White Nose Syndrome News

Conservation biologists and wildlife health professionals around the country have been anxiously awaiting news indicating further geographic expansion of white nose syndrome (WNS), and on February 16, 2010, it was announced that WNS was confirmed in Tennessee for the first time in tri-colored bats (*Perimyotis subflavus*) from Sullivan County. First recognized in a New York cave in 2007, WNS was reported at more than 65 sites in 9 states through the winter of 2008-09. To assist in detecting newly affected sites, SCWDS has joined the U.S. Geological Survey's National Wildlife Health Center (NWHC) in providing diagnostic support for ongoing surveillance for *Geomyces destructans*, the fungus believed to be the primary pathogen of WNS.

We currently are processing samples by histopathology, fungal culture, and polymerase chain reaction (PCR). However, as accessions increase in number and as the test is validated on a larger number of samples, we intend to rely more on PCR as a screening assay. Submissions are encouraged from SCWDS member states and federal natural resources agencies. If there is clinical evidence of WNS in a hibernating colony, bat biologists are encouraged to submit 5-10 freshly dead bats. Please contact SCWDS personnel prior to submitting the samples and fill out the WNS Surveillance Form that we provide. Strict biosecurity measures must be observed to avoid spreading the disease. For unexplained mortality of bats, please submit fresh chilled carcasses accompanied by our diagnostic accession form available on our website http://www.uga.edu/scwds/diagnostic.htm.

Additional information and submission guidelines also are available on the NWHC website at http://www.nwhc.usgs.gov.

A wildlife health bulletin released on December 11, 2009, (http://www.nwhc.usgs.gov/publications/wildlife_health_bulletins/WHB_2009-03_WNS.pdf) succinctly describes the status of NWHC research projects. Preliminary findings of one research project indicate genetic material specific for *G. destructans* is present in sediments of WNS-affected caves. This suggests viable fungus could be present in the sediments and could be transported by humans visiting the caves. Preliminary results from other studies indicate that bat-to-bat transmission of the fungus can occur in a controlled environment. The group also has developed a PCR assay that can be used as a screening test for surveillance samples. Websites for additional information and for sample collection protocols also are available in the bulletin.

It still is uncertain why WNS became a problem in North American bat colonies, but some suspect that it could have been introduced from Europe. A recent report in *Emerging Infectious Diseases* (16:290-293) described the first confirmation of *G. destructans* infection in a greater mouse-eared bat (*Myotis myotis)* from France. Researchers previously had suspected that this fungus was present in European bats, but this was the first time it was demonstrated by culture and molecular techniques as the same fungal species affecting bats in the United States. The bat was not underweight, had no signs of clinical disease, and it was released after swabs were made of the visible fungal growth on its nose. This finding does not indicate that Europe is the source of the fungus that affects North American bats, but it does confirm its presence on both continents.

Currently, there have been no reports of WNS-associated mortality in Europe, nor in any countries other than the United States.

White nose syndrome has caused catastrophic declines in bat populations in affected caves in the northeastern United States, with caves longest affected exhibiting declines approaching 100% in the numbers of bats roosting there. The recent detection of WNS in Tennessee, the past history of its dramatic geographic spread, and the proximity of affected sites to state borders suggest that WNS may appear in additional states. More WNS information, including biosecurity protocols, is available from the Northeastern Region of the U.S. Fish and Wildlife Service (http://www.fws.gov/northeast/white_nose.html). (Prepared by Kevin Keel)

CWD Found in Virginia

The Virginia Department of Game and Inland Fisheries (VDGIF) announced on January 20, 2010, that chronic wasting disease (CWD) had been confirmed in a 2-year-old, wild white-tailed deer killed by a hunter in Frederick County. Frederick County is adjacent to Hampshire County, West Virginia, where CWD has been detected in 62 free-ranging deer since 2005, and the positive Virginia animal was taken within a mile of the state line.

Officials with VDGIF have been paying close attention to CWD since 2002, when the disease was found for the first time east of the Mississippi River, and they have tested nearly 5,000 deer statewide. The agency developed a CWD Response Plan in 2002 and activated it in 2005, following detection of CWD in West Virginia. The response plan, which has been revised several times (as recently as 2009), was designed to delineate the prevalence and distribution of CWD and to control its transmission. With the 2005 discovery of CWD in adjacent West Virginia, the VDGIF designated an Active Surveillance Area in western Frederick and Shenandoah counties, where samples from hunter-killed and road-killed deer are collected and tested for CWD. In addition, wildlife officials in Virginia continue to share information and coordinate CWD responses with the West Virginia Division of Natural Resources and consult regularly with the U.S. Department of

Agriculture and the Virginia Department of Agricultural and Consumer Services.

Since 2002, the VDGIF has taken several other steps to proactively reduce the risk of CWD in the state:

- Conducted active CWD surveillance statewide that is concentrated where risk factors exist.
- Banned movement of privately owned live deer and elk into and within the state.
- Strengthened captive cervid operation requirements regarding animal identification, record-keeping, facility inspections, and mortality reporting.
- Banned importation of whole cervid carcasses and certain tissues from states known to have CWD.
- Prohibited feeding wild deer from September 1 through the first weekend in January annually.
- Prohibited release of deer rehabilitated in Frederick or Shenandoah counties outside of either county.
- Provided accurate and timely CWD information to hunters and the general public.

Virginia is the 12th U.S. state in which CWD has been found in wild cervids and is the only new state to detect the disease since 2005, when it was found in New York and West Virginia. Although West Virginia continues to confirm CWD in wild deer (16 additional positive animals recently were announced), testing of more than 1,500 wild deer in New York's CWD Containment Area has failed to identify any affected animals since the first two were found in April 2005. (Prepared by John Fischer with information from the VDGIF website, www.dgif.virginia.gov).

Serosurveys of Feral Swine

Pseudorabies and swine brucellosis have been detected in feral swine in more than 10 states, and their presence in the feral reservoir threatens the health of domestic swine. The role feral swine may play in the epidemiology of other domestic swine disease agents, namely swine influenza virus (SIV), porcine circovirus-2 (PCV-2), and porcine respiratory and reproductive syndrome virus (PRRSV) is largely

unknown. Feral swine also could be important in the spread or maintenance of an introduced foreign animal disease, such as classical swine fever or foot-and-mouth disease.

Data often are lacking on the prevalence of swine disease agents in feral populations. Antibodies against SIV have been reported in feral swine in California, Kansas, Oklahoma, Mississippi, and Texas and in European wild boar in Spain. Porcine circovirus-2 was isolated from Eurasian wild boar raised on pasture in western Canada. Antibodies against PRRSV were found in feral swine in Oklahoma and in European wild boar in Germany and France, and positive PCR results for PRRSV were reported from a road-killed wild boar in Italy.

SCWDS recently published results of a study funded by USDA-APHIS-Veterinary Services that compared antibody prevalence in feral swine populations associated with backyard or "transitional" domestic swine operations in South Carolina to those in feral populations associated with intensive commercial swine production facilities in North Carolina (*Journal of Wildlife Diseases* 45:713-721). The study areas were identified using maps depicting the distribution of feral swine, backyard swine premises in South Carolina, and commercial swine production premises in North Carolina. In the feral swine populations associated with backyard swine premises in South Carolina, 10 of 50 (20.0%) feral swine were seropositive for pseudorabies virus (PRV), 7 of 50 (14.0%) were seropositive for *Brucella suis,* 29 of 49 (59.2%) were seropositive for PCV-2, 0 of 49 were seropositive for PRRSV, and 0 of 49 were seropositive for any of the SIV subtypes.

In feral swine populations associated with intensive commercial swine production facilities in North Carolina, 0 of 120 feral swine were seropositive for PRV, 0 of 120 were seropositive for *B. suis*, 1 of 120 (0.8%) was seropositive for PRRSV, 86 of 120 (71.7%) (80 positives plus six suspects) were seropositive for PCV-2, and 108 of 119 (90.7%) were seropositive for at least one SIV subtype.

The presence of PRV and *B. suis* in the selected feral swine populations in South Carolina may have been due to the previous introduction of infected feral swine into the area or to the association of feral swine in these areas with infected backyard swine at some time in the past. The absence of PRV and *B. suis* in feral swine in the North Carolina populations may have been due to the absence of these disease agents in feral swine originally introduced into the area, or the lack of potential for contact with infected commercial swine. In contrast, feral swine associated with commercial swine in North Carolina may have been exposed to SIV subtypes circulating in commercial swine via airborne spread of SIV from high-density commercial swine facilities.

Feral swine seropositive for PCV-2 were prevalent in both states, which may indicate efficient transmission from commercial swine and backyard swine, or that PCV-2 is widespread in feral swine. The low prevalence of animals with antibodies against PRRS may indicate a less than efficient means of transmission from commercial to feral swine.

Additional epidemiological studies are needed to better understand disease transmission risks between domestic and feral swine, the role of feral swine as reservoirs and disseminators of these diseases, and the mechanisms by which disease agents are transmitted between domestic and feral swine. Such data are important for developing disease control measures in domestic swine and will be invaluable in the event of a foreign animal disease introduction into domestic or feral swine. (Prepared by Joseph Corn)

AI Serology in Wild Birds

Surveillance for avian influenza (AI) viruses in wild birds traditionally has relied almost exclusively on virus isolation and/or reverse transcriptase polymerase chain reaction (RT-PCR) to detect AI virus in cloacal swabs collected from individual birds or, less commonly, from wild bird fecal samples collected from the environment. These diagnostic approaches have provided our existing knowledge of AI epidemiology and host range in wild birds, but virus isolation and PCR have certain limitations. Virus isolation is expensive, time consuming, and requires appropriate biosafety precautions to safely work with infectious virus. The use of RT-PCR

requires specialized equipment and expertise that may limit its use in some laboratories.

In regard to wild bird surveillance for AI, another potential limitation of virus isolation and RT-PCR is that both approaches are dependent on the host shedding virus when sampled. This limitation is not problematic when sampling avian populations in which the epidemiology is defined, such as shorebirds or ducks. For these wild birds, long-term data sets indicate when, where, and how best to sample the migratory populations in order to efficiently detect infected birds. For example, based on historic isolation rates reported in the literature, the best known opportunity to isolate AI virus from shorebirds is from ruddy turnstones (*Arenaria interpres*) at Delaware Bay in May and June during their annual migratory stop-over. Similarly, the best opportunity to isolate AI virus from ducks is from juvenile dabbling ducks (*Anas spp.*) during the late summer to early fall at pre-migration staging areas. The dependence of virus isolation and RT-PCR on viral shedding particularly becomes a problem when sampling a species, geographic location, or time period without previous data to guide the surveillance effort.

Serologic testing for antibodies to AI virus commonly is used in domestic poultry to screen for previous exposure to AI on a population level. The benefit of serologic testing is that antibodies directed against AI virus persist longer than viral shedding, increasing the duration and overall likelihood of detecting evidence of previous infection in an avian population. Testing for antibodies to AI would seem quite useful for wild bird surveillance as a compliment to virus isolation or RT-PCR; however, serologic testing traditionally has been underutilized. The primary reason for the underutilization is that most assays used for AI surveillance in domestic poultry either perform poorly in certain important avian groups or are not efficient screening tools when applied to wild bird surveillance.

The agar-gel immunodiffusion (AGID) test is a commonly used serologic assay in domestic poultry that detects antibodies to internal proteins of all AI viruses, regardless of hemagglutinin (HA) or neuraminidase (NA) subtype. It is a simple test to perform, requires minimal equipment, and has good specificity.

The major limitation of the AGID test for AI surveillance among wild birds is that the assay has poor diagnostic sensitivity in ducks, presumably due to the overall poor antibody response and lack of precipitating antibodies produced by duck species. The hemagglutinin inhibition (HI) and neuraminidase inhibition (NI) tests are serologic assays also frequently used in poultry. These tests detect neutralizing antibodies directed against the HA or NA surface glycoproteins of AI. The major limitation of the HI and NI tests for wild bird surveillance is that the assays are subtype-specific and, consequently, are not efficient screening tools due to the large number of tests that would have to be run on each sample.

There are multiple commercial enzyme-linked immunosorbent assays (ELISA) available as serologic tests for poultry that detect antibodies to internal proteins of AI virus regardless of subtype, similar to the AGID test. These ELISA kits are available as indirect or blocking formats. The indirect ELISAs are specific to galliforms and, therefore, have minimal use in wild birds. Multiple companies recently have developed commercially available epitope blocking ELISA (bELISA) kits. Based on the mechanism of these assays, they have the potential to perform well across a wide-diversity of avian species.

As part of a larger research program funded by the National Institutes of Health, SCWDS has led a collaborative research project to evaluate the ability of the IDEXX bELISA (Flockchek AI MultiS-Screen Ab ELISA, IDEXX Laboratories, Westbrook, ME) to detect antibodies to AI virus in wild birds. To date we have evaluated the IDEXX bELISA on experimental serum samples collected from previous AI infection trials (n=281) and field samples (n=2,249), both representing a wide diversity of avian taxa. The assay yielded relatively good diagnostic sensitivity 0.820 (95% CI: 0.756-0.874) and excellent specificity 1.000 (95% CI: 0.965-1.00), based on the experimental samples. In the field samples, the bELISA results were consistent with the known host range of AI, based on historic isolation reports. The bELISA readily identified known AI virus reservoirs and yielded negative results for taxonomic orders from which AI viruses rarely have been isolated. Collectively, the results of this research indicate that the IDEXX bELISA is a reliable and

markedly improved serologic assay in multiple wild avian species.

With the availability of a reliable serologic test for wild bird AI surveillance, there is enormous potential to improve our existing knowledge of AI epidemiology. Serology is an excellent cost-efficient diagnostic approach to screen wild bird populations in which AI infection status is unknown, and, as a supplement to virus isolation, serologic data can greatly improve our abilities to interpret AI host range. Although there are numerous potential benefits and applications for serologic testing in wild bird AI surveillance strategies, the following facts should be considered when serology results are interpreted:

- The bELISA identifies antibodies directed against AI viruses. A simple positive sample provides no information on viral subtype, pathotype, or when the infection occurred.
- It currently is not known how long detectable antibodies persist in an individual wild bird.
- AI viruses are maintained and most frequently detected in wild avian species in the Orders Anseriformes and Charadriiformes. However, AI viruses frequently spill-over into, and have been isolated from, a wide diversity of avian species and mammals. Consequently, as serology is used more frequently in wild bird AI surveillance, we should expect to discover antibodies to AI virus in numerous avian species. These positive results will not necessarily reflect new reservoirs for AI virus; they may relate to spillover events. (Prepared by Justin Brown)

Chagas Disease Studies

Since 2006, SCWDS has been investigating the natural history of *Trypanosoma cruzi,* the vector-borne protozoan parasite that causes Chagas disease in humans. The parasite is endemic from the southern United States to southern South America. Chagas disease may result from high numbers of parasites circulating in the blood during early stages of infection or from damage to muscle, especially cardiac tissue, during the chronic stage. The parasite has a known host range of approximately 200 species or subspecies of mammals. While many species of wildlife may act as asymptomatic reservoirs for the parasite, humans and some mammalian species, most notably domestic dogs, may become ill or die.

Few human cases acquired in the United States have been reported, but recent screening of blood donations conducted by the American Red Cross and Blood Systems, Inc. revealed over 1,000 seropositive individuals. This indicates exposure to *T. cruzi* and suggests that occurrence of the disease in this country may be higher than previously observed. In addition, fatal canine and exotic animal cases are reported annually. Previous studies have shown that several wildlife species, including raccoons, Virginia opossums, striped skunks, woodrats, and nine-banded armadillos are commonly infected with *T. cruzi*.

In August of 2007, SCWDS was awarded a grant from the National Institutes of Health to investigate the ecology of *T. cruzi* in the United States. The primary objectives were to investigate infection dynamics of *T. cruzi* isolates from different wildlife hosts and characterize these strains using a combination of gene sequence analysis, experimental laboratory growth studies in cell lines, transmission trials, and experimental inoculation trials in laboratory mice.

One important finding has been that carnivores might not play an important role in *T. cruzi* transmission by feeding on carcasses of infected animals. The primary route of transmission to human or other animals occurs when the infective trypomastigote stage of the protozoan is shed in the feces of a reduviid bug (kissing bug) during feeding and enters through a break in the skin or mucus membrane. Because alternate transmission routes such as ingestion of infected reduviid bugs and/or meat have been suggested, we conducted an experimental trial to test both of these potential transmission routes. Our trials revealed that ingestion of *T. cruzi*-infected reduviid bugs by raccoons resulted in infection, which supported previous studies with opossums and skunks. In contrast, we were unable to transmit *T. cruzi* among raccoons by feeding *T. cruzi*-infected tissues to raccoons. These results suggest that wildlife reservoirs are unlikely to become infected when scavenging on *T. cruzi*-infected carcasses, but

that ingestion of infected reduviid bugs can result in infection.

Another interesting finding was that raccoons and Virginia opossums are generally infected with different genetic strains of the parasite. Testing of isolates from naturally infected animals showed that Virginia opossums were infected only with Type I *T. cruzi,* while raccoons were more often infected with Type II *T. cruzi.* Subsequent experimental infection trials with these two important wildlife reservoirs supported our findings in the field. Experimentally inoculated raccoons became infected with both Type I and Type II strains of *T. cruzi* from the United States, while opossums became infected with only Type I strain from the United States. Although raccoons became infected with both strains, infections with Type II *T. cruzi* resulted in higher numbers of parasites in the blood and a longer period of detectable infection. In general, the numbers of parasites in the blood of opossums slowly increased but declined rapidly; whereas, parasite numbers in raccoons peaked sooner and remained high for five weeks. In addition, raccoons developed an antibody response to infection sooner compared with opossums. None of the raccoons or opossums experimentally inoculated with *T cruzi* developed clinical disease. Similarly, disease has not been reported in naturally infected raccoons or opossums.

Collectively, these data suggest that infection dynamics of different *T. cruzi* strains can differ considerably in different wildlife hosts. These differences must be considered when conducting surveillance for the parasite and when establishing the role of each host species as a reservoir for *T. cruzi.*

SCWDS recently received another grant from the National Institutes of Health to conduct related studies. The goals of this new study will be to characterize the immune response of selected host species to United States strains of *T. cruzi* and to better delineate the role of woodrats as reservoirs in the southwestern United States. (Prepared by Dawn Roellig and Michael Yabsley)

SCWDS Bont Tick Surveillance

SCWDS has been involved in field studies and surveillance for the tropical bont tick, *Amblyomma variegatum,* in the Caribbean region since 1985. *Amblyomma variegatum* is the vector of heartwater, a foreign animal disease that can cause morbidity and mortality in domestic ruminants and white-tailed deer. Over the last ten years we conducted surveillance for this and other exotic ticks in Puerto Rico and studied the role of wildlife in the maintenance and dissemination of *A. variegatum* in St. Croix, United States Virgin Islands. These programs are conducted in cooperation with USDA-APHIS-Veterinary Services and USDA-Agricultural Research Services.

Amblyomma variegatum is native to Africa and was introduced into the West Indies on cattle brought from West Africa to Guadeloupe in the late 1700s or early 1800s. Antigua and Marie Galante also were infested in the 19th century, but further spread in the region was not reported until Martinique became infested in 1948. Spread of the tick accelerated after 1948, and it has been found on islands from Barbados to Puerto Rico. The tick was declared to be eradicated from St. Croix in 1970 and from Puerto Rico in 1987. However, *A. variegatum* was found again in St. Croix during 2000, and an eradication program is ongoing.

Amblyomma variegatum is a three-host tick, and in Africa wildlife hosts include a wide range of mammals and birds. In the Caribbean, wildlife known to be infested by larvae and nymphs are the black rat, house mouse, small Asian mongoose, black-faced grassquit, cattle egret, and common ground dove. These species are present in St. Croix, as are white-tailed deer and feral cattle, both of which are potential hosts for larvae, nymphs, and adults of the tick. Wildlife infested by *A. variegatum* may hinder control or eradication efforts in the Caribbean because they may serve as maintenance hosts for the tick, and may disseminate the tick within a given island or between islands.

Amblyomma variegatum is a vector of *Ehrlichia ruminantium*, the etiologic agent introduced from Africa that is found in domestic livestock in Antigua, Guadeloupe, and Marie Galante. *Amblyomma variegatum* also is a vector of African tick-bite fever, a rickettsial zoonosis caused by *Rickettsia africae* reported in Guadeloupe, and it is strongly associated with acute bovine dermatophilosis, a systemic skin disease caused by *Dermatophilus congolensis* found on several islands in the region.

SCWDS conducted surveys for infestations of wildlife by *A. variegatum* in St. Croix during 2001, 2005, and 2006. Small mammals, birds, white-tailed deer, and feral cattle were examined in western St. Croix, where all known tropical bont tick-infested premises have been found. Small Asian mongooses and black rats yielded 1,710 ectoparasite specimens, including three tick species: a soft tick, *Carios puertoricensis;* the tropical horse tick, *Anocentor nitens;* and the southern cattle tick, *Rhipicephalus* (*Boophilus*) *microplus.* Birds yielded 116 ectoparasites representing at least 14 species of lice and mites, but no ticks. White-tailed deer and feral cattle yielded all life stages of *A. nitens* and *R. microplus* ticks. *Amblyomma variegatum* was not found on any wild or feral species sampled.

The absence of *A. variegatum* on small mammals, ground-feeding birds, white-tailed deer, and feral cattle examined in St. Croix suggests either absence or low local abundance when the surveys were conducted. Previous studies in Guadeloupe, Antigua, and Puerto Rico revealed infestations on both small mammals and birds, but populations of the tick on these islands, gauged by abundance of adult *A. variegatum* on cattle, were higher than in St. Croix. If mongooses, rats, or birds were infested in St. Croix during our survey period, the prevalence was below a level detectable with the sample sizes we used. Any such infestations probably were insignificant; but, even incidental infestations might allow small numbers of ticks to survive on wildlife in isolated areas.

We did not detect *A. variegatum* on white-tailed deer or feral cattle in St. Croix, so we have no evidence that these potential hosts were factors in the maintenance or dissemination of the tick during the study. White-tailed deer previously were examined for *A. variegatum* in the Caribbean Region on two occasions. None were found on 18 white-tailed deer examined in St. Croix in 1967-1968, or on five deer examined in Culebra, Puerto Rico, in 1989. However, tick-infested stray cattle significantly hinder control and eradication programs on other Caribbean islands, and the initial finding of *A. variegatum* in St. Croix in 2000 was on a stray or feral bull. The absence of *A. variegatum* on deer and feral cattle in our surveys does not rule out deer or feral cattle as sylvatic hosts if the tick becomes more abundant in St. Croix, nor does it rule out the possibility that current infestations of deer or feral cattle may exist at a low prevalence.
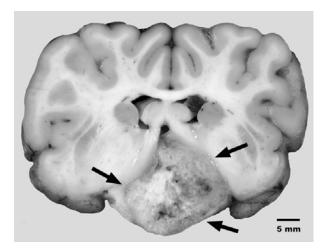
Notwithstanding the absence of *A. variegatum* in our surveys, we found abundant ticks and a diversity of other arthropod ectoparasites on wildlife examined. Chewing lice collected from a spotted sandpiper and feather mites collected from bananaquits and black-faced grassquits may represent new, undescribed species. The presence of *R. microplus* and *A. nitens* on white-tailed deer and feral cattle is of veterinary significance. Both previously have been found on deer in St. Croix, and we found a high prevalence and intensity of both *R. microplus* and *A. nitens* on white-tailed deer and abundant *R. microplus* on feral cattle, confirming that these hosts would represent a complicating factor in control programs where *R. microplus*, *A. nitens*, white-tailed deer and/or feral cattle coexist. (Prepared by Joseph Corn)

Brain Tumor in Deer

Last summer, wildlife personnel with the Louisiana Department of Wildlife and Fisheries received a call concerning very strange behavior of a white-tailed deer. The adult doe was found inside a fence near Baton Rouge and refused to leave, even though the gate was open. The deer was thin, not afraid of humans, and staggered periodically without falling. The animal was euthanized due to poor condition and neurological signs, and samples, including the head, were sent to SCWDS for examination.

When the brain was removed, the doe's problem was evident. On the bottom surface of the brain, there was a tumor about 1.5-2 centimeters in diameter. On cut surfaces, the tumor was soft and gray and it greatly compressed the parts of

the brain above it. In some areas, it was well demarcated from the brain tissue, but in other areas it blended in, suggesting invasion of normal tissue.

Microscopically, this tumor had characteristics typical of anaplastic oligodendrogliomas. These tumors arise from oligodendrocytes, which are cells that insulate neuronal axons. The tumors can invade surrounding tissue. In this deer, the tumor also invaded the brain, the optic nerves, and extended to the back of one eye. The animal's abnormal behavior was attributed to the compression and destruction of portions of the brain by the tumor mass. The deer also may have been blind as a result of the invasion of the optic nerves.

This is only the second white-tailed deer with an oligodendroglioma diagnosed at SCWDS, and there appear to be no reports in the literature of this type of tumor in this species.

Most neoplasms are more common in mature or aged animals, but white-tailed deer populations generally are dominated by younger age classes. As a result, the incidence of neoplasia in free-ranging white-tailed deer is relatively low, and most spontaneous tumors are not likely to have a significant impact on the population. This affected doe had significant dental wear, and her age was estimated to be greater than six years. (Prepared by Kevin Keel)

Exotic Animal Imports and Public Health

A study published in the November 2009 issue of *Emerging Infectious Diseases* (15:1721-1726)

by affiliates of the World Health Organization and the Wildlife Trust analyzed the risk of importing zoonotic diseases into the United States via the wildlife trade. The authors assessed the zoonotic disease risk from live mammals imported into 14 of the 18 designated United States animal importation ports from 2000 through 2005. To do so, they examined the volume and diversity of live mammals imported during the period and identified the zoonotic diseases that the imported species are known to host. They did not quantitate the risk or actually test any animals in the study. The authors concluded that their findings demonstrated "myriad opportunities for zoonotic pathogens to be imported and suggest that, to ensure public safety, immediate proactive changes are needed at multiple levels."

The study identified 27 "risk zoonoses" comprising viral, bacterial, and parasitic diseases that potentially could be harbored by imported wildlife, and determined which mammalian genera and families could serve as sources of these diseases. Each pathogen had to meet the following criteria for the disease to be listed as a risk zoonosis:

- It must be zoonotic.
- It must cause serious illness or death.
- It must be present in animals in the wild.
- It must not currently be widespread in the United States, or it must have potential for new epidemiology with regard to transmission.
- It must have competent vectors in the United States, if it uses a vector.

During the five year study, 246,772 live mammals representing 190 genera in 68 families were imported into the United States. The most common imports were long-tailed macaques, desert hamsters, rhesus macaques, raccoons, and chinchillas. The most common countries of origin were China, Guyana, the United Kingdom, Vietnam, and Indonesia. However, these source data must be interpreted cautiously, because the common practice of importation and re-exportation among multiple countries often makes the true country of origin difficult to determine. In fact, in more than 25% of cases the stated country of origin did not match the known natural geographic distribution of the animals that were listed as "wild-caught."

The zoonotic agents capable of infecting the greatest number of represented genera were rabies viruses, *Mycobacterium tuberculosis, Bacillus anthracis*, and *Echinococcus* spp. Imported genera capable of harboring the greatest number of risk zoonoses were dogs and cats, followed by rats, horses, macaques, and rabbits and hares. The family group that includes Old World mice and rats and their relatives posed the highest risk for zoonotic disease among the represented families; followed by New World rats and mice, gerbils and their relatives; dogs, coyotes, foxes, wolves, and jackals; antelope, cattle, goats, sheep and their relatives; and cats.

The authors cautioned that the study likely underestimates the risk posed by wildlife imports for the following reasons: This study involved only mammals, which tend to be imported in lower numbers than fish and reptiles. It examined only the disease risk from live animals and not from animal products or parts. The available data pertained only to legally imported animals, and the risk from illegal imports could not be determined. It was not possible to estimate the risk from potential pathogens that have yet to be identified.

The United States imports more wild animals than any other country, and over one billion live wild animals were brought here from other countries from 2000 through 2005. Because importing wild species carries the risk of inadvertently introducing zoonotic diseases, as well as diseases that could impact our native wildlife and domestic animals, the authors suggested several ways that zoonotic disease risks from imported wildlife could be reduced:

- Improve data collection, including the true country of origin, not only the most recent point of origin.
- Restrict the importation or enhance the surveillance of certain species known to present a high risk for a particular disease agent.

- Reduce disease transmission to humans by educating professionals and the general public about zoonotic diseases and safe wildlife handling.

Complete text of the article may be accessed at: http://www.cdc.gov/EID/content/15/11/1721.htm. (Prepared by Meaghan Broman, senior veterinary student, University of Wisconsin, Madison)

Loss of Two SCWDS Friends

In 2009, tragic accidents took the lives of two retired wildlife biologists with long histories of collaborative work with SCWDS.

In March, David Nelson, who had recently retired from the Alabama Division of Wildlife and Freshwater Fisheries, was killed in a tree cutting accident. David had over 30 years service with the agency, and in his capacity as statewide deer biologist assisted with many SCWDS deer herd evaluations in Alabama. In recognition of his productive career, the Southeastern Deer Study Group currently is considering a posthumous Career Achievement Award for Deer Management for David. We wholeheartedly support David's nomination for this award.

In November, John Collins, retired bear biologist with over 30 years service with the North Carolina Wildlife Resources Commission, drowned in a boating accident while squirrel hunting on the Johns River. John had been retired for several years and spent much of his retirement time hunting and fishing. For the past four years, I had the pleasure of spending a few days each September camping and fishing on the New River in West Virginia with John and several other retired or current North Carolina wildlife biologists.

John and David were good friends, good people, and were excellent resource stewards. They are sorely missed. (Prepared by Randy Davidson)

SCWDS BRIEFS

Southeastern Cooperative Wildlife Disease Study
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