January 2001

SCWDS BRIEFS: Volume 16, Number 4 (January 2001)

SCWDS BRIEFS

A Quarterly Newsletter from the
Southeastern Cooperative Wildlife Disease Study
College of Veterinary Medicine
The University of Georgia Athens, Georgia 30602
Phone (706) 542-1741 Fax (706) 542-5865
Gary L. Doster, Editor

Volume 16 January 2001 Number 4

AVM Kills Georgia Eagles

Avian vacuolar myelinopathy (AVM) was confirmed in eight bald eagles and is suspected in another six eagles that died from mid-November 2000 through late January 2001 at Clarks Hill Lake in eastern Georgia. The main body of the lake is formed by the Savannah River, which constitutes the Georgia/South Carolina border. On the South Carolina side, the reservoir is known as Lake Strom Thurmond. During the mortality event, SCWDS confirmed AVM in numerous American coots, two Canada geese, two great-horned owls, and a killdeer in the area surrounding the lake.

AVM was first recognized in the winter of 1994-95 when 29 bald eagles were known to have died from the disease in Arkansas. To date, AVM has caused the deaths of at least 82 bald eagles in Arkansas, Georgia, North Carolina, and South Carolina. Eagles with AVM exhibit difficulty or inability to fly or walk and have extensive vacuolar lesions in the white matter of the central nervous system. The cause of AVM remains undetermined despite extensive diagnostic and research investigations by experts with several state and federal wildlife resource agencies and universities. A natural or manmade neurotoxin is suspected because there has been no evidence of viruses, bacteria, prions, or other infectious agents and the lesions are consistent with toxicosis. AVM also has been detected in numerous American coots since 1996, and it is hypothesized that eagles are exposed to the causative agent of AVM via ingestion of affected coots.

AVM was diagnosed in ducks at Woodlake, North Carolina, in late 1998 and was suspected in two Canada geese at Clarks Hill Lake in late 1999 (SCWDS BRIEFS, Vol. 16, No. 2). The confirmation of AVM in geese, owls, and a killdeer this winter adds to the number of known susceptible species. The affected killdeer is the first bird with AVM that is not a species of waterfowl or a raptor. There has been no indication that mammals, including humans, are affected by AVM. However, public health and wildlife authorities recommend that, as with any sick wild animal, birds suspected of having AVM should be considered unfit for consumption.

While eagle mortality due to AVM has not been documented elsewhere this winter, coots with brain lesions have been found at other sites in Arkansas, Georgia, North Carolina, South Carolina, and Texas. AVM has been documented in these sites in past years. At Woodlake, North Carolina, where AVM is known to have occurred in coots since 1990, a cooperative AVM research project is underway that involves SCWDS, the National Wildlife Health Center, U.S. Fish and Wildlife Service, North Carolina Wildlife Resources Commission, and North Carolina State University. Investigators are continuing studies to determine the cause of AVM, its source and mode of transmission, and the range of affected species. (Prepared by John Fischer)

Bovine TB in Manitoba Elk

In 1997 an outbreak of bovine tuberculosis (TB) in cattle near Riding Mountain National Park in Manitoba, Canada, sparked considerable debate as to whether a wildlife reservoir was involved. A wildlife survey was initiated in and around the park using elk killed by hunters, elk and other animals

found dead, and road-killed animals. In the first year, TB was not found in a total of 200 white-tailed deer, elk, and moose that were examined. However, TB was confirmed in 2 of 563 elk examined during 1998-1999 and 2 of 453 elk examined during 1999-2000.

This was not the first time TB had been found in wild elk in this area. TB previously had been confirmed in a wild elk killed near Riding Mountain National Park in 1992. This elk was killed by a hunter in the vicinity of a cattle farm considered to be the source premise of a TB outbreak involving five cattle herds in Manitoba. The infected cattle herds were depopulated, and a subsequent survey of 55 hunter-killed white-tailed deer, elk, and moose was completed the following hunting season. From 1992 to 1997, no further evidence of TB was found in wild cervids in the area of the park, and the initial positive wild elk was believed to have been an isolated case of spillover from the infected cattle herd.

Riding Mountain National Park is a 1,500 square mile park located about 100 miles north of the United States - Canada border. In 1996 there were an estimated 5,500 wild elk in the park, while the surrounding agricultural region included about 174,460 head of cattle. A joint stakeholder group is working to develop a TB strategy for the area. The stakeholder group includes the Canadian Food Inspection Agency, Parks Canada, Manitoba Agriculture, Manitoba Natural Resources, livestock industry associations, and local livestock producers. Currently the wild elk population is being reduced through an increase in the number of hunting permits. Also being considered are exclusion fencing and capture, test, and cull programs. Studies are being conducted to identify movement patterns of elk from within the park. All cattle, farmed bison, and farmed cervid herds in the vicinity of the park have been tested for bovine TB and all were negative. (Prepared by Joe Corn)

CWD in a Nebraska Deer

Chronic wasting disease (CWD) of cervids has been found for the first time in a free-ranging cervid outside Colorado and Wyoming. The positive 3-year-old male mule deer was killed by a hunter in southwestern Kimball County, Nebraska, during November. Kimball County is adjacent to the CWD endemic focus in wild mule deer, elk, and white-tailed deer in northeastern Colorado and southeastern Wyoming.

CWD of cervids is a transmissible spongiform encephalopathy (TSE) related to, but distinct from, scrapie of sheep and bovine spongiform encephalopathy ("mad cow disease"). CWD was first recognized as a syndrome in cervids in the 1960s and was identified as a TSE in the 1980s. In addition to the endemic focus of CWD in wild deer and elk, CWD has been diagnosed in captive elk in Colorado, Montana, Nebraska, Oklahoma, South Dakota, and the Canadian province of Saskatchewan.

The Nebraska Game and Parks Commission has been concentrating CWD surveillance of wild cervids in the area between the North Platte and South Platte rivers. Since 1997, brain samples from more than 750 wild deer and elk voluntarily submitted by hunters have been examined for CWD. Agencies cooperating with the Game and Parks Commission to determine the extent of CWD in wild cervids and the steps necessary to control its spread include the Nebraska Department of Agriculture, the Animal Plant and Health Inspection Service of the U.S. Department of Agriculture, the Colorado Division of Wildlife, and the Wyoming Game and Fish Department. Targeted surveillance for additional wild deer with CWD has begun in the area where the positive wild deer was found, and the Nebraska Game and Parks Commission will expand its ongoing program to monitor hunter-killed deer for CWD.

Additional cases of CWD in Nebraska recently were detected in captive elk not geographically or epidemiologically associated with the wild mule deer in Kimball County. An individual elk was diagnosed with CWD in a herd from which previous CWD cases had been found; this herd subsequently was depopulated. CWD also was found for the first time in two captive elk at a game farm in the northwestern corner of Nebraska. The fate of this herd, which is currently under quarantine, is yet to be determined. (Prepared by

John Fischer with information provided by Bruce Morrison, Nebraska Game and Parks Commission)

Tropical Bont Tick on St. Croix

The U.S. Department of Agriculture recently announced that the tropical bont tick (Amblyomma variegatum) had been found on St. Croix in the U.S. Virgin Islands after several specimens were collected from a stray bull during August 2000. The tropical bont tick is native to Africa but was introduced to the Western Hemisphere in the 19th Century on African cattle brought to Guadeloupe, a French possession in the Caribbean. The tick has spread to 15 more islands in the Caribbean in the last 50 years. Two programs currently are in progress to eliminate the tick from the Caribbean countries in the region.

Inter-island movement of livestock, both legal and illegal, and passage of cattle egrets among the islands may both play a role in the spread of this tick. Collaborative research conducted by SCWDS and French scientists in the eastern Caribbean during 1988-1991 demonstrated that cattle egrets were moving among islands in the region and from the eastern Caribbean to the United States. Studies by SCWDS and French scientists also confirmed that cattle egrets were occasional hosts for small numbers of larvae and nymphs of the tick.

The tropical bont tick is recognized as a vector of Cowdria ruminantium, the etiologic agent of heartwater, an acute disease of domestic and wild ruminants. Cowdria ruminantium, a rickettsial organism, is native to Africa but has been confirmed on the islands of Antigua, Guadeloupe, and Marie Galante in the Caribbean. Although experimental data are limited, many domestic and wild ruminants in the Western Hemisphere probably are fully susceptible to heartwater disease. In one experimental trial, all untreated white-tailed deer died after being inoculated with C. ruminantium. If this organism becomes established in the United States, mortality rates among susceptible species such as cattle and white-tailed deer could be high. Because there is no officially recognized treatment or practical vaccine, prevention relies on control of the tick vectors. To complicate the situation, three species of Amblyomma native to the Americas (A. cajennense, A. dissimile, and A. maculatum) have been shown experimentally to be capable vectors, thus native hosts and vectors for C. ruminantium already are present and abundant if the organism reaches the mainland.

The tropical bont tick also is associated with two other diseases: African tick-bite fever and acute bovine dermatophilosis. African tick-bite fever is a tick-borne spotted-fever rickettsiosis caused by Rickettsia africae. It is a recently recognized disease of humans, cattle, and possibly other animals and is found in parts of Africa and Guadeloupe and may be present on other Caribbean Islands.

Acute bovine dermatophilosis is caused by the bacterium Dermatophilus congolensis. The tropical bont tick is linked epidemiologically to the presence of acute bovine dermatophilosis in the Caribbean, and the occurrence of this disease has resulted in extensive morbidity and mortality among cattle on affected islands in the region. On Nevis, for example, an epidemic of acute bovine dermatophilosis that began after introduction of the tick resulted in the loss of 90% of the cattle on the island in less than 10 years.

During September 2000, surveillance of domestic animals on St. Croix was begun by the USDA and the U.S. Virgin Islands Department of Agriculture, and a wildlife assessment was conducted by SCWDS. Additional specimens of A. variegatum were found on animals on the island during January-February 2001. Surveillance on the island has been complicated by the fact that the origin of the infested bull found in August is unknown, but it may have been associated with feral cattle on the island. White-tailed deer also inhabit the island and are being included in the surveillance effort. Feral cattle are restricted to the "rain forest" and surrounding areas on the western end of the island, but white-tailed deer are widespread; both are potential hosts for the tick. (Prepared by Joe Corn)

Aussie Rabbits Protected from Calicivirus

In recent years, Australian and New Zealand officials have been exploring several methods of

biological control in order to reduce burgeoning populations of introduced rabbits. In March 1995, researchers released Rabbit Calicivirus Disease (RCD) into a fenced quarantined area on Wardang Island, which is 2.5 miles from the Australian mainland. The virus soon escaped from Wardang Island and reached the mainland where it rapidly spread throughout the country (see SCWDS BRIEFS, Vol. 11, No. 4). Despite its initial uncoordinated release, the "runaway" disease caused a rapid and dramatic reduction of rabbit numbers on mainland Australia, particularly in arid regions. RCD was less effective, however, in controlling rabbit populations in temperate regions.

Now, evidence of a so-called "mild cousin" to the deadly RCD has been detected. It is possible that this similar, non-pathogenic virus may be responsible for the variable effectiveness of RCD in population control of the wild European rabbit in Australia.

Also known as viral hemorrhagic disease of rabbits, RCD affects only the European rabbit, Oryctolagus cuniculus, the species to which all pet and commercial rabbits in the United States belong. Wild rabbits of North America are not susceptible to RCD. European rabbits with RCD die within 6-24 hours of onset of fever. Lesions consist of hemorrhage and necrosis in the liver, intestine, and lymphoid tissues.

RCD was first recognized as a fatal rabbit disease in China in 1984. After its appearance in China, RCD was observed in Africa, Asia, and Europe. RCD first appeared in the Western Hemisphere in Mexico City in 1988. The disease subsequently was eradicated from Mexico. In the United States, RCD was recognized for the first time in March 2000, when an Iowa farm experienced acute mortality in 25 of 27 domestic rabbits (SCWDS BRIEFS, Vol. 16, Number 1). The remaining rabbits were depopulated, the premise was quarantined, and RCD was not detected elsewhere in the United States.

Recent advances in diagnostic assays have prompted scientists to test serum that was collected from wild Australian rabbits prior to introduction of RCD in 1995. Results indicate that the rabbits had antibodies to an RCD-like virus. Since there were no reports of an RCD-like disease in Australia prior to RCD's release, the virus that triggered those antibodies may have been a non-pathogenic but similar virus. In addition to testing stored serum, scientists tested serum from rabbits currently in RCD-free areas of Australia. These rabbits also had antibodies to the RCD-like virus.

In order to determine whether antibodies to the RCD-like virus might confer protection from RCD, scientists conducted a challenge study using wild rabbits with antibodies to RCD-like virus that were captured from an RCD-free region. In the challenge study, 11 of the 23 seropositive rabbits survived challenge with RCD virus. Furthermore, there was correlation between titer and survival: rabbits with high levels of antibodies to RCD-like virus had a better chance of surviving RCD. In essence, the pre-existing RCD-relative may be acting like a natural vaccine. Another aspect of the study showed that seroprevalence and titers of antibodies to the RCD-like virus were higher in cooler, wetter areas of Australia. This may explain why RCD killed more rabbits in the arid inland than in Australia's temperate areas.

Researchers now are working to develop a more specific serologic test to distinguish antibodies against the virulent and purported avirulent caliciviruses. In the meantime, this new information serves as a reminder of the inherent complexities involved when using microbiologic agents to control wild animal populations. (Prepared by Cynthia Tate)

Secondary Poisoning from Euthanasia Drug

Veterinarians use highly concentrated solutions containing sodium pentobarbital for euthanasia of both pets and farm animals. This potent drug, which is usually given intravenously, produces rapid unconsciousness without pain or distress to the animal, and lethal injection is considered an ideal method of euthanasia. Unfortunately, few people are aware that there is a hazard for secondary poisoning with euthanasia solution. In a notable case, 26 bald eagles were poisoned, 5 fatally, following ingestion of a cow that had been euthanized in British Columbia. The problem has

occurred sporadically in both bald and golden eagles throughout the United States, and barbiturate toxicosis has become a too familiar diagnosis in eagles submitted to the National Wildlife Health Center, Madison, Wisconsin, and the U.S. Fish and Wildlife Service Forensics Laboratory in Ashland, Oregon.

At present, the problem appears to be limited to eagles; however, it is likely to affect other wildlife species that ingest the euthanized carcass. There is one reported case of a lion being poisoned after it was fed a euthanized horse. Farm animals, mainly cows and horses, that are left exposed in remote locations are frequent sources of toxicosis, but dog and cat carcasses that are not properly covered in landfills also are hazardous. Cases have been diagnosed more frequently in the winter and early spring, probably as a result of the difficulties associated with burying carcasses in frozen ground and the shortage of natural foods.

Sodium pentobarbital is a well-known sedative/ anesthetic, and therefore, clinical signs in affected birds include drowsiness, incoordination, and ultimately, unconsciousness and death. Poisoned birds may be found near the tainted carcass source or at distant locations. Birds that are not dead may recover if given supportive care; removal of the crop contents may be helpful. The best diagnostic samples to confirm sodium pentobarbital poisoning are stomach contents or liver from the affected animal. Tissue samples from a suspect source carcass also would be useful. Veterinarians, landfill operators, and farmers who improperly dispose of euthanized animal carcasses may be held legally responsible for wildlife toxicoses under authority of several federal regulations, so awareness of this potential problem is in everyone's best interest. (Prepared by Vic Nettles)

USDA Funds VS Study

SCWDS researchers recently were awarded a USDA-funded grant to investigate the role of black flies in the transmission of the New Jersey serotype of vesicular stomatitis virus (VSV-NJ) during epizootics. This virus is one of the causative agents of vesicular stomatitis (VS), an arthropod-borne disease which primarily affects cattle, swine, and horses, causing vesicular lesions on the mouth, coronary bands of the hooves, and teats. Other livestock and wildlife species also can be infected.

The goals of this 3-year study are to determine if VSV-NJ epizootics are dependent on transmission cycles involving black flies, to understand how these vectors become infected with VSV-NJ, to determine if black flies can transmit the virus to livestock species, and to document the clinical response in the animals infected via black fly bite. Our specific objectives are to: (1) confirm the vector competence of black flies (Simulium vittatum and S. notatum) using a large animal model (horses and pigs) and to document the clinical response to VSV-NJ infection in these hosts; and (2) determine if black flies can be infected with VSV-NJ by direct feeding on infected hosts and/or feeding simultaneously with black flies or uninfected hosts.

Within the United States, VSV-NJ is enzootic on Ossabaw Island, Georgia. VSV-NJ also is the serotype most often associated with the recurring and unpredictable epizootics in the western states. Recent VSV-NJ epizootics occurred in the western United States in 1982-83, 1985, 1995, and 1997.

Despite intensive study, several aspects of the epizootiology of VSV-NJ, including modes of transmission and enzootic maintenance, remain largely unknown and highly controversial. Ossabaw Island, Georgia, is the only area where a biological vector (the sand fly Lutzomyia shannoni) has been identified, and while it generally is accepted that sand flies (Lutzomyia spp.) are important enzootic vectors of VSV-NJ, they are not believed to be important epizootic vectors because of their limited flight range. This has been demonstrated on Ossabaw Island where VSV-NJ transmission among sand flies and wild swine is spatially restricted to areas with maritime forests. The epizootic vector(s) of VSV-NJ have not been fully identified.

Information regarding the transmission of VSV-NJ during epizootics is based largely on the limited observational and entomological studies conducted during the sporadic epizootics in the western United States. During epizootics, VSV-NJ has

been isolated from biting midges, mosquitoes, black flies, and non-biting flies. It is not known how these insects become infected with the virus because viremia has not been documented for any wild or domestic animal species commonly affected or exposed in VSV-NJ epizootics. Animals evaluated for viremia have included naturally and experimentally infected swine, cattle, horses, white-tailed deer, and pronghorn antelope. Viremia was detected in juvenile and suckling deer mice that were infected with VSV-NJ by intra-nasal and intra-dermal inoculation and in adult deer mice following intra-nasal inoculation (see SCWDS BRIEFS, Vol. 13, No. 2), however, it was not determined whether viremia was sufficient to infect blood-feeding insects. In addition, it has yet to be demonstrated that an insect infected with VSV-NJ can transmit the virus to large animals. Our project directly addresses these issues. (Prepared by Danny Mead)

SCWDS E-mail Addresses

We have a new Internet server, therefore e-mail addresses for SCWDS staff members and graduate students have been changed. We know that some of you have had difficulty contacting us recently, so following is a list of the email addresses of current SCWDS personnel:

Jeanenne Brewton = jbrewton@vet.uga.edu
Joe Corn = jcorn@vet.uga.edu
Sarah Cross = scross@vet.uga.edu
Randy Davidson = rdavidso@vet.uga.edu
Gary Doster = gdoster@vet.uga.edu
Vivien Dugan = vgdugan@vet.uga.edu
John Fischer = jfischer@vet.uga.edu
Joe Gaydos = jgaydos@vet.uga.edu
Britta Hanson = bhanson@vet.uga.edu
Darrell Kavanaugh = dkavanau@vet.uga.edu
Kali King = kking@vet.uga.edu
Lynn Lewis = llewis@vet.uga.edu
Page Luttrell = luttrell@vet.uga.edu
Cindy McElwee = cmcelwee@vet.uga.edu
Danny Mead = dmead@vet.uga.edu
Vic Nettles = vnettles@vet.uga.edu
Rob Olson = rolson@vet.uga.edu
Charlotte Quist = cquist@vet.uga.edu
David Stallknecht = dstall@vet.uga.edu
Mike Stevenson = michaels@vet.uga.edu
Cynthia Tate = ctate@vet.uga.edu
Donna Wood = dwood@vet.uga.edu
Michael Yabsley = myabsley@vet.uga.edu
Anna Yellin = anna@vet.uga.edu

* *

Information presented in this Newsletter is not intended for citation in the scientific literature. Please contact the Southeastern Cooperative Wildlife Disease Study if citable information is needed.

* *

Recent back issues of SCWDS BRIEFS can be accessed on the Internet at www.SCWDS.org.