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A Quarterly Newsletter from the
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More Chronic Wasting Disease

Chronic wasting disease (CWD) recently has been identified in more free-ranging and captive cervids at additional locations in New Mexico, Wisconsin, and Colorado. The detection and management of this disease continues to be a front-burner issue for state wildlife management and animal health agencies, as well as the U.S. Department of Agriculture (USDA), U.S. Department of the Interior, and the U.S. Congress.

On June 19, 2002, the New Mexico Department of Game and Fish (NM DGF) announced that CWD had been confirmed in New Mexico for the first time in a free-ranging mule deer at the White Sands Missile Range. The animal was collected on March 28, 2002, and tested because it had clinical signs of CWD (emaciation and neurological disease), again emphasizing the importance of targeted CWD surveillance. The origin of CWD in this instance remains unknown because there are no known captive cervid facilities in the vicinity, which is several hundred miles from Colorado's endemic area. Wildlife management officials in New Mexico declared an Animal Health Emergency and closed the state's borders to importation of live cervids. The NM DGF will initiate testing of more deer as soon as possible in order to identify any other infected animals and to determine the geographic distribution of the disease. Additional information on CWD in New Mexico may be found at www.gmfsh.state.nm.us.

In Wisconsin's affected area, four additional wild white-tailed deer have been diagnosed with CWD via testing of lymphoid tissues. These deer, which were among 516 animals collected during active CWD surveillance in March and April of 2002, previously had tested negative via brainstem examination. Ongoing studies suggest that among deer, examination of lymph nodes may offer a more sensitive technique than the currently standard method of brainstem testing. A total of 18 wild deer now have tested positive in adjacent portions of 2 counties (Iowa and Dane) just west of Madison.

Week-long deer hunts are scheduled in Wisconsin's CWD Eradication Zone each month from June–September, and extended hunting seasons will begin this autumn. Landowners and Department of Natural Resources marksmen killed 262 deer from June 8-14; CWD test results from these animals are pending. The goal of increased deer harvest is to reduce deer numbers as much as possible in the zone to prevent the spread of CWD throughout Wisconsin's vast and valuable wild deer herd. Other strategies to prevent CWD's spread in Wisconsin include a recently enacted statewide ban on feeding and baiting of deer. Feeding and baiting cause unnatural congregations of wild animals which enhances transmission of infectious diseases, including CWD. Additional information on CWD in Wisconsin may be found at www.dnr.state.wi.us.

In Colorado, CWD has been detected in additional wild and captive cervids. CWD was

confirmed south of Boulder for the first time in wild deer. A single deer, which was found dead last winter in a yard in Game Management Unit 38, just to the west and north of Denver, apparently died from the disease. Among captive elk, CWD recently was found in three herds in northeastern Colorado where CWD is endemic in wild cervids. Owners of all but two captive elk facilities in the endemic area accepted an offer by USDA and the Colorado Department of Agriculture to buy them out during the spring and summer of 2002. During the depopulation and testing of approximately 1,345 elk owned by program participants, 2 additional herds among 16 were found to contain CWD-positive elk, and CWD recently was confirmed in captive elk at one of the two ranches that did not participate in the program. Additional information on CWD in Colorado's captive elk may be found at www.ag.state.co.us.

The Colorado Wildlife Commission recently enacted carcass transportation regulations in order to prevent the potential spread of CWD. It is now unlawful to remove intact carcasses of hunter-killed deer and elk from Colorado's northeast CWD endemic area. However, the following portions of such carcasses may be removed from the 18 game management units comprising the endemic area: meat that is cut and wrapped (either commercially or privately); quarters or other portions of meat with no part of the spinal column or head attached; meat that has been boned out; hides with no heads attached; skull plates with antlers or antlers alone with no tissue attached; upper canine teeth, also known as "buglers," "whistlers," or "ivories"; and finished taxidermy heads. New Colorado regulations also prohibit the importation of dead deer or elk from any specific area of the United States or other country in which there has been a diagnosis of CWD in the wild, with the same exceptions noted above. Additional information, including the actual regulations, may be found at www.wildlife.state.co.us.

In Washington, D.C., a joint task force, co-chaired by the Administrator of USDA's Animal and Plant Health Inspection Service and the Director of the U.S. Fish and Wildlife Service, developed a *Plan for Assisting States, Federal Agencies, and Tribes in Managing Chronic Wasting Disease in Wild and Captive Cervids*. SCWDS personnel served on the Task Force and Working Groups that addressed issues such as CWD diagnostics, research, surveillance, management, information, and communications. The strategic plan was finalized in late June and presented to interested members of the U.S. Congress. Federal appropriations are being sought for implementation of the plan, which may be viewed at www.aphis.usda.gov/oa/cwd/newsoper.html.

For a complete review of CWD and other transmissible spongiform encephalopathies, see the April 2002 issue of the SCWDS BRIEFS, Vol. 18, No. 1, at www.scwds.org. (Prepared by John Fischer)

Low Pathogenicity AI in Virginia

An outbreak of low pathogenicity avian influenza (LPAI) virus apparently has been contained in domestic poultry in the Shenandoah Valley of Virginia. Disease control operations have been handled primarily by the Virginia Department of Agriculture and Consumer Services (VDACS), with assistance from the Regional Emergency Animal Disease Eradication Organization (READEO) of USDA's Animal and Plant Health Inspection Service (APHIS). Active surveillance continues for the H7N2 virus; however, no new cases have been diagnosed since July 2, 2002. Approximately 4.7 million commercial birds, primarily domestic turkeys, have been depopulated on the 197 Virginia poultry farms where the virus has been detected since March 2002. Small outbreaks of LPAI H7N2 also occurred and were contained in Pennsylvania, North Carolina, and West Virginia during 2001 and 2002. Eradication efforts have been aimed at protecting domestic poultry by eliminating the virus before it spreads to additional facilities

and by preventing mutation of this LPAI to a highly pathogenic form.

An outbreak of highly pathogenic H5N2 AI (HPAI) occurred in Pennsylvania, New Jersey, and Virginia in 1983-1984. Eradication of the disease costs more than $60 million and resulted in the destruction of more than 17 million domestic birds. The domestic poultry industry in Virginia currently adds approximately $745 million annually to the state's economy.

SCWDS personnel participated in evaluating the potential for wild bird involvement in the outbreak, including consultations regarding possible exposure of wild turkeys and bald eagles to treated poultry litter and carcasses. Wild bird surveillance was conducted with assistance from SCWDS, the Virginia Department of Game and Inland Fisheries, APHIS' Veterinary Services and Wildlife Services, and the U.S. Army. Samples from 467 Canada geese from the Shenandoah Valley and eastern Virginia were tested by VDACS and USDA's National Veterinary Services Laboratories. Although antibodies against AI viruses were detected among the geese, antibodies specific for the H7N2 serotype associated with the current outbreak in domestic poultry were not found. Results of virus isolation tests are pending.

Wild birds often are suggested as AI virus reservoirs and as sources of infection for domestic poultry. Although AI viruses are not uncommon among wild aquatic birds, the serotypes almost invariably differ from the highly pathogenic viruses associated with domestic poultry outbreaks. Natural AI infections are most common among waterfowl and shorebirds, although AI viruses also have been isolated from peridomestic species including, house sparrows, European starlings, house finches, and pigeons. During the 1983-1984 outbreak of HPAI (H5N2), wildlife surveillance conducted by SCWDS and other agencies yielded 187 virus isolates from 6,509 wild birds and rodents in the affected area. However, only one H5N2 isolate was found in a wild duck, and this isolate was genetically distinct from the H5N2 serotype involved in the outbreak.

Influenza viruses are found in domestic and wild avian species, humans, swine, horses, and marine mammals, including whales and seals. The different strains of influenza viruses are classified serologically based on two viral surface proteins, hemagglutinin (HA) and neuraminidase (NA). Presently 15 HA subtypes and 9 NA subtypes are recognized. Low pathogenicity AI viruses are not a threat to human health, and they do not affect the quality or safety of poultry products. Infections with LPAI are not reportable to animal health authorities, but state and federal agriculture agencies must be informed when HPAI infections occur. Infections with highly pathogenic AI viruses may result in significant poultry mortality, as well as international trade restrictions. (Prepared by Rick Gerhold and Joseph Corn)

West Nile Virus Continues to Spread

West Nile virus (WNV) was first recognized in Africa in the 1940s and since has been diagnosed in numerous locations in Africa, Asia, and Europe and, more recently, North America. The virus is spread by biting arthropods such as mosquitos and affects many species of birds and some mammals, primarily horses. For a comprehensive and concise review of the disease, see SCWDS BRIEFS Vol. 15, No. 3 or the CDC website at www.cdc.gov.

WNV was first diagnosed in humans in the United States in 1999 in the New York City area. Seven people died and more than 50 were hospitalized with viral encephalitis that year. WNV also has affected horses and numerous species of wild birds. Since its appearance in the United States, WNV has spread steadily southward and westward while the number of cases has increased dramatically. In 2000, WNV activity was reported from 138 counties in 12 states and the District of Columbia; in

2001, activity was reported from 359 counties in 27 states, the District of Columbia, and Ontario, Canada.

So far in 2002, West Nile virus has been documented in 26 states, the District of Columbia, and Ontario, Canada. Virus first was detected this year in a wild bird in Florida in January, indicating that year-round transmission is occurring in that state. In other states, the earliest documented cases were reported in April. States reporting WNV activity through July 28, 2002, are AL, CT, FL, GA, IL, IN, KY, LA, MA, MD, MI, MN, NC, ND, NE, NJ, NY, OH, OK, PA, RI, SD, TN, TX, VA, and WV. The finding of WNV-infected birds for the first time in North Dakota, Oklahoma, and Texas indicates that the virus continues to move westward.

SCWDS continues to conduct WNV surveillance among wild birds and mosquitoes in Georgia in collaboration with the Georgia Department of Human Resources' Division of Public Health. Between January 1 and June 30, 2002, 473 mosquito pools and tissue samples from 451 dead birds submitted by Georgia county health departments were tested for WNV. Since May 15, WNV has been isolated from four birds, all in the metro-Atlanta area, but not from any mosquito pools. SCWDS also analyzed tissues from 80 individual wild birds and mammals submitted by member states to our diagnostic service; however, WNV was not isolated from any of these cases.

The 2001 nationwide data now available reflect the continued spread of WNV as well as enhanced surveillance for the virus in many states. Nearly 35,000 dead wild birds, including almost 10,000 crows, were tested last year. WNV was found in 53% of the crows and in 9% of all other birds tested. Dead crows were the first indicators of WNV activity in 66% of the 359 counties reporting the virus. Approximately 1.4 million mosquitoes were tested for WNV in 2001, with virus detected in 919 mosquito pools (27 species) from 71 counties in 16 states. Two species, *Culex pipiens* and *C. restuans*, accounted for 59% of WNV-positive mosquito pools. There were 66 human cases of WNV infection reported from 10 states through December 2001, and the first human case was preceded by at least 1 report of a WNV-infected bird, sentinel animal, horse, or mosquito pool in more than 90% of the counties reporting human cases. These findings re-emphasize the need for continued public education, increased WNV surveillance aimed at early viral detection, and sustained, integrated mosquito control activities.

As we enter our 4th year of known WNV transmission in the United States, we must acknowledge that WNV has become permanently established in temperate regions of North America and that it will continue to spread. CDC suggests that increased surveillance geared toward early viral detection, mosquito-control, and avoidance activities that interrupt amplification cycles will potentially decrease the risk for human and domestic animal infection with WNV. Prevention activities, as stated by CDC, should continue to include (1) public education programs urging reduction of mosquito breeding sites around residential areas and personal protective measures to reduce mosquito exposure; (2) development of sustained, community-level integrated mosquito-surveillance and management programs; and (3) high-priority emphasis on the control of urban *Culex* mosquitoes. (Prepared by Danny Mead)

Update on *Cryptosporidium* and *Giardia* in Wildlife

Cryptosporidium and *Giardia* are protozoan parasites that can infect a wide range of vertebrate hosts, including humans, domestic animals, and wildlife. Asymptomatic infections are common, although severe persistent diarrhea may occur for approximately 1-3 weeks. Transmission is via the fecal-oral route. Infective oocysts of *Cryptosporidium* and infective cysts of *Giardia* may be transmitted directly, as well as through contamination of food and/or water. Traditionally, infection with

either parasite is initially diagnosed by finding microscopic oocysts or cysts in the feces. Recently, a rapid fecal screen utilizing a fluorescent antibody (FA) assay has become available.

Multiple species of *Cryptosporidium* and *Giardia* are known to exist, and there is debate about taxonomy of members of both genera, but *C. parvum* and *G. intestinalis* (=*lamblia*) are the species most commonly responsible for human disease. Additional host-adapted species continue to be discovered, and various host-adapted genotypes exist within certain *Cryptosporidium* species. The extent to which other genotypes and species of *Cryptosporidium* and *Giardia* infect humans is still in question. Thus, identification of oocysts and cysts to species and genotype level is of public health importance. Techniques such as polymerase chain reaction (PCR) allow for molecular identification of oocysts and cysts, and novel gene targets for diagnostic PCR assay and subsequent genetic sequencing allows for genotype differentiation.

Public awareness of these pathogens has grown as a result of the increased number of human outbreaks linked to contamination of public water supplies, including the 1993 cryptosporidiosis outbreak in Milwaukee, Wisconsin, that affected more than 400,000 people. Molecular typing has shown that two genotypes are responsible for such waterborne cryptosporidiosis outbreaks: genotype 1, isolated from humans almost exclusively; and genotype 2, isolated from humans, livestock such as cattle, sheep, and goats, and some wild animals.

In the past, wildlife species such as white-tailed deer have been implicated in the epidemiology of human disease, often without substantial evidence (see SCWDS BRIEFS Vol. 10, No. 2). Oocysts of *Cryptosporidium* sp. and cysts of *Giardia* sp. have been identified in free-ranging white-tailed deer from Virginia and Mississippi. More recently, molecular techniques have enabled researchers to identify a specific cervine genotype of *C. parvum*. Retrospectively, this cervine genotype has been found in reservoirs that received a great amount of storm runoff, and it has been associated with a small number of human cryptospoidiosis cases.

Wild birds also have been suggested as sources of human infection for *Cryptosporidium* and *Giardia.* In a survey of feces obtained from Chesapeake Bay Canada geese, seven of nine sites sampled were positive for *Cryptosporidium* sp., and all sites were positive for *Giardia* sp. The *Cryptosporidium* sp. oocysts collected from these geese were infectious to mice and were characterized as a zoonotic genotype via PCR assay, therefore it was concluded that waterfowl potentially could disseminate infectious *C. parvum* oocysts in the environment. In a recently published survey of hunter-harvested ducks along the Rio Grande River Valley, 59% of duck fecal samples were positive for either *Cryptosporidium* or *Giardia* and 14% were positive for both parasites by FA testing. However, none of the samples tested positive when assayed via PCR specific for *Cryptosporidium parvum* and *Giardia intestinalis*. The authors concluded that ducks sampled did not appear to be carrying the parasites most commonly associated with human disease.

As human outbreaks with these parasites continue to occur and public awareness increases, future research may focus on identifying additional wildlife species susceptible to infection with both *Cryptosporidium* and *Giardia.* Additionally, molecular characterization of new isolates could lead to the discovery of additional species and/or strains, further increasing our understanding of these protozoan pathogens and their epidemiology in humans, domestic animals, and wildlife. (Prepared by Cynthia Tate and Vivien Dugan)

HD Viral Persistence

The accurate diagnosis of epizootic hemorrhagic disease virus (EHDV) in white-tailed deer is

crucial to understanding the epidemiology of these viruses and their potential impact on deer populations. At present, a confirmatory diagnosis is dependent on virus isolation from fresh tissue or blood samples. SCWDS receives approximately 50 submissions yearly from southeastern wildlife biologists and veterinarians requesting EHDV and bluetongue virus isolations.

Fresh spleen and lung samples should be double-wrapped in clean plastic bags, packed in a cooler with "blue ice" packs, and shipped to SCWDS via overnight courier within 24 hours. If the samples cannot be shipped immediately, they should be kept on ice or in a refrigerator and sent as soon as possible. **Do not freeze the tissues**.

Most samples are shipped properly and the tissues arrive chilled. However, approximately 5% to 10% of the submissions have undergone unfavorable storage conditions or have originated from animals in more advanced stages of decomposition. In 2000, for example, five shipments arrived 5 or more days after necropsy, another arrived warm after being shipped by 3-day mail, another arrived frozen, and one shipment arrived after being refrigerated for 10 days. No viruses were isolated from any of these samples. It is unknown what effect, if any, the storage conditions had on the lack of virus recovery.

In an effort to determine the effect common shipping conditions have on virus recovery, we evaluated virus recovery from tissue samples from three known infected deer stored under differing storage conditions. Spleen and lung tissues were recovered within 3 hours after death from immature white-tailed deer infected with EHDV serotype 1. Each tissue sample was divided into 11 similar-sized sections. Samples were then stored at 4°C or 27°C. Virus titrations for each sample were performed on the initial day of sampling and on days 2, 4, 6, and 8.

Samples refrigerated at 4°C contained the same amount of EHD virus after 8 days as the original samples on day zero. A decrease in virus titer was observed in the samples held at 27°C, but it did not appear to be significant as virus still could be detected after 8 days. This finding reinforces our recommendations that samples should be stored at refrigerator temperature (4°C) prior to and during shipping. At this temperature these viruses are very stable, and we have isolated EHDV from blood samples stored at 4°C for more than 14 years (see *SCWDS BRIEFS* Vol. 11, No. 2).

While we do not recommend subjecting tissue samples to undesirable shipment conditions, these results indicate that virus isolation from such samples is possible. However, a negative result from a "problem" sample must be interpreted with caution as even a minor loss in viral titer can potentially impact our ability to detect virus. (Prepared by Britta Hanson)

Support for SCWDS

The Southeastern Wildlife Health Development Fund, which provides financial support to SCWDS, is supported by donations from individuals and organizations that believe wildlife health is a measure of environmental quality. The Fund recently has received donations from a new benefactor, as well as from some old friends. In June of 2002, the Disney Wildlife Conservation Fund made a contribution to help support SCWDS' continuing investigations of avian vacuolar myelinopathy (AVM). This fatal neurological disease has caused the deaths of at least 90 bald eagles in Arkansas, Georgia, North Carolina, and South Carolina since 1994. AVM also has been documented in coots, ducks, geese, owls, and other birds; however, its cause remains unknown despite extensive diagnostic and research investigations.

The recent contribution of the Disney Wildlife Conservation Fund marks the first time this organization has assisted SCWDS in its efforts to identify the cause of AVM and its impact on

bald eagles and other wild birds. The Disney Wildlife Conservation Fund was established in 1995 by Walt Disney Attractions to promote and enable global wildlife conservation through partnerships with qualified scientists, educators, and organizations committed to preserving biodiversity.

The Arcadia Wildlife Preserve, Inc. has been a regular supporter of SCWDS wildlife health research projects for several years. They recently contributed to our projects regarding AVM in bald eagles and in the past helped sponsor our investigations of the causes of morbidity and mortality among endangered Key deer. Arcadia Wildlife Preserve, Inc. is a non-profit organization dedicated to long-term enhancement of free-ranging wildlife populations and the re-establishment of diminishing native species.

Another long-time supporter and friend of SCWDS is the Camp-Younts Foundation, established by the Camp family of Franklin, Virginia, in the 1950s. The Camp-Younts Foundation provides scholarships and support for persons and programs in its home community in Southampton County, Virginia, and supports projects associated with education, religion, public safety, health, natural resource conservation, and community activities that help the needy.

SCWDS is extremely grateful for these contributions. Please consider supporting SCWDS by making a gift or by providing information about SCWDS and our commitment to wildlife health to individuals and organizations that support wildlife conservation projects. To learn more about the Southeastern Wildlife Health Development Fund, please visit the SCWDS website at www.scwds.org or contact our Director, Dr. John Fischer, at 706-542-1741.

Meeting Announcement

A 2-day symposium called Brucellosis in the Greater Yellowstone Area will be held at the Snow King Resort in Jackson, Wyoming, September 17-18, 2002. Symposium speakers will discuss all aspects of brucellosis in elk and bison, including past, current, and future research and management activities. Government and non-government agencies will also present their perspectives on this issue. Registration before August 15 is $75, afterwards $100, and includes a copy of the proceedings. For further information and registration materials contact Becky Russell, Wyoming Game and Fish Department, phone 307-766-5616, or email rrussell@uwyo.edu.

New CWD Video Available

A new 30-minute VHS videotape on chronic wasting disease (CWD) in captive and free-ranging deer and elk is now available from the Wyoming Game and Fish Department. Completed in the summer of 2002, this updated version of the agency's 1998 video contains current information on CWD's history, clinical signs, geographical distribution, and diagnostic testing. Recommendations for handling deer and elk carcasses are included for hunters and wildlife professionals, and there is a detailed demonstration of the proper collection and handling of brainstem, lymph node, and tonsil samples for CWD testing.

The cost of the video is $20 (plus $4 for shipping), and it can be purchased by using the order form below or by contacting the Wyoming Game and Fish Department at 1-800-548-9453. (Prepared by Rick Gerhold)

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CHRONIC WASTING DISEASE (CWD) VIDEO

CWD VIDEO _____ Copies @ $20/ea _____

Shipping & Handling** _____

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**$4 FOR 1 VIDEO; $5 FOR 2 VIDEOS; $6 FOR 3 OR MORE VIDEOS

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Recent SCWDS Publications Available

Below are some recent publications authored or co-authored by SCWDS staff. If you would like to have a copy of any of these papers, fill out the request form and return it to us.

____Baumann, C.D., W.R. Davidson, D.E. Roscoe, and K. Beheler-Amass. 2001. Intracranial abscessation syndrome in white-tailed deer of North America. *Journal of Wildlife Diseases*. 37(4): 661-670.

____Dinkins, M.B., D.E. Stallknecht, E.W. Howerth, and B.G. Brackett. 2001. Photosensitive chemical and laser light treatments decrease epizootic hemorrhagic disease virus associated with *in vitro* produced bovine embryos. *Theriogenology* 55(8): 1639-1655.

____Gaydos, J.K., A.B. Allison, B.A. Hanson, and A.S. Yellin. 2002. Oral and fecal shedding of epizootic hemorrhagic disease virus, Serotype 1 from experimentally infected white-tailed deer. *Journal of Wildlife Diseases* 38(1): 166-168.

____Gaydos, J.K., D.E. Stallknecht, D.M. Kavanaugh, R.J. Olson, and E.R. Fuchs. 2002. The dynamics of maternal antibodies to hemorrhagic disease viruses (*Reoviridae: Orbivirus*) in white-tailed deer. *Journal of Wildlife Diseases* 38(2): 253-257.

____Hanson, B.A. 2001. Shorebirds and avian influenza viruses. *International Wader Study, Group Bulletin* 96: 4.

____Lewis, L.A., R.J. Poppenga, W.R. Davidson, J.R. Fischer, and K.A. Morgan. 2001. Lead toxicosis and trace element levels in wild birds and mammals at a firearms training facility. *Archives of Environmental Contamination and Toxicology* 41: 208-214.

____Linhart, S.B., J.C. Wlodkowski, D.M. Kavanaugh, L. Motes-Kreimeyer, A.J. Montoney, R.B. Chipman, D. Slate, L.J. Bigler, and M.G. Fearneyhough. 2002. A new flavor-coated sachet bait for delivering oral rabies vaccine to raccoons and coyotes. *Journal of Wildlife Diseases* 38(2): 363-377.

____Noon, T.H., S.L. Wesche, D. Cagle, D.G. Mead, E.J. Bicknell, G.A. Bradley, S. Riplog-Peterson, D. Edsall, and C. Reggiardo. 2002. Hemorrhagic Disease in Bighorn sheep in Arizona. *Journal of Wildlife Diseases* 38(1): 172-176.

____Stallknecht, D.E. 2001. VSV-NJ on Ossabaw Island Georgia: The truth is out there. *Proceedings of the Society of Tropical Veterinary Medicine*, New York Academy of Sciences 916: 431-436.

____Stallknecht, D.E., D.E. Perzak, L.D. Bauer, M.D. Murphy, and E.W. Howerth. 2001. Contact transmission of vesicular stomatitis virus New Jersey in pigs. *American Journal of Veterinary Research* 62(4): 516-520.

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

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