
were detected in: Alabama, Florida, Illinois, Kansas, Kentucky, Louisiana, Maryland, Missouri, Montana, New Jersey, North Carolina, Pennsylvania, and Virginia. At present, we have not identified any exotic EHDV or BTV in our 2011 samples but testing is not completed.

A striking aspect of this year’s surveillance was the wide geographic range from which EHDV-2 viruses were isolated. In most years, outbreaks are regional and disconnected across the United States. An example of this occurred in 2007 where a widespread EHDV-2 outbreak occurred in the eastern United States while BTV-17 predominated in the western states. From what we have observed in our isolation attempts or have heard related to preliminary reports of mortality this year, there appears to be significant EHDV activity in Montana, Wyoming, North Dakota, and South Dakota. In North Dakota, mortality was extensive enough that the Department of Game and Fish suspended sales of hunting licenses and offered refunds for licenses purchased by hunters in deer management units in the western portion of the state. Eastern Kansas also was hard hit, as were eastern Pennsylvania and New Jersey.

Based on our isolation results, we do know that EHDV-2 was associated with all of these outbreaks but we do not know if these outbreaks were connected. This part of the puzzle will be filled in with the hemorrhagic disease (HD) morbidity and mortality data annually provided to SCWDS by state wildlife agencies throughout the country. This annual HD survey has been in place for more than 30 years and with the detection of exotic viruses, more numerous reports of EHDV activity in northern states, and outbreaks as extensive as seen in 2007, the value of this long-term data set has increased. Providing quality data to understand HD outbreaks is a two-step process, both of which are dependent on continued support from the wildlife community. With 2011, thanks to the samples that you submitted, we have the viruses in hand to define and characterize the agent(s) responsible for recent HD problems. The quality morbidity and mortality data that you will supply in the 2011 annual survey will provide the perspective we need to better define this year’s outbreaks and to better understand potential impacts to our wildlife resources.

In an attempt to put these data and isolates to further use, a collaborative project to map the distribution of EHDV and BTV subtypes within the United States and to determine if these distributions have changed over time was initiated with the Centers for Epidemiology and Animal Health (CEAH), APHIS, USDA. Supporting data for more than 750 EHDV and BTV isolates made by SCWDS from 1992 through 2010 were submitted to CEAH to include in these analyses. The 2011 data will be submitted upon completion of annual testing in December. (Prepared by Dave Stallknecht)

A Case of Inhalation Anthrax

The Minnesota Department of Health reported a case of inhalation anthrax in a 61-year-old man in early August 2011. The Florida native was hospitalized in Minnesota with symptoms of pneumonia after three weeks of traveling through Montana, North Dakota, South Dakota, and Wyoming. Doctors concerned by the appearance of his chest radiographs ordered further testing that ultimately confirmed anthrax. Public health officials have been unable to identify the source of anthrax in this case despite extensive investigations that included review of all potential anthrax cases investigated by state public health, agriculture, and wildlife agencies. There was no evidence to suggest the case resulted from a criminal or terrorist act, and it is presumed to be due to exposure to naturally occurring anthrax spores in the environment. During his travels, the patient had exposure to both soil and animal remains in areas where anthrax cases have occurred in the past. Fortunately, the man was discharged from the hospital after three weeks of intensive therapy and is expected to make a full recovery.

Anthrax is caused by the spore-forming bacterium *Bacillus anthracis*. It became well known after the bioterroristic attacks in 2001; however, anthrax is naturally present nearly worldwide and has been for centuries. Wild and domestic herbivores are highly susceptible to the disease and become infected by grazing in areas contaminated with spores. The spores germinate into a vegetative form in the low oxygen environment inside the animal and cause disease. When the animal dies and the
carcass is opened by scavengers, spores form again. Carnivores are less susceptible to anthrax, but can also contract disease from feeding on infected carcasses. Humans typically are infected by contact with an infected animal or contaminated animal product.

Anthrax spores can survive for decades in areas with calcium-rich, alkaline soil. In these regions, sporadic outbreaks of anthrax occur, often during hot, dry summers that follow a wet spring. Typically, an outbreak consists of just one or two mortalities, but occasional epidemics develop, killing hundreds of animals. Mosquitoes and biting flies may play a role in these epidemics by mechanically spreading disease. In the United States, most wildlife and livestock outbreaks have been in states west of the Mississippi River including Montana, Wyoming, North Dakota, South Dakota, Minnesota, and Texas.

The clinical course of disease in most herbivores is a rapid septicemia, and animals are found dead without showing clinical signs. They may have bloody discharge coming from multiple orifices, bloating, incomplete rigor mortis and their blood may not clot. Occasionally, clinical signs such as depression and difficulty walking or running may be observed. Carnivores and pigs may show clinical signs such as swelling of the face, neck or ventral body before they either recover or die.

Early recognition of anthrax is essential to reduce risk of human exposure. A necropsy should NEVER be performed on any animal that is suspected to have anthrax. Opening the carcass of an infected animal facilitates oxygen exposure that can result in release of infective spores in the environmental and potential exposure of the individual examining the carcass. Suspect cases should be reported immediately to the state veterinarian for proper carcass sampling and disposal.

Human cases of anthrax occur in three main forms: cutaneous, gastrointestinal, and inhalation. Cutaneous anthrax is most common, representing approximately 95% of human anthrax cases worldwide and results from anthrax spores entering a wound or abrasion. Initially, a small, raised sore forms; this becomes a blister and later a necrotic ulcer with a black center. Cutaneous anthrax is rarely fatal with appropriate treatment. Gastrointestinal anthrax is less common and often results from ingestion of undercooked, contaminated meat and typically results in non-specific symptoms such as nausea, anorexia, vomiting, diarrhea, and fever. Without treatment the infection can lead to fatal septicemia with case fatality rates from 25% to 60%. Although uncommon, inhalation anthrax is the most severe form, but only 82 cases were reported in the U.S. between 1900 and 2005. Symptoms also are non-specific and include fatigue, fever, and cough that progress to dyspnea and eventually to respiratory arrest. Historically, case fatality in inhalational anthrax was 92%. In the 2001 terrorist attacks, mortality was only 45% due to aggressive treatment and early case recognition.

Anthrax was a significant cause of mortality in wildlife and domestic species prior to development of a livestock vaccine in 1937. Since then, national control programs and livestock vaccinations have drastically reduced the number of anthrax cases. As a result, many veterinarians, biologists, and livestock producers lack familiarity with anthrax and outbreaks are not always recognized. Anthrax epidemics still occur in wildlife: white-tailed deer comprised 1216 of 1637 mortalities in a 2001 event in Texas. Wild animals pose a particular problem because vaccination is not feasible in free-ranging populations and infected carcasses are not easily found. As a result, anthrax cases in wildlife likely are under recognized and reported.

This case highlights the importance of early recognition and treatment of anthrax. Initial symptoms of anthrax in humans can be non-specific and difficult to recognize. Wildlife professionals are at increased risk for zoonoses and should inform their physicians of any potential exposure. Furthermore, wildlife professionals should remain aware of how to recognize anthrax in wildlife populations. Early recognition and reporting of wildlife cases may help prevent human exposure and reduce animal mortality. (Prepared by Mary Wood of the University of Minnesota)
New Jersey Joins SCWDS

We are quite pleased to announce that the New Jersey Division of Fish and Wildlife (NJDFW) is the newest member of the Southeastern Cooperative Wildlife Disease Study. The NJDFW joined SCWDS on July 1, 2011, bringing the total number of member states to 19. Doug Roscoe, a long-time friend of SCWDS who handled wildlife disease issues for New Jersey, retired earlier this year, and the NJDFW elected to join SCWDS in order to help fill the large void that Doug left behind. We are proud of the confidence they have shown in SCWDS and look forward to assisting their biologists, managers, and administrators with the management of healthy wildlife populations. And, we wish Doug all the best in retirement.

Antleromas in a Deer

Most deer hunters have a fascination with antlers and this is even truer when deer have exceptional or just plain weird head gear. The cause of antler malformations often is unknown, and this may be part of the mystique of nontypical or abnormal antlers. Certainly few cases are stranger than the deer that was submitted by the Florida Fish and Wildlife Conservation Commission (FFWCC) to SCWDS this past June.

On June 17, 2011, a north Florida resident found a dead white-tailed deer. The landowner realized that this was not a normal deer. Where it should have had normal antlers, it had massive growths covered in what appeared to be antler velvet (Figure 1). In addition, the deer was horribly emaciated and its face was grotesquely misshapen.

The FFWCC personnel took an interest in the case and submitted the carcass to SCWDS for examination. Instead of forming a typical branching pattern, both antlers of this deer formed irregular dome-shaped masses that covered the top of the head. The left side of the face was swollen and distorted (Figure 1, arrow). The antlers were hard but were completely covered in velvet, suggestive of antleromas.

We cut a cross section of the skull to check for tumor invasion of internal structures of the head. The masses originating from both antlers directly invaded adjacent tissues. The nasal cavities contained multinodular masses of bone. The bony tumors in the left nasal cavity were so large that the adjacent cheek bones had been remodeled resulting in the externally visible protrusions of the face. The tumor invaded the cranial vault but did not directly invade the brain. Although it compressed the brain at multiple sites, we could not determine whether this affected brain function.

The invasive portions of the tumor resulted in necrosis of the tissues of the left eye; externally the eye appeared dry and opaque. There also was extensive damage to the muscles associated with chewing.

Cases of massive uncontrolled proliferation of antlers similar to those described here are occasionally reported in castrated male deer. In...
red deer, the growths have been termed perukes, but they have also been called antleromas.

Antleromas are considered benign, but they can become massive. They typically cause problems only if they become infected or are so large that they interfere with vision or chewing. The current case differs from previous descriptions of antleromas by the tremendous local invasion and destruction of other tissues of the head. Although there was no tumor metastasis to other sites, the extensive local invasion and tissue damage caused the death of this deer.

In male white-tailed deer, testosterone levels normally rise in the autumn, resulting in antler hardening and shedding of the antler velvet. After the mating season, decreasing testosterone levels cause bone resorption at the pedicle and shedding of the antlers.

Several studies have suggested a link between abnormal testosterone levels and antler growth abnormalities. Reduced testicular development, castration, and the presence of estrogenic compounds have been cited as causes for low testosterone levels leading to retained velvet or abnormal antler growth. In this deer the testicles were of normal size with no obvious lesions. However, they were inactive due to the time of year and we cannot be certain whether hormonal abnormalities were involved in the development of these antleromas. (Prepared by Kevin Keel)

**My Own Backyard**

September 24th was a beautiful Saturday in Athens, Georgia, and I put the afternoon to good use by blowing leaves. Heavy business travel didn’t leave much time to stay caught up, and there were lots of leaves on the ground due to our worsening drought. Following my usual pattern, I blew the leaves around the front of the house, then down back along the path through the woods and across the footbridge over the creek to the hammock. After blowing the leaves off the hammock, I realized that it needed testing so I turned off the leaf blower and climbed into the hammock to listen to the woods rather than the 2-cycle engine.

It was only two or three minutes before I heard an unusual grunt followed by even more unusual splashing in the creek. It’s a remote location; the creek is only a few feet wide, and with the exception of the three river otters that I saw only once a few years ago, I couldn’t think of any animals that might be splashing water 6-8 feet into the air. I was going to have to get out of the hammock and have a look.

I could not have been more surprised by what I saw: a mature, 9-point white-tailed buck on its side in the water. The animal was in excellent nutritional condition but unaware of my presence. It was trembling and unable to rise or even lift its head out of the mud. I euthanized the deer and playing the percentages, I began to run through the potential causes of its moribund condition: Archery season was open, but there were no external wounds. This left two more possibilities high on my list.

It was early fall, we’d been experiencing high temperatures and drought, and the buck was not just near water, it was in the water! These factors all were suggestive of hemorrhagic disease (HD), and SCWDS had been confirming outbreaks around the country, although nothing in the Athens area. However, a field necropsy failed to reveal any characteristic HD lesions.

The last likely cause required a trip to SCWDS to confirm. I removed the head and took it to the lab, along with the samples collected during the necropsy. Opening the skull confirmed my last suspicion: a large brain abscess. Bacterial culture yielded *Arcanobacterium pyogenes*, the organism most commonly associated with brain abscesses in deer. The brain also was tested for rabies because of the severe neurological signs; results were negative.

Brain abscesses occur most frequently in bucks, in which they typically are associated with the antler pedicles and occur when bacteria enter via skin damaged by antler rubbing, fighting, or other trauma. Following development of a subcutaneous infection, the bacteria may enter the cranial vault through the natural seams (sutures) holding the skull bones together. Once it has penetrated the cranium, the infection may
develop into severe, suppurative meningitis, or into one or more abscesses in the brain ultimately causing fatal central nervous system disease.

Brain abscesses are a growing concern for biologists and landowners practicing quality deer management. This approach typically involves the protection of young bucks (yearlings and some 2.5 year-olds) and other practices to maintain a healthy population in balance with the habitat. With a predilection for mature bucks, brain abscesses could significantly impact populations with a larger number or proportion of older bucks.

Several studies have been conducted to better understand this disease. A review of brain abscess/suppurative meningitis cases in SCWDS diagnostic records from 1971-1989 revealed they represented 4% of diagnoses in 683 whitetails. There was strong gender bias: 88% of the cases occurred in males and were more prevalent in older bucks. Cases occurred only from October to April. Necrosis, erosion, and pitting of skull bones were common, and these lesions are useful in determining cause of death in skeletal remains.

In 1996-97, SCWDS surveyed diagnostic laboratories and examined skulls from natural mortalities and hunter-harvested deer in North America. Diagnostic laboratories reported that intracranial abscesses were found in 2.2% of nearly 4,500 white-tailed deer from 12 states and four Canadian provinces. Approximately 87% of cases occurred in males, and the overall prevalence among males was 4.9%. *Arcanobacterium pyogenes* was isolated from 61% of cases; 18 other genera of bacteria also were isolated.

Nine per cent of 119 skulls from deer found dead in the southeastern USA had characteristic bone lesions, whereas skulls from hunter-harvested bucks in the region had a lesion prevalence of 1.4%. The similarity of disease prevalence among bucks found dead in the field (9.0%) and deer examined at southeastern diagnostic laboratories (8.4%) suggests that this disease kills nearly 10% of yearling and adult bucks in the southeastern region. Researchers at the University of Georgia’s Warnell School of Forestry and Natural Resources, in collaboration with SCWDS personnel, currently are investigating the prevalence of brain and subcutaneous cranial abscesses in deer throughout Georgia.

There are a few things to be learned from this personal experience. The first is that like everything else, wildlife diseases do not observe office hours. The second is that it takes a thorough diagnostic examination to determine the causes of morbidity and mortality in wildlife, no matter how much you might think you know. And the third is that you need to turn off the leaf blower and get in the hammock every once in a while to really know what’s going on in your own backyard. (Prepared by John Fischer)

**Our New Look**

Every so often it is time to make some changes in The SCWDS BRIEFS, our quarterly newsletter. We have made some formatting changes in the past, but one thing we never changed was the green paper used for the printing stock. Many people did not know the name of the newsletter, but immediately recognized it by the color of the paper. So, it is not without hesitation that we change the look of the BRIEFS by printing it on white paper. We like to include photos, maps, and illustrations in the newsletter when we can and believe they will look better in color on white paper. The one thing that will never change is the content of the SCWDS BRIEFS: informative, up-to-date, factual stories on current health issues involving wildlife.
Information presented in this newsletter is not intended for citation as scientific literature. Please contact the Southeastern Cooperative Wildlife Disease Study if citable information is needed.

Information on SCWDS and recent back issues of the SCWDS BRIEFS can be accessed on the internet at www.scwds.org. If you prefer to read the BRIEFS online, just send an email to Jeanenne Brewton (brewton@uga.edu) or Michael Yabsley (myabsley@uga.edu) and you will be informed each quarter when the latest issue is available.