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WWW.SCWDS.ORG

SCWDS has obtained its own web site domain...SCWDS.ORG, which will provide a user-friendly way to rapidly obtain information. Through the efforts of SCWDS Research Technician Bob Longe, a tremendous amount of information has been installed for use by SCWDS cooperators, wildlife conservationists, and the public. For example, all previous articles published in the newsletter, SCWDS BRIEFS, are available. The most recent issues of the BRIEFS are presented in their entirety under the heading "SCWDS BRIEFS." Earlier articles are accessible by going to the "Topics" section and searching by key words. There is a section on how diagnostic cases should be submitted, along with a printable submission form; there also is an order form for the Field Manual of Wildlife Diseases in the Southeastern United States. Directions to the SCWDS Wildlife Health Building, e-mail addresses for SCWDS personnel, lists of publications, information about educational opportunities, and other material are there as well. Please give our web page a visit and let us know what you think. (Prepared by Vic Nettles)

HD Virus Cross-Immunity

Clinical hemorrhagic disease (HD) in white-tailed deer is caused by orbiviruses in two serogroups, epizootic hemorrhagic disease (EHD) and bluetongue (BT). Both serogroups have multiple serotypes; two serotypes of EHD virus (EHDV-1 and -2) and four serotypes of BT virus (BTV-10, -11, -13, and -17) have been associated with disease in deer. Not all deer infected with EHDV or BTV will die; this is known because many normal deer have antibodies that indicate prior exposure to various HD viruses. We are confident that deer that recover develop immunity to the specific virus serotype that protects against reinfection by the same virus. However, it is not known how well this immunity cross-protects deer against other HD viruses. When deer survive infection with a virus from one serogroup (EHDV or BTV), there is good evidence to indicate they are not protected from disease caused by subsequent infection with a virus from the opposite serogroup. For example, research performed by SCWDS demonstrated that deer infected with EHDV-2 were not protected when later infected with BTV-10. It is less certain how well recovery from one virus within a serogroup cross-protects against other serotypes of HD viruses within the same serogroup.

Since 1989, the majority of virus isolations made by SCWDS from clinically ill white-tailed deer have been EHDV-2. However, last fall EHDV-1 was the predominant virus isolated from dead deer in the eastern United States (see SCWDS BRIEFS, Vol. 15, No. 3). The potential for cross-immunity among viruses exists when there is a change in the predominant HD virus or where there are multiple HD viruses in the same herd. Understanding the extent of HD virus cross-immunity will help wildlife managers better understand how deer herd immune status relates to severity and timing of HD die-offs.

At SCWDS, an experiment was conducted to
determine if deer that survived infection with EHDV-2 would be protected against EHDV-1. Penned white-tailed deer were infected with EHDV-2 virus from a prior epizootic and monitored for disease using an extensive array of physical evaluations, blood counts, blood clotting times, virus isolations, and antibody titrations. Clinical disease caused by the initial infection with EHDV-2 varied from mild to severe, but all deer recovered. After recovery, deer were challenged with an isolate of EHDV-1 from the 1999 outbreak and monitored as before. Unexposed control deer developed severe HD and died when infected with EHDV-1, but clinical disease was absent or mild in deer that were previously exposed to EHDV-2. In spite of the mild clinical effect of the EHDV-1 challenge on the EHDV-2 protected deer, they developed relatively high levels of virus in their blood. This suggests that deer that survive EHDV-2 will be protected against sub-sequent EHDV-1 infection but may still serve as virus amplifying hosts. (Prepared by Joe Gaydos and David Stallknecht)

Did West Nile Virus Survive the Winter?

Last summer an outbreak of encephalitis caused by West Nile Virus (WNV) occurred in people in the New York City area and resulted in 61 cases of severe disease, including 7 deaths (see SCWDS BRIEFS, Vol. 15, No. 3). This was the first documented occurrence of this disease in the Western Hemisphere; however, WNV has caused human illness in southern Europe, the Middle East, southeast Asia, and Africa since its discovery over 60 years ago in Uganda.

West Nile Virus is an arthropod-borne virus (arbovirus) transmitted by mosquitoes and is closely related to St. Louis encephalitis virus, a native arbovirus that occurs across the United States. Like most other arboviruses, the transmission cycle of WNV involves infection of vertebrate amplifying hosts, which circulate virus in their blood and serve as a source of virus for mosquitoes. Although a variety of wild and domestic vertebrates can be infected, birds are particularly important amplifying hosts for WNV. During last year’s outbreak, the virus also caused deaths among horses and a variety of wild and captive birds, notably crows and birds housed at the Bronx Zoo. How WNV got to the United States is unknown, but public health officials were keen to learn whether it had come and gone or whether it might reappear this year. The answer may be the latter, based on a report by the Centers for Disease Control and Prevention (CDC) which describes the isolation of West Nile Virus from a pool of overwintering mosquitoes collected at Fort Totten, New York, during January-February 2000 (CDC Morbidity & Mortality Weekly Report, Vol. 49, No.10).

Since it is likely that many questions will arise again this year regarding WNV, accurate sources of information will be important to those trying to respond to inquiries. An especially useful source of information designed to answer basic questions about WNV and other arboviruses is the CDC’s web site on Arboviral Encephalitides at www.cdc.gov/ncidod/dvbid/arbo/arboinfo.htm (Prepared by Randy Davidson)

West Nile Virus Surveillance-2000

A surveillance system for West Nile Virus (WNV) coordinated by the Centers for Disease Control and Prevention has been implemented to include priority states that were affected by last year’s WNV outbreak or have a high potential for being affected because of bird migration patterns. These include states along the Atlantic and Gulf coasts from Massachusetts to Texas. Surveillance activities are being managed at the state level, depending upon specific needs and expertise. However, surveillance strategies in all states include components of monitoring mosquitoes, birds (sentinel chickens and free-flying birds), horses, and human beings.

SCWDS currently is cooperating with the Georgia Department of Human Resources’ Division of Public Health to handle the wild bird surveillance in Georgia, which will include
testing free-flying birds for the virus and antibodies to the virus. A second part of this work involves dead bird surveillance; last year’s outbreak clearly demonstrated that bird mortality can occur. Although several species were affected, American crows appeared most susceptible. Based on past mortality and the fact that WNV can be readily isolated from affected birds, dead bird surveillance may provide a sensitive means for documenting viral spread should it occur. If dead birds, especially crows, are encountered in your state, please discuss this with authorities at your state public health department. Each state is developing a protocol to handle such submissions due to the potential for human disease and the importance of these samples to surveillance efforts. (Prepared by David Stallknecht)

Calicivirus Hits Iowa Rabbitry

Rabbit calicivirus disease (RCD), also known as viral hemorrhagic disease of rabbits, was recognized for the first time in the United States in early March 2000 when this highly contagious disease killed 25 of 27 domestic rabbits at a farm in rural Iowa. The remaining two rabbits were euthanized, the premise was quarantined, and RCD has not been detected elsewhere in the United States. Rabbit owners and veterinarians throughout the country have been encouraged to report all incidents of excess acute mortality of unknown cause to animal health authorities.

Rabbit calicivirus affects only the European rabbit, Oryctolagus cuniculus, the species to which all pet and commercial rabbits in the United States belong. Rabbits native to North America, such as cottontails (Sylvilagus sp.) and jackrabbits (Lepus sp.), are not considered susceptible to rabbit calicivirus, and the virus is not pathogenic for human beings or other mammals. European rabbits with RCD die with hemorrhagic and necrotic lesions in the liver, intestine, and lymphoid tissues within 6-24 hours of the onset of fever.

Rabbit calicivirus was first recognized as a cause of rabbit mortality in 1984 in the People’s Republic of China. Since 1984, RCD has been diagnosed in several countries in Asia and Europe. RCD made its first appearance in the Western Hemisphere in Mexico City in 1988; it subsequently was eradicated from Mexico. RCD also has occurred in New Zealand and Australia, where it spread rapidly through free-ranging European rabbits (SCWDS BRIEFS, Vol. 11, No. 4).

The source of the virus in the Iowa rabbitry remains unknown. There had been no new rabbits brought onto the farm for two years, and rabbits had not been taken off-site and returned since August 1999. Results of tests conducted on samples from the Iowa rabbits indicated that the virus was most similar to viral subtypes isolated from rabbits in Europe, and it was unlike rabbit caliciviruses found in Australia and New Zealand. The Iowa Department of Agriculture and Land Stewardship and the U.S. Department of Agriculture are continuing their investigations into this outbreak.

This article was prepared with information from USDA’s Veterinary Services, Animal and Plant Health Inspection Service. Additional information on rabbit calicivirus may be found at the USDA web site www.aphis.usda.gov under “Animal Emergency Information” and “Hot Issues.” (Prepared by John Fischer)

Screwworms

Screwworm fly (Cochliomyia hominovorax) larvae were recovered from a horse in Florida on March 2. The horse was imported from Argentina with 16 other horses on February 27 and was released from quarantine on March 1. Larvae submitted by a private practitioner, who noted a discharge and bad odor from the horse’s prepuce, were confirmed as screwworms by the National Veterinary Services Laboratories, USDA. Upon confirmation of the presence of this exotic pest, the Animal and Plant Health Inspection Service, USDA, implemented an emergency management plan. The horses and
premises were treated, and surveillance and epidemiologic investigations were conducted. Screwworms were not found on any of the other 16 horses in the original shipment.

The screwworm fly once was found throughout tropical and subtropical areas of the Americas. Currently, screwworms are found only in South America, Panama, and on some of the Caribbean islands. Screwworms have been eradicated from the United States (1966), Mexico (1991), and some countries in Central America. Screwworm eradication is achieved through the release of millions of sexually sterile flies in infested areas. After mating with the sterile males, female flies, which mate only once, produce infertile eggs.

The parasitic larvae of the screwworm fly cause significant damage to livestock and wildlife by feeding on living flesh, and screwworm infestations can lead to death in a matter of days if left untreated. The benefits of screwworm eradication for livestock producers in the United States alone are estimated at $500 million annually.

The screwworm was the most important arthropod pest of white-tailed deer in the southern United States prior to initiation of the eradication program in 1957. On coastal islands in Georgia and South Carolina, deer populations were reduced by as much as 75% in some years by screwworms. In south Texas, it was estimated that up to 80% of the annual fawn crop was lost during years of heavy infestation. Therefore, maintenance of screwworm-free status will continue to provide benefits to wildlife as well as livestock health. Wildlife biologists are encouraged to remain vigilant for screwworms and report the presence of fly larvae found in wounds of live animals. (Prepared by Joe Corn)

MCF Plot Thickens

Researchers with USDA-ARS, Washington State University, and North Dakota State University authored an article in the current issue of the Journal of Clinical Microbiology (April 2000, Vol. 38, No. 4, pp.1313-1318) reporting that a newly recognized herpesvirus was the cause of malignant catarrhal fever (MCF) in white-tailed deer in a small zoo in the northcentral United States. Five of six whitetails housed at the zoo died during January and February 1999. Clinical signs and histopathologic lesions were compatible with MCF which has been diagnosed previously among captive white-tailed deer on several occasions. Sophisticated laboratory procedures confirmed that the deer were infected with a newly recognized herpesvirus belonging to the MCF group of gammaherpesviruses.

Wildebeest and sheep are reservoir hosts for the two previously known pathogenic gammaherpesviruses that cause MCF. The wildebeest and sheep viruses are designated alcelphine herpesvirus 1 (AHV-1) and ovine herpesvirus 2 (OHV-2), respectively. These viruses usually cause asymptomatic infections in the reservoir hosts; however, they may cause serious disease in other species of domestic and wild ruminants, including at least 13 species of deer. The zoo where the whitetails died housed about 200 animals, including mule deer, Reeve’s muntjac deer, white-lipped deer, white-tailed deer, pygmy goats, domestic goats, unspecified species of antelope, and several species of nonruminant animals. The reservoir for the virus that killed the whitetails was not determined.

The authors point out that there is mounting evidence that strains of MCF viruses vary in virulence, but at least some of these viruses, including AHV-1, OHV-2, and the one that killed deer in this outbreak, can be highly pathogenic. Furthermore, the authors note that at least four other gammaherpesviruses currently considered to be MCF viruses are known to infect exotic ruminants (hartebeest, topi, roan antelope) and domestic goats, but currently these viruses have not been associated with any clinical illness. Finally, they note that it is unknown if virus involved in this outbreak is pathogenic for cattle, bison, or other ruminants.
This event illustrates two important points relative to health issues that should be considered when wild animals are maintained in captivity or translocated. These are: (1) mixing of reservoir and susceptible species is a prescription for epizootics among susceptible hosts, and (2) the existence of unknown pathogens, such as the MCF virus that killed the deer in this case, confounds assessment of health risks during animal relocation. (Prepared by Randy Davidson)

**SCWDS Cooperative Network**

A review of the SCWDS publications list provides insight into the breadth of the "networking" that occurs with other scientists during SCWDS research and service activities. Of more than 150 publications in the past 10 years, only about 29% were authored solely by SCWDS personnel. Authorship for the remaining 71% was shared with collaborators in 75 other state or federal agencies, universities, and private organizations. Fourteen co-authors were from foreign countries. SCWDS shared joint authorship with colleagues in six other departments in the College of Veterinary Medicine and collaborated on publications with individuals in seven other colleges within The University of Georgia.

The placement of the publications was as diverse as the authorship. There were over 45 journals, symposia, proceedings, books, and miscellaneous publications involved. This diversity of partnerships with other scientists is testimony to the complexity of wildlife health topics that must be addressed in wildlife health management. It is folly to think that one organization can "be all things to all people" in regard to capabilities for wildlife health investigations. There is a need to collaborate with the best experts available, and SCWDS provides an excellent core unit to facilitate the "cooperative approach" among scientists. We are extremely grateful to the many talented people who have worked with SCWDS, and we extend our appreciation to everyone who has contributed their time and knowledge to the goal of healthy wildlife. (Prepared by Vic Nettles)

**Personnel Changes**

There’s some good news and there’s some bad news. The bad news is that Dr. David Stallknecht has accepted a position as Assistant Professor in the Department of Medical Microbiology and Parasitology in UGA’s College of Veterinary Medicine.

The good news is that we aren’t really losing Dave. He will maintain his office, laboratory, and support staff here in our building and continue his ongoing research projects with SCWDS. Dave’s new responsibilities will include teaching courses on epidemiology and public health to veterinary students and graduate students. He also will continue to develop and expand an externally funded research program in veterinary epidemiology and direct graduate students. Dave is a brilliant, hardworking scientist and enjoys an international reputation among wildlife and agricultural animal health researchers as an expert on such diseases as vesicular stomatitis, epizootic hemorrhagic disease, bluetongue, and avian influenza. He will do well in his new position.

There’s more good news. We have a new Post Doctoral Associate, Dr. Daniel G. Mead. Danny will be working directly with Dave Stallknecht on research areas of mutual interest, including vesicular stomatitis, epizootic hemorrhagic disease, and mosquito-borne encephalitis. Danny comes to us from the University of Arizona where he earned degrees in Wildlife Biology (BS), Medical Entomology (MS), and Pathobiology (PhD). He currently is completing the requirements for a Master’s of Public Health degree. Danny is a valuable addition to our staff, and we welcome him. (Prepared by Gary Doster)
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.

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