
SCWDS BRIEFS

A Quarterly Newsletter from the
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CWD News from Nebraska and Kansas

Infection with the chronic wasting disease (CWD) agent recently was found in 28 of 58 formerly wild white-tailed deer in a high-fenced enclosure adjacent to a pen containing CWD-affected captive elk in northern Sioux County, Nebraska. Four of the positive deer were fawns approximately 8 months old, which is unusually young for animals testing positive for CWD. A January survey of 39 free-ranging deer collected within 15 miles of the positive elk and deer pens detected 8 (20%) infected animals. Test results are pending for additional deer collected inside and outside of the enclosure, and additional surveillance is planned for free-ranging deer in northwestern Nebraska. Previously, CWD had been documented in Nebraska in only two wild mule deer, both of which came from Kimball County in the southwestern panhandle adjacent to the endemic area of northeastern Colorado and southwestern Wyoming.

The origin of CWD in this situation remains unknown, and there has been no documented commingling of the captive elk, enclosed deer, or free-ranging deer in the area. The elk facility and deer enclosure have been in existence since 1993. CWD first was found there in two elk in December 2000, and five additional positive elk have been detected since then. The Nebraska Department of Agriculture is formulating a plan to depopulate the remaining 80 captive elk at this facility, and the Nebraska Game and Parks Commission (NGPC) is working with the owner to destroy and test the remaining deer in the enclosure.

The prevalence of CWD in deer in the enclosure and in free-ranging deer is alarmingly high and is an indicator of the great risk this disease represents to wild white-tailed deer populations. The NGPC has declared CWD a wildlife disease emergency and has taken action to determine the extent of the problem and control it. During the 2001 hunting season, samples were collected from 805 hunter-killed deer in western Nebraska, including 125 deer from Sioux and Dawes counties. Laboratory results are pending on approximately 500 samples, but 1 of 37 deer from Kimball County tested positive. The positive animal was taken within 7 miles of the two positive wild mule deer found last year in Kimball County. CWD was not detected in 32 additional deer from throughout the state that fit the CWD target profile of an animal older than 1½ years with emaciation and neurological signs. All wild elk harvested in Nebraska since 1997 have tested negative for CWD, and results are pending on 31 elk harvested during the 2001 hunting season.

The NGPC recently decided to disallow private ownership and importation of mule deer. Currently there are five captive mule deer facilities with approximately 300 animals operating in the panhandle region. These operations may keep the deer and their offspring; however, they may not import animals into the state and cannot sell live animals within the state to other operators.

In addition to the Nebraska cases, CWD has been diagnosed for the first time in Kansas. In December 2001, CWD infection was confirmed in a captive elk that had been moved from a

positive herd in Colorado to a small herd of captive elk and deer in south-central Kansas. Elk from the positive Colorado herd had been shipped to commercial elk facilities in 19 states, and positive animals have been found in 2 of more than 40 other Colorado elk ranches that received animals from the source herd. The remaining 17 elk and 2 white-tailed deer in the Kansas herd were depopulated in mid-January 2002. The state of Kansas is providing one-half of the compensation to the owner for the destruction of the animals and the USDA's Animal and Plant Health Inspection Service (APHIS) is providing the remainder. (Prepared by John Fischer with assistance from Bruce Morrison of the Nebraska Game and Parks Commission)

Tropical Bont Tick Threat

The tropical bont tick, *Amblyomma variegatum*, was discovered on cattle on St. Croix, United States Virgin Islands, in 1967 and 1987 and was eradicated on both occasions. The tick made another appearance in August 2000, when an infestation was detected on a stray bull. Additional ticks were found on domestic cattle and horses in 2001, and efforts were undertaken to determine the extent of the infestation and to develop a plan to eradicate the tick.

The tropical bont tick is native to Africa and is a vector of *Cowdria ruminantium*, the rickettsia that causes heartwater, an acute and potentially devastating disease of ruminants. Introduction of heartwater and its vector into the United States would severely threaten the nation's cattle, sheep, and goats, as well as white-tailed deer and other wild ruminants. The tropical bont tick also is associated with African tick-bite fever and acute bovine dermatophilosis. *Amblyomma variegatum* was first introduced to the Western Hemisphere in the 19th Century on African cattle brought to Guadeloupe. Three islands in the Caribbean became infested in the 19th Century, but the tick has spread to 15 more islands in the region in the last 50 years.

As part of the investigation of the tick's incursion onto St. Croix, Emergency Programs of USDA's Animal and Plant Health Inspection Service (APHIS) requested that SCWDS evaluate the current and potential role of the island's deer and feral cattle in the maintenance and dissemination of the tick. During May through September 2001, SCWDS personnel conducted an extensive search for evidence of white-tailed deer and feral cattle activity on St. Croix, and 10 deer and 20 feral cattle were collected and examined for ticks. Special efforts were made to detect deer and feral cattle activity in the vicinity of the known location of the tick infestation. Although evidence of deer and feral cattle activity was not apparent in the immediate vicinity of the tick infestation, both deer and feral cattle were seen in nearby areas. Specimens of the tropical bont tick were not recovered from any of the deer or feral cattle, but tropical cattle ticks, *Boophilus microplus*, were recovered from all of the deer and cattle, and 5 of the deer and 10 of the cattle were infested by the tropical horse tick, *Anocentor nitens*.

Although deer and feral cattle on St. Croix currently do not appear to be infested with *A. variegatum*, the potential exists for the tick to be spread into areas where deer and feral cattle are present or for deer and/or feral cattle to disperse into the currently infested area. The immediate elimination of livestock movements and treatment of livestock in the infested area are needed to stop current spread of the tick, but eradication will be necessary to avoid future spread. The numbers of deer and feral cattle on St. Croix may be too low for these animals to maintain a population of *A. variegatum*; however, domestic livestock provide ample hosts to perpetuate the life cycle of the tick. If deer and feral cattle become infested, they could potentially serve as a source of re-infestation for domestic livestock and would likely spread the tick to other parts of the island. (Prepared by Joe Corn)

AVM Eagle Toll Climbs

Avian vacuolar myelinopathy (AVM) was confirmed by SCWDS diagnosticians in two dead bald eagles and is suspected in another four decomposed eagle carcasses recovered since mid-November at Clarks Hill Lake along the Georgia/South Carolina border. The reservoir is known as Lake J. Strom Thurmond on the South Carolina side and is the site where AVM was confirmed or suspected in 13 dead bald eagles during the winter of 2000-2001 (SCWDS BRIEFS Vol. 16, No. 4). AVM also has been found in several Canada geese and American coots from the lake since late October 2001. The disease has not been confirmed in any other species at the site this year, although it was found in two great horned owls and a killdeer during the previous winter.

Elsewhere, the National Wildlife Health Center (NWHC) has confirmed AVM in one dead eagle and suspects the disease in another eagle from Lake Ouchita, Arkansas. SCWDS found AVM in American coots at Lake Ouchita shortly before the first dead eagle was found. Avian vacuolar myelinopathy first was recognized as a cause of eagle mortality when it killed 29 bald eagles at nearby DeGray Lake, Arkansas, in the winter of 1994-95. Since then, AVM has killed at least 85 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. AVM can be confirmed only by microscopic examination of brain tissue from a fresh specimen. The cause of AVM remains undetermined despite extensive diagnostic and research investigations. A natural or manmade compound 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 was suspected that eagles were acquiring AVM by ingesting affected coots. This hypothesis was

proven earlier this year when SCWDS researchers reproduced the disease in unreleasable, rehabilitated red-tailed hawks that were fed tissues from coots with AVM (SCWDS BRIEFS Vol. 17, No. 2). Additional species in which AVM has been confirmed include mallard and ring-necked ducks and Canada geese. To date, AVM has not been confirmed in mammals, and it remains unknown whether the causative agent of AVM could affect humans. 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.

Eagle mortality due to AVM has not been documented elsewhere this winter. However, AVM has been found by SCWDS and NWHC in American coots again this winter at sites where it has previously occurred, including DeGray Lake, Arkansas; Lake Juliette, Georgia; Woodlake, North Carolina; the Savannah River Ecological Laboratory in South Carolina; and Sam Rayborn Reservoir, Texas. Personnel from SCWDS, NWHC, Arkansas Game and Fish Commission, Georgia Department of Natural Resources, South Carolina Department of Natural Resources, U.S. Army Corps of Engineers, U.S. Fish and Wildlife Service, and others are continuing cooperative efforts to determine the cause of AVM, its source and mode of transmission, and the species susceptibility range. (Prepared by John Fischer)

U.K. Regaining FMD Free Status

On January 29, 2002, the Office International des Epizooties (OIE) agreed to restore the United Kingdom's Foot-and-Mouth Disease (FMD) free status without vaccination for the purposes of international trade. This followed the declaration on January 15 that Northumberland County is FMD free. Northumberland was the last county to achieve this status following the United Kingdom's nearly year-long battle to eradicate FMD. The OIE decision clears the way for the United Kingdom to resume trade in animals and animal

products for member countries of the OIE. Trade restrictions placed on imports from the United Kingdom by other countries likely will be eased following the OIE determination, however they remain in effect until changed. This declaration does not mean that all work on the FMD problem is concluded, because some premises have yet to be fully cleaned and disinfected. If secondary cleaning and disinfection is not undertaken on these premises, they will have to remain under restriction for 12 months. In addition, some other restrictions remain in place, such as access to a few hiking footpaths that cross formerly infected premises. Ireland, France, and The Netherlands, which had cases linked to the United Kingdom, regained their FMD free status during September 2001. Details of the outbreak and associated regulatory issues can be found at web sites for the United Kingdom Department for the Environment, Food and Rural Affairs (www.defra.gov.uk) and the OIE (www.oie.int).

This outbreak clearly illustrates the impact that FMD or other diseases can have when they gain entry into countries where they do not occur. Compared to the relatively small geographic size of the United Kingdom, its smaller livestock industry, and fewer numbers of FMD-susceptible wild animals, one does not need much imagination to recognize the problems that FMD or other foreign animal diseases could cause in the United States. The 2001 FMD outbreak was contained and eliminated through prompt and aggressive responses. This event should serve as an important lesson of the need for constant vigilance for destructive foreign animal diseases. (Prepared by Randy Davidson)

Raccoon Roundworms – Public Health Update

Baylisascaris procyonis is a common intestinal roundworm of raccoons that causes central nervous system disease, known as neural larval migrans, in many species of birds and mammals, including humans. A less severe form of disease, called ocular larval migrans, affects vision. When a non-raccoon host ingests

eggs passed in the feces of infected raccoons, larvae migrate through visceral organs and frequently enter the spinal cord and brain. In 2000, two human cases were diagnosed in Illinois and California. Both boys, aged 2½ and 17, had a history of pica (the ingestion of unnatural objects such as dirt and bark) which has been common among previous human cases of *B. procyonis* infection. One boy died following a year-long coma while the other boy remains severely disabled. Five of the other 10 previously reported human neurological cases have been fatal. Surviving patients have had blindness, seizures, paralysis, and severe mental and physical retardation. Cases have been reported mainly in children (9 mo – 6 yr) from the Midwest, Northeast, and West Coast. In addition, dozens of ocular larval migrans cases have been reported, with clinical presentation ranging from mild sight loss to blindness.

Many other species of roundworms can cause larval migrans. Larvae of the common dog and cat roundworms, *Toxocara canis* and *T. cati*, undergo migration through visceral organs, similar to *Baylisascaris* spp. Although *T. canis* is the most common cause of larval migrans in humans, this parasite rarely enters brain tissue and is much less pathogenic than *Baylisascaris*. Exposure of children to *Toxocara* is common, and the prevalence of antibodies in humans is 30% in some areas of the United States. Other *Baylisascaris* spp. that can cause larval migrans in domestic and wild animals include *B. columnaris* of skunks, *B. melis* of badgers, and *B. transfuga* in bears.

In addition to human cases, neural larval migrans caused by *B. procyonis* has been reported in more than 90 species of mammals and birds, and extensive losses have been documented in domestic and research animals. Outbreaks have been documented in prairie dogs, rabbits, guinea pigs, chickens, and pheasants in research and commercial facilities. All of the bobwhite quail kept in a pen previously occupied by infected pet raccoons died from larval migrans. Recently, *B. procyonis* larval migrans killed a group of 10

pet parrots. All of these cases resulted from using litter, pens, or feeds contaminated by *B. procyonis* eggs in raccoon feces. Some animals, such as opossums, domestic livestock, cats, and raptors, appear somewhat refractory to infection.

Baylisascaris larval migrans has been diagnosed in numerous species of free-ranging wildlife including beavers, coyotes, foxes, mice, rabbits, squirrels, woodchucks, woodrats, bobwhite quail, herons, mourning doves, owls, and wild turkeys. Infected animals exhibit a range of neurological signs. Many woodchucks in New York with *B. procyonis* larval migrans originally were submitted as rabies suspects due to abnormal behavior. Most previous reports of wildlife cases of *Baylisascaris larval migrans* have been sporadic events in single or small groups of animals, although 25% mortality was estimated for a cottontail rabbit population in Virginia. Currently, the impact of *Baylisascaris larval migrans* on wildlife populations is not known. Fatal cases also have been diagnosed in wildlife rehabilitation centers where animals were housed in cages that previously contained raccoons.

Prevention of *B. procyonis* infection in humans is important because of the grave prognosis for infected individuals and the lack of effective treatment. Areas endemic for *B. procyonis* can quickly become highly contaminated as about 70% of adult raccoons and more than 90% of juvenile raccoons may be infected, and an infected raccoon is capable of shedding 2 to 10 million eggs per defecation. The eggs of *B. procyonis* are extremely resistant to common disinfectants (e.g., bleach, quarternary ammonium, chlorohexidine) and environmental conditions and can survive in the soil for years. Heat (boiling water or fire) and very strong lipid solvents (50/50 solution of xylene and absolute alcohol) are the most effective ways of killing *B. procyonis* eggs, but the latter is not safe for field use. Minimizing raccoon access is critical to prevent contamination of premises, bedding, or feed with raccoon feces. Any bedding or feed suspected of being contaminated with

raccoon feces should be burned. Pens previously used to house raccoons at research or rehabilitation facilities should be thoroughly decontaminated before the addition of any other animals, and keeping raccoons as pets should be discouraged. (Prepared by Michael Yabsley)

CSF Control Targets Wild Boars

Classical swine fever (CSF), also called hog cholera, is a severe disease of swine caused by a pestivirus in the family Flaviviridae. Although eradicated from the United States in the 1970s, CSF remains a significant problem in domestic swine around the world, and its recurrence in the United States could be economically devastating. In some European countries, wild swine (called European wild boars) play a role in the epidemiology of CSF, and some disease control measures are targeted at these animals.

The origin of CSF is uncertain, but by the 1860s, the disease was widespread in Europe and America. The United States launched an eradication program in 1961 and the last case was reported in 1976. However, CSF remains a costly problem to swine producers in several countries in Central and South America, the Caribbean, Asia, and Europe, as well as a potential threat to the United States.

In 1980, the European Union instituted measures with the goal of CSF eradication. Control strategies in domestic swine include depopulation of affected and suspect animals, surveillance, and restriction of animal movements. Efforts also have been made to control CSF in wild boars because endemic infections or disease outbreaks have been identified in boar populations in parts of Austria, France, Germany, Italy, Slovakia, and the Ukraine. In Germany, wild boars are regarded as a primary risk factor for infection of domestic swine.

Since 1998, the European Commission has promoted selective hunting to control CSF in wild boars. Under this protocol, hunting is discouraged when an outbreak is first identified

in order to reduce potential dispersal of infected animals. After 6 months, selective hunting of young pigs may be employed to reduce the susceptible population. Reduction of older animals in the affected population is regarded as unnecessary because they most likely have developed immunity. These management methods reportedly eliminated a 1998 outbreak in wild boars in Switzerland.

The European Commission also advocates targeted vaccination campaigns among well-defined wild boar populations. Vaccination campaigns will last at least 2 years. Additional objectives include minimal seroconversion rates of 80% in populations of 1,000 animals and 60% in populations of 500. In a German field trial in the mid-1990s, oral vaccine was administered to wild boars via baits. Bait uptake ranged from 85 to 100%, and seroconversion rates among animals older than 2 years ranged between 63.2 and 100%. However, seroconversion rates were only 44% among animals under 1 year, even after distributing vaccine-laden oral baits four times.

Additional studies have shown that domestic pigs vaccinated with the oral C-strain vaccine still become viremic and shed virus after challenge with CSF virus, but viral spread to unvaccinated animals was decreased. Several recent studies have been devoted to development of "marker vaccines" that would allow the distinction between vaccinated and naturally infected pigs. Although safe, the effectiveness of one candidate vaccine was less than ideal because transplacental infection was decreased but not eliminated. An effective marker vaccine and companion serologic test are desirable components of CSF control and eradication strategies, thus their development remains an area of active research. (Prepared by Laura Kelley)

Maternal Antibodies and Innate Resistance to HD

Hemorrhagic disease (HD) is one of the most important infectious diseases of white-tailed

deer. Spatially, HD is not uniformly distributed throughout the United States. Epizootics in northern latitudes are infrequent and characterized by severe clinical disease and mortality, whereas epizootics in southern latitudes are more frequent and often result in mild or inapparent infections. In some areas, such as Texas, disease is extremely rare, but antibody testing indicates that a high proportion of adult deer are routinely exposed to both epizootic hemorrhagic disease (EHD) and bluetongue (BT) viruses (SCWDS BRIEFS Vol. 8, No 4). It is thought that this represents a case of enzootic stability where the viruses and host have achieved a near-perfect balance. It has been hypothesized that this may be due to innate host resistance and/or protection of fawns through maternal antibody transfer. Last year, we investigated both of these hypotheses at SCWDS.

In the spring of 2000, white-tailed deer fawns were obtained from an outdoor white-tailed deer research facility operated by the Texas Parks and Wildlife Department. All of the fawns had maternal antibodies to EHD and BT viruses, supporting the theory that this represented a site of enzootic stability. These fawns were moved indoors at the University of Georgia, and blood samples were collected from them weekly. Maternal antibodies to EHD and BT viruses could not be detected in fawns after 13-18 weeks of age. Fawns that remained outdoors in Texas never had clinical signs of HD, but by October, when most fawns were over 18 weeks old, they had elevated antibody titers to EHD and BT viruses suggestive of natural exposure. More significantly, EHD viruses were isolated from 18% of these fawns, confirming that EHD and BT viruses were circulating in the absence of disease. These data suggest that subclinical infection by EHD and BT viruses occurs at this site despite the presence of maternal antibodies. This suggests that passive transfer of maternal antibodies may be a factor in the maintenance of enzootic stability of EHD and BT viruses at this location. These data did not exclude the possibility that these deer also may have innate resistance to these viruses.

To test the theory of innate resistance, additional fawns were acquired from Pennsylvania. When all maternal antibodies had completely disappeared, subsets of fawns from both Texas and Pennsylvania were experimentally infected with EHD virus, serotype 1 (EHDV-1), and other subsets were experimentally infected with EHD virus, serotype 2 (EHDV-2). Deer were closely monitored, and physical examination, clinical pathology, and clotting profiles were used to assign each deer a clinical disease severity score. These scores were dramatically higher for Pennsylvania deer than for Texas deer infected with EHDV-1 and -2. Clinical disease was absent or mild in Texas deer, but in the Pennsylvania deer EHDV-1 and -2 infection caused 100% and 20% mortality, respectively. Viremia and humoral immune response were similar in both groups of deer, suggesting that deer from this site in Texas have innate resistance to clinical disease caused by infection with EHDV-1 and -2. Despite this innate resistance to clinical disease, these deer still became infected and could serve as virus amplifying hosts in a natural setting.

Of these two findings, innate resistance to EHD may be more important in maintaining enzootic stability in this area. Demonstration that deer populations of different geographic origins differ in their innate resistance to EHD has even greater implications. Genetic differences in resistance to EHD may be responsible for the

severe HD epizootics in the northern latitudes compared to the milder outbreaks in the southern latitudes.

Genetic differences in resistance to EHD also have implications concerning the translocation of white-tailed deer. Deer in Texas experience frequent and intense exposure to EHD and BT viruses, and over time this exposure probably has selected for resistance to HD in this population. Conversely, deer in more northern latitudes are not constantly exposed to these viruses and therefore have not experienced this selection pressure. In light of this, it makes sense that SCWDS often receives reports about deer translocated into southern states from northern latitudes dying of HD while sympatric native southern deer are unaffected. Deer translocated from northern latitudes that pass on their genes before dying of HD may actually be diluting a beneficial disease resistance trait that has evolved over time in native southern deer. The idea that disease resistance can vary within a species depending on the animal's genetic makeup now highlights the concern that animal translocation may not only introduce unwanted pathogens into an area but also introduce unwanted genetic material into a population. (Prepared by Joe Gaydos)

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