

# SCWDS BRIEFS

A Quarterly Newsletter from the  
**Southeastern Cooperative Wildlife Disease Study**  
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## Unexplained Sea Bird Mortality

Since June 12, 2005, there have been numerous reports of dead or dying sea birds along the beaches of the eastern seaboard from Florida to Maryland. Birds found alive were severely depressed, unable to fly, walk, or swim, and all died within a few days of being found. More than 700 mortalities have been reported, with more than 50% of the reports coming from Florida. Nearly 30 birds from FL, GA, NC, SC, and VA have been examined at SCWDS and the USGS National Wildlife Health Center (NWHC). The specific cause of the mortality remains undetermined.

All of the examined birds were moderately to severely emaciated. The ingesta in the gastrointestinal tracts of the birds was highly variable, ranging from squid beaks, to squid meat and fish, to virtually empty. No other consistent abnormalities or lesions were apparent on gross necropsy or microscopic examination of tissues from all major organs. Results of diagnostic tests for infectious agents, including West Nile virus, avian influenza virus, and Newcastle disease, virus were negative. Tissues analyses for algal biotoxins associated with red tides also yielded negative results. Samples of liver, kidney, and brain tissues were tested for toxicological agents and were negative for organochlorine insecticides and heavy metal compounds.

Most of the birds involved in this mortality event belong to the Order Procellariiformes, which are pelagic birds that normally spend the majority of their lives in the open ocean and are rarely seen close to shore. Greater shearwaters represent over 65% of the mortality reports, with the remaining cases

composed of other species of shearwaters, boobies, gannets, gulls, loons, petrels, and terns.

Many of these birds migrate long distances in fly-ways located well off the east coast of the United States and Canada. Large amounts of energy are needed to complete this annual migration, and the birds usually are lean and lacking fat reserves by the time they arrive on the summer grounds. Small fluctuations in food resources (squid and small fish), poor weather, water temperature, and other factors can have significant effects on the success of the migration. It is possible that such environmental changes may have resulted in the emaciation and mortality of these sea birds, but at this time the role of ecologic fluctuations remains uncertain.

Reports of sea bird mortality in this event are presently declining. Continued examination and analysis of the birds and their environment are in progress. Anyone observing multiple sick or dead sea birds should notify state or federal fish and wildlife agency officials. (Prepared by Justin Brown with information from Emi Saito at NWHC)

## Deer as Hosts of Anaplasmosis

SCWDS recently completed one portion of a four-year study funded by the National Institutes of Health on the natural history of zoonotic tick-borne diseases caused by multiple species of *Anaplasma* and *Ehrlichia*. The completed project evaluated the susceptibility of white-tailed deer (WTD) to the rickettsia, *Anaplasma phagocytophilum*. *Anaplasma phagocytophilum* is the causative agent of human granulocytic anaplasmosis

(HGA), a potentially fatal disease characterized by organisms in granulocytic blood cells, notably neutrophils. In North America, prior to the recognition of HGA in 1994, *A. phagocytophilum* was of concern only to the veterinary community as a tick-borne pathogen of horses and dogs. After the recognition of *A. phagocytophilum* as a significant human pathogen, intensive efforts were undertaken to identify reservoirs of the organism in nature.

White-tailed deer are a critical host for the adult stages of tick vector, *Ixodes scapularis*. Although serologic and molecular evidence of *A. phagocytophilum* has been demonstrated in WTD, data on the course of infection of *A. phagocytophilum* in this species was not available. The serologic evidence of *A. phagocytophilum* in wild WTD could not be fully validated because antigenic cross-reactivity between *A. phagocytophilum* and other rickettsiae can occur, and WTD populations are known to be co-infected with multiple species. Likewise, molecular detection of *A. phagocytophilum* in WTD was confounded by the widespread presence of a genetically similar *Anaplasma* sp. known as "WTD-agent." Furthermore, molecular detection (PCR evidence) of *A. phagocytophilum* among wild WTD was limited to a single point in time per animal, further limiting interpretation of the data.

For these reasons, SCWDS evaluated the response of WTD to experimental infection with *A. phagocytophilum*. In the study, four deer were injected with a human isolate of *A. phagocytophilum* propagated in tick cells, and two additional deer served as negative controls. All four principal deer developed antibodies (titers  $\geq 64$ ) to *A. phagocytophilum*, as determined by the indirect fluorescent antibody test, between 14 and 24 days post-infection (DPI), and two deer maintained titers  $\geq 64$  through the end of the 66-day study. Although organisms were not observed in deer granulocytes and *A. phagocytophilum* was not re-isolated via tick cell culture of blood,

16S PCR test results indicated that *A. phagocytophilum* circulated in peripheral blood of three deer through at least 17 DPI and was present in two deer at 38 DPI. Femoral bone marrow from one deer was PCR positive for *A. phagocytophilum* at necropsy on 66 DPI. None of the deer exhibited clinical disease.

These data confirm that WTD are susceptible to infection with a human isolate of *A. phagocytophilum* and verify that WTD produce detectable antibodies upon exposure to the organism. The organism circulated in peripheral blood of the deer for a sufficient amount of time for hypothetical transmission to ticks to occur. However, the predominant life stage of *I. scapularis* found on deer is the adult, and adult *I. scapularis* do not transmit *A. phagocytophilum* transovarially. Therefore, it is unlikely that WTD are a significant source of *A. phagocytophilum* for immature ticks, even though deer have a high probability of natural infection. Despite the limited potential of WTD to serve as a reservoir host of *A. phagocytophilum*, their susceptibility and immunologic response to *A. phagocytophilum* render them suitable candidates as natural sentinels for this zoonotic tick-borne organism. This work will be published in the August 2005 issue of the *Journal of Clinical Microbiology*. (Prepared by Cynthia Tate)

## Vesicular Stomatitis Update

In the April 2005 issue of the SCWDS BRIEFS (Vol. 21, No. 1) we reported on the occurrence of vesicular stomatitis (VS) in livestock in the United States. This is the second consecutive year that VS has been confirmed in livestock in the western states. Vesicular stomatitis New Jersey virus (VSNJV) infection initially was confirmed in two horses on one premises in Grant County, New Mexico, in April 2005, and in one horse in Maricopa County, Arizona, in May 2005. Additional cases subsequently have been identified in these two states, as well as in Colorado, Texas, and Utah. To date, VSNJV infection in livestock has been confirmed on 27 premises in Arizona (5

counties), 8 premises in Colorado (5 counties), 12 premises in New Mexico (5 counties), 1 premises in Texas, and 12 premises in Utah (5 counties). USDA-APHIS-Veterinary Services and the state department of agriculture in each of the affected states continue to monitor the situation and conduct response activities in an effort to minimize the impacts of the outbreak.

Studies on VSNJV are being conducted at SCWDS, where scientists have a strong history of VSNJV research, with the long-range goal to better elucidate the epidemiology of the virus. In recent SCWDS studies, significant progress was made in understanding VSNJV transmission and the role of insects in the epidemiology of the virus. These studies were supported by a grant from the USDA's Cooperative State Research, Education, and Extension Service, National Research Initiative Competitive Grants Program (USDA-NRICGP). For a comprehensive and concise review of these studies, see SCWDS BRIEFS, Vol. 20, No. 2. Recently we have learned that USDA-NRICGP will fund a new a SCWDS grant titled "Transmissibility and Host Predilection of Epidemic Vesicular Stomatitis New Jersey Virus Strains." There are two major objectives of this research. The first is to determine the extent to which clinical outcome and patterns of virus shedding in VSNJV-infected cattle and horses are dependent on the virus strain and the inoculation route. The second objective is to define the potential for virus transmission by insects and by animal-to-animal contact in relation to livestock infection with epidemic VSNJV strains. (Prepared by Danny Mead)

### **Deadly Virus Kills Transplant Recipients**

In May 2005, the Centers for Disease Control and Prevention (CDC) reported that four transplant recipients had developed severe illnesses after receiving organs from a Rhode Island donor. Three of the four organ recipients, all of whom were taking immunosuppressive drugs to prevent organ rejection, died within a month after their

transplant. Lymphocytic choriomeningitis virus (LCMV) infection was confirmed in all four patients, making it the second time that organ transplant transmission of this virus has been recorded. In 2003, a cluster of deaths of patients in Wisconsin who had received organ transplants was associated with LCMV infection; however, the source of infection was not found.

Epidemiologic investigations were conducted by the CDC, the Massachusetts Department of Public Health, and the Rhode Island State Health Department to identify the source of the virus. It was found that a member of the donor's family recently had acquired a pet hamster, and LCMV subsequently was isolated from this animal. Serologic testing of the family revealed that the owner of the hamster was the only member of the household who carried antibodies against LCMV, and testing of available tissues from the donor failed to detect the virus. Molecular genetic testing is underway to compare the virus carried by the hamster with the virus isolates from the organ recipients.

The house mouse (*Mus musculus*) is the primary reservoir of LCMV and both have world-wide distribution. Although rates vary by location, it is estimated that 5% of house mice in the United States carry the virus. Other animals susceptible to LCMV infection include hamsters and guinea pigs. Once infected, rodent reservoirs carry and shed LCMV for life without showing clinical signs. Transmission to humans occurs primarily through aerosol or direct exposure to secretions or excretions of infected animals. Although LCMV infection in humans can occur at any time of the year, it is more common in the winter months when reservoir hosts are more likely to move indoors. Most human infections are acquired from house mice, but infections also have been linked to laboratory animals and to pet rodents that acquired LCMV from wild rodents at the breeder, pet store, or in the home. A large outbreak of LCMV in 1975 that affected 181 people in 12 states was associated with

pet hamsters sold by a single distributor. Other human infections, including tularemia and multi-drug-resistant salmonellosis, recently have been associated with handling pet rodents, including hamsters.

Lymphocytic choriomeningitis virus infection in immunocompetent humans usually is asymptomatic or causes mild self-limiting illness. The virus can cause aseptic meningitis, but fatal cases are rare. Human-to-human transmission has not been reported, with the exceptions of organ transplantation and vertical transmission of the virus from mother to fetus during pregnancy. Infection of the fetus during the first or second trimesters of pregnancy may cause severe fetal illness and miscarriage or ocular and brain deformities of the fetus.

Preventive measures should be directed toward eliminating contact with house mice and practicing good hygiene when handling pet rodents. More information on this outbreak, LCMV, and preventive measures can be found at the websites of the CDC ([www.cdc.gov](http://www.cdc.gov)) and the Rhode Island State Health Department ([//www.health.state.ri.us/media/050523b.php](http://www.health.state.ri.us/media/050523b.php)). (Prepared by Katy Tolbert)

## **Rabbit Hemorrhagic Disease in Indiana**

Rabbit hemorrhagic disease (RHD) recently was identified in the United States for the fourth time. In this occurrence, approximately 100 rabbits died at a backyard rabbitry in Vanderburgh County, Indiana. In late May 2005, 8 of 11 rabbits that were purchased at a flea market in Kentucky died 3 days after being introduced into the Indiana rabbitry. Subsequently, nearly half of the 200 rabbits at the location died, and a foreign animal disease investigation was conducted by the Indiana Board of Animal Health and USDA-APHIS-Veterinary Services. The diagnosis of RHD, which is considered a foreign animal disease in the United States, was confirmed by

Veterinary Services' Plum Island Foreign Animal Disease Laboratory. No additional outbreaks have been identified. Previous outbreaks of RHD occurred in 2000 in Iowa (SCWDS BRIEFS, Vol 16, No. 1), and in 2001 in Illinois, New York, and Utah.

Rabbit hemorrhagic disease, also known as rabbit calicivirus disease and viral hemorrhagic disease of rabbits, was first recognized in China in 1984 and now is endemic in many parts of the world, including much of Europe and Asia, as well as Australia, New Zealand, and Cuba. The disease affects only the European rabbit (*Oryctolagus cuniculus*), which includes all pet and commercial rabbits in the United States. Rabbits native to North America, such as cottontails (*Sylvilagus*) and jackrabbits (*Lepus*), are not susceptible to RHD, nor are other mammals, including humans.

The disease is highly contagious and can be spread by many routes. The most frequent are direct contact with infected rabbits or their excreta; rabbit products, such as meat, skin, and offal; insects and rodents that serve as mechanical vectors; and fomites, such as cages, feeders, and clothing.

Disease may be severe, with up to 90% mortality among affected rabbits. The incubation period is short, and rabbits frequently are found dead without clinical signs from 1-3 days after exposure. European rabbits with RHD may have hemorrhages from body orifices, as well as necrotic and hemorrhagic lesions in the liver, intestines, and lymph nodes. Rabbits that survive infection may become carriers and spread RHD to other rabbits.

Because of its highly infectious nature and its status as a foreign animal disease, RHD should be reported to state and federal animal health officials whenever it is suspected. If animals exhibit sudden death or clinical signs of depression, foamy nasal discharge, and/or neurological dysfunction, the owner should

contact a veterinarian. Suspect animals should not be moved from the site to other locations where other rabbits are present and they should be prevented from having direct contact with healthy rabbits. Healthy rabbits should not be exposed to possibly contaminated cages, feeders, or bedding, and new rabbits should not be introduced to any affected facility.

Additional information on this outbreak and on RHD can be found at the websites of USDA-APHIS ([www.usda.aphis.gov](http://www.usda.aphis.gov)) and the Indiana Board of Animal Health ([www.in.gov/serv/presscal?PF=aiin&Clist=17&Elist=83886](http://www.in.gov/serv/presscal?PF=aiin&Clist=17&Elist=83886)). (Prepared by John Fischer)

### **Award & Fund Established to Honor Tom Thorne and Beth Williams**

The Wildlife Disease Association (WDA) and the American Association of Wildlife Veterinarians (AAWV) have established a memorial fund honoring Drs. E. Tom Thorne and Elizabeth S. Williams. The fund will be used to endow an award named for the husband and wife team and will be given "in acknowledgment of an exemplary contribution either combining wildlife disease research with wildlife management policy implementation, or elucidating particularly significant problems in wildlife health." Tom and Beth died in an automobile accident on December 29, 2004. Their contributions to wildlife health research and wildlife veterinary medicine are legendary.

Tom recently had retired from the Wyoming Game and Fish Department, having risen through the ranks from Wildlife Veterinarian, to Branch Chief, to Interim Director. He was one of the nation's leading wildlife veterinarians for over 30 years, a co-founder and officer of AAWV and the Wyoming Wildlife Society, and served both as president. His work on brucellosis in wildlife, diseases of wild sheep, conservation of black-footed ferrets and other sensitive species, and his efforts to develop communication and collaboration between

livestock and wildlife interests, brought him national acclaim and many awards .

Beth was a professor at the University of Wyoming, a diagnostic pathologist at the Wyoming State Veterinary Laboratory, Editor of the *Journal of Wildlife Diseases*, and had been an adviser or consultant to the National Academy of Sciences, National Institutes of Health, United Nations, Morris Animal Foundation, and the U.S. Food and Drug Administration. She was the world's leading expert on the pathology of chronic wasting disease (CWD) in deer and elk and co-discovered it during her doctoral work. Beth also was noted for her work on brucellosis, plague, tularemia, canine distemper, keratoconjunctivitis of deer, and efforts to conserve the black-footed ferret and Wyoming toad.

The Tom Thorne and Beth Williams fund established by WDA and AAWV will be used to support the award, which will be given to wildlife veterinarians or health professionals who have made outstanding contributions to their field. It is hoped that sufficient funds will be raised to endow this award in perpetuity. Anyone who wishes to make a contribution toward this joint WDA/AAWV memorial effort may do so by sending a check to the WDA at P.O. Box 1897, Lawrence, KS 66044, or to AAWV Treasurer Mike Ziccardi at One Shields Ave, Wildlife Health Center, Davis, CA 95616. Please note that your contribution is for the Thorne-Williams Memorial Fund.

### **Second CWD Symposium**

The Second International Symposium on Chronic Wasting Disease (CWD) was held in Madison, Wisconsin, July 12-14, 2005, to provide a venue for reporting information developed since the first symposium in 2002. The conference was dedicated to the memory of Drs. Tom Thorne and Elizabeth Williams, who made invaluable contributions to the fields of animal health and wildlife management, particularly in the research and management

of CWD. The event was hosted by the Wisconsin Department of Natural Resources and the U.S. Geological Survey's National Wildlife Health Center and was attended by nearly 300 wildlife managers, researchers, and others. Additional sponsors included USDA-APHIS-Veterinary Services and USDA-APHIS-Wildlife Services, the CWD Alliance, and the U.S. Fish and Wildlife Service.

The symposium comprised two-and-a-half days of plenary and split sessions, followed by afternoon field trips to a captive cervid operation or to the CWD-affected area southwest of Madison. Scientific sessions were held on several important issues, including the management of CWD; ecology and epidemiology of CWD; biology of prions; human dimensions of CWD management; environmental contamination, disposal, and disinfection; diagnostics; and CWD surveillance. The conference program, including abstracts for the papers that were presented, can be found at the CWD Alliance website ([www.cwd-info.org](http://www.cwd-info.org)).

It was readily apparent that much progress had been made on several issues identified in the 2002 *Plan for Assisting States, Federal Agencies, and Tribes in Managing CWD in Wild and Captive Cervids* (National CWD Management Plan). Progress was easy to measure in some areas, such as CWD surveillance of wild and captive deer and elk, laboratory experiments, animal inoculations, public opinion surveys, and computer models but was harder to document for issues at the landscape level, such as the ecological effects of CWD in wild populations and the effectiveness of CWD management actions.

Interest was high for all of the sessions but was focused particularly on the different approaches undertaken by states and provinces to manage CWD in free-ranging cervid populations. Management goals and strategies differed primarily on the basis of the perceived duration and extent of CWD in wild deer and elk. In general, wildlife management agencies are attempting to contain the disease

and reduce its prevalence in states and provinces where CWD covers large geographic areas, apparently where the disease was introduced long ago. However, in locations where the introduction appears to be recent and small areas are affected, the goal is eradication, typically through dramatic reduction of wild cervid populations. Both of these management approaches are consistent with those recommended in the National CWD Management Plan. Neither of these strategies is a desirable substitute for preventing the introduction of CWD when it comes to a truly effective approach to managing CWD in deer and elk.

Chronic wasting disease remains a high priority issue for wildlife managers, animal health officials, hunters, captive cervid owners, public health agencies, scientific researchers, and the general public. Fortunately, results of research and epidemiological investigations continue to indicate there is no strong evidence that CWD is transmissible to domestic livestock or humans, and there is a low probability of CWD jumping the species barrier. Efforts to better understand the biology of CWD and other prion diseases will continue, with emphasis on the susceptibility of species other than mule deer, white-tailed deer, and elk; the pathogenesis and epidemiology of CWD in wildlife; and the effect of this disease on wildlife populations. The Third International CWD Symposium, which will occur at a time and place yet to be decided, will offer the opportunity to present these results. (Prepared by John Fischer)

## **Changes at SCWDS**

On July 1, 2005, The University of Georgia's College of Veterinary Medicine formed a new Department of Population Health, which includes SCWDS, the Poultry Diagnostic and Research Center, the Food Animal Medicine Program, and the Laboratory Animal Medicine Program. Since the 1980s, SCWDS has been a separate administrative unit within the College, and our multi-disciplinary faculty

members had academic homes in Pathology, Infectious Diseases, and the Warnell School of Forest Resources. SCWDS faculty now will be members of the Department of Population Health; however, most will maintain ties with their former departments through adjunct faculty appointments.

The Department of Population Health was formed primarily to create an academic arena focused on training veterinarians for "public practice." Public practice is experiencing a growing shortage of trained individuals and primarily comprises those veterinary occupations dedicated to population health rather than individual animal treatment. It includes wildlife, domestic livestock and poultry health, and public health. The department will maintain strong research commitments and is building expertise in epidemiology to support these missions. The new department head is Dr. John Glisson, former Head of the Department of Avian Medicine and former Associate Dean for Public Service and Outreach at the college. We have worked with John in the past, and he is a strong supporter of SCWDS. We look forward to working with him in the future as the new department and training program develop.

In addition to this administrative change, we are experiencing changes in some of the familiar faces at SCWDS. Ms. Kali King left in June 2005, to take another position at The University of Georgia. Our new Business Manager, Ms. Brenda Yuhas, started in July and got right to work on the grants and cooperative agreements that keep SCWDS going. Dr. Cynthia Tate has received her PhD degree in Medical Microbiology under the direction of Dr. Randy Davidson and has taken a position as a wildlife veterinarian with the Wyoming Game and Fish Department. Cynthia received the 2005 Wildlife Disease Association's Scholarship Award in her last year at SCWDS. Another one of Randy's graduate students, Ms. Vivien Dugan, will receive her PhD in Medical Microbiology in August 2005 and start a post-doctoral fellowship at the Institute for Genomic Research and the Armed Forces Institute of Pathology, Molecular Pathology Department, located in Rockville, Maryland. Dr. Samantha Gibbs, who conducted West Nile virus research under Dr. David Stallknecht, will receive her PhD in Infectious Diseases in August 2005 and will begin working as a post-doctoral research associate at SCWDS after graduation. We thank all of these people for their commitment to SCWDS and the great work they have done or will do at SCWDS, and we wish them the best in their new endeavors. (Prepared by John Fischer)

## 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, fill out the request form and return it to: Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602

- \_\_\_\_\_ Allison, A.B., D.G. Mead, S.E.J. Gibbs, D.M. Hoffman, and D.E. Stallknecht. 2004. West Nile virus viremia in wild rock pigeons. *Emerging Infectious Diseases* 10(12): 2252-5.
- \_\_\_\_\_ Birrenkott, A.H., S.B. Wilde, J.J. Hains, J.R. Fischer, T.M. Murphy, C.P. Hope, P.G. Parnell, and W.W. Bowerman. 2004. Establishing a food-chain linkage between aquatic plant material and avian vacuolar myelinopathy in mallard ducks (*Anas platyrhynchos*). *Journal of Wildlife Diseases* 40(3): 485-492.
- \_\_\_\_\_ Brown, J.D., J.M. Richards, J. Robertson, S. Holladay, and J.M. Sleeman. 2004. Pathology of aural abscesses in free-living eastern box turtles (*Terrapene carolina carolina*). *Journal of Wildlife Diseases* 40(4): 704-712.
- \_\_\_\_\_ Dugan, V.G., A.S. Varela, D.E. Stallknecht, C.C. Hurd, and S.E. Little. 2004. Attempted experimental infection of domestic goats with *Ehrlichia chaffeensis*. *Vector-borne and Zoonotic Diseases* 40(3): 131-136.
- \_\_\_\_\_ Dugan, V.G., J.K. Gaydos, D.E. Stallknecht, S.E. Little, A.D. Beall, D.G. Mead, C.C. Hurd, and W.R. Davidson. 2005. Detection of *Ehrlichia* spp. in raccoons (*Procyon lotor*) from Georgia. *Vector-Borne and Zoonotic Diseases* 5(2): 162-171.
- \_\_\_\_\_ Ellis, A.E., D.G. Mead, A.B. Allison, S.E.J. Gibbs, N.L. Gottdenker, D.E. Stallknecht, and E.W. Howerth. 2005. Comparison of immunohistochemistry and virus isolation for diagnosis of West Nile virus. *Journal of Clinical Microbiology* 46(6): 2904-2908.
- \_\_\_\_\_ Gaydos, J.K., J.M. Crum, W.R. Davidson, S.S. Cross, S.F. Owen, and D.E. Stallknecht. 2004. Epizootiology of an isolated epizootic hemorrhagic disease outbreak in West Virginia. *Journal of Wildlife Diseases* 40(3): 383-393.
- \_\_\_\_\_ Gerhold, R.W. and J.R. Fischer. 2005. Avian tuberculosis in a wild turkey. *Avian Diseases* 49:164-166.
- \_\_\_\_\_ Gerhold, R.W., E.W. Howerth, and D.S. Lindsay. 2005. *Sarcocystis neurona* associated meningoencephalitis and description of muscular sarcocysts in a fisher (*Martes pennanti*). *Journal of Wildlife Diseases* 41(1): 224-230.
- \_\_\_\_\_ Gibbs, S.E.J., D.M. Hoffman, L.M. Stark, N.L. Marlenee, B.J. Blitvich, B.J. Beaty, and D.E. Stallknecht. 2005. Persistence of antibodies to West Nile virus in naturally infected rock pigeons (*Columbia livia*). *Clinical and Diagnostic Laboratory Immunology* 12(5): 665-667.
- \_\_\_\_\_ Lewis-Weis, L.A., R.W. Gerhold, and J.R. Fischer. 2005. Attempts to reproduce vacuolar myelinopathy in domestic swine and chickens. *Journal of Wildlife Diseases* 40(3): 476-484.

\_\_\_\_\_ Rocke, T.E., N.J. Thomas, C.U. Meteyer, C. Quist, J.R. Fischer, T. Augspurger, and S.E. Ward. 2005. Attempts to identify the source of avian vacuolar myelinopathy for water birds. *Journal of Wildlife Diseases* 41(1): 163-170.

\_\_\_\_\_ Stallknecht, D.E., J. Greer, M. Murphy, D.G. Mead, and E.W. Howerth. 2004. Vesicular stomatitis virus New Jersey in pigs: effect of strain and serotype on contact transmission. *American Journal of Veterinary Research* 65: 1233-1239.

\_\_\_\_\_ Tate, C.M., E.W. Howerth, D.E. Stallknecht, A.B. Allison, J.R. Fischer, and D.G. Mead. 2005. Eastern equine encephalitis in a wild white-tailed deer (*Odocoileus virginianus*). *Journal of Wildlife Diseases* 41(1): 241-245.

\_\_\_\_\_ Varela, A.S., D.E. Stallknecht, M.J. Yabsley, V.A. Moore, E.W. Howerth, W.R. Davidson, and S.E. Little. 2005. Primary and secondary infection with *Ehrlichia chaffeensis* in white-tailed deer. *Vector-borne and Zoonotic Diseases* 5(1): 48-57.

\_\_\_\_\_ Yabsley, M.J., T.N. Norton, M.R. Powell, and W.R. Davidson. 2005. Molecular and serologic evidence of multiple tick-borne pathogens in free-ranging lemurs from St. Catherine's Island, Georgia. *Journal of Zoo and Wildlife Medicine* 35: 503-509.

\_\_\_\_\_ Yabsley, M.J., C. Dresden-Osborne, E.E. Pirkle, J.K. Kirven, and G.P. Noblet. 2004. Filarial worm infections in shelter dogs and cats from northwestern South Carolina, U.S.A. *Comparative Parasitology* 71(2): 154-157.

\_\_\_\_\_ Yabsley, M.J., M.C. Wimberly, D.E. Stallknecht, S.E. Little, and W.R. Davidson. 2005. Spatial analysis of the distribution of *Ehrlichia chaffeensis*, causative agent of human monocytotropic ehrlichiosis, across a multi-state region. *American Journal of Tropical Medicine and Hygiene* 72(6): 840-850.

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