SCWDS BRIEFS

A Quarterly Newsletter from the
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VS Outbreak in the Western U.S.

On May 10, 2004, a foreign animal disease investigation was initiated at a calf-roping facility in Balmorhea, Texas, due to a report of vesicular lesions in horses on the premises. On May 18, 2004, the National Veterinary Services Laboratories in Ames, Iowa, confirmed vesicular stomatitis (VS) in horses at one premises in Texas. As of July 21, 2004, infected animals were identified on a total of 45 premises in Colorado (11), New Mexico (21), and Texas (13). These are the first reports of VS in livestock in the United States since the 1998 epizootic. Attempts to control the spread of VS is centered on identification of clinical cases and the imposition of a quarantine on animals at and near affected premises. In response to this year's outbreak, some states have increased restrictions regarding importation of animals from the affected states.

VS is caused by a group of antigenically related but distinct viruses of the genus *Vesiculovirus* in the family *Rhabdoviridae*. Two VS virus serotypes (New Jersey and Indiana) are known to periodically cause epizootics in the western United States. Clinical disease, regardless of virus serotype, is characterized by vesicular lesions in or around the oral cavity, coronary bands, and teats.

VS is an economically important disease that primarily affects cattle, swine, and horses. Additionally, this disease is extremely important to animal health authorities because the clinical signs in cattle, swine, and other cloven-hoofed animals mimic those of foot-and-mouth disease, one of the most devastating livestock diseases. Therefore, when VS occurs in the United States it is extremely important that the diagnosis is confirmed and that infection with the highly contagious foot-and-mouth disease virus is ruled out.

Although clinical VS has never been a problem in any wildlife species, this possibility cannot be dismissed. If you encounter wild ruminants or swine with clinical signs related to mouth or hoof lesions (drooling, poor appetite, lameness), please contact your state veterinarian or Veterinary Services of USDA's Animal and Plant Health Inspection Service.
(Prepared by Danny Mead)

SCWDS VS Studies

Vesicular stomatitis (VS) has been recognized as a disease of livestock and other animals for over a century, and while the causative agents have been studied intensively in the laboratory, specific virus transmission routes have remained obscure. Arthropod vectors are thought to be involved in epizootic transmission, however their specific role has remained controversial. It is not known how vectors become infected because a sustainable viremia (virus in the blood), which is believed to be necessary for the infection of arthropod vectors, is absent in livestock following natural or experimental VS virus infections.

In recent SCWDS studies supported by a grant through the USDA's National Research Initiative Competitive Grants Program, significant progress was made in understanding the transmission of the New Jersey serotype of VS (VSNJV) and the role of insects in VSNJV epidemiology. We demonstrated black fly transmission of VSNJV to livestock hosts and have determined that viremia is not necessary for an insect to become infected with the virus while feeding on an infected host. Our studies showed that black flies could become infected with VSNJV by feeding on virus-rich lesions and by co-feeding with infected black flies. Additionally, we demonstrated that the clinical course of VSNJV infection in horses and pigs following transmission by an infected insect is related to the insect bite site. In our studies, when VSNJV-infected insects fed on the horses' muzzles or the pigs' snouts, lesions consistently formed at the bite sites. Conversely, when insect feeding was restricted to haired areas, such as the abdomen, the consistent result was seroconversion without lesion formation. These data suggest that the bite of a VSNJV-infected black fly at a site other than the muzzle or snout likely would lead only to seroconversion.

Because clinical VS developed only in hosts when feeding occurred on the muzzle or snout, an area that is not a preferred feeding site of black flies, it is likely that during epidemics most of the bites received from VSNJV-infected black flies result in unapparent infection and seroconversion. These findings may offer an explanation for serological surveys conducted after VSNJV outbreaks in the western United States which revealed that the majority of livestock exposed to VSNJV (as determined by seroconversion) never developed clinical VS.

Identification of the specific routes of VSNJV transmission has important implications for disease control measures. Based on our results, limiting VSNJV animal-to-animal contact transmission via livestock quarantines on VS-positive premises might provide only a partial solution. Restrictions on animal movement would be less effective where insects such as black flies play a role in biological transmission of VSNJV. Therefore, the presence of blood-feeding insects in VS epidemic regions should be considered in the development of VS control and eradication programs. In addition, these data support the need for protecting animals against insect feeding, as well as the need for basic insect control measures. (Prepared by Danny Mead)

CWD Developments

The *Plan for Assisting States, Federal Agencies, and Tribes in Managing Chronic Wasting Disease in Wild and Captive Cervids* (CWD National Management Plan) was completed in June 2002. The first progress report on the plan was released in May 2004. The progress report lists accomplishments and the next steps necessary to achieve the objectives set forth in the CWD National Management Plan. According to the report, nearly 118,000 wild white-tailed deer, mule deer, and elk were tested in the United States from October 2002 to September 2003, with 592 animals testing positive for the CWD prion. More than $38,000,000 was spent by federal and state wildlife and animal health agencies on CWD-related activities during this same period. Surveillance data from individual states are contained in the report, which also includes a list of ongoing and completed CWD research projects conducted since 1978. The complete report and the CWD National Management Plan can be accessed at the CWD Alliance website (www.cwd-info.org).

Several research papers contributing to the body of information available on CWD have been published in recent months. Two CWD articles appeared in the June 2004 issue of the journal *Emerging Infectious Diseases*. In Chronic Wasting Disease and Potential Transmission to Humans, the authors reported on epidemiological studies of cases of fatal human neurological disease to identify any links to exposure to CWD. Cases that were investigated included prion disease in unusually young patients, Creutzfeldt-Jakob disease (CJD) in two persons with a history of exposure to venison obtained from the known CWD endemic areas, the highly publicized fatal neurological diseases in three persons who attended a wild game feast, and others. Additionally, the incidence and age distribution of CJD patients in Colorado and Wyoming, where CWD has been endemic for decades, was compared to other parts of the United States. The study showed, "...no human cases of prion disease with strong evidence of a link with CWD were identified," and the authors concluded, "… lack of evidence of a link between CWD transmission and unusual cases of CJD, despite several epidemiologic investigations, and the absence of an increase in CJD incidence in Colorado and Wyoming suggest that the risk, if any, of transmission of CWD to humans is low." However, they recommended that additional epidemiologic studies be conducted and that hunters minimize their risk for exposure to the CWD agent by following the recommendations of public health authorities and wildlife agencies.

In the second article in *Emerging Infectious Diseases*, Environmental Sources of Prion Transmission in Mule Deer, investigators reported on a study to determine if CWD can be transmitted to susceptible animals indirectly from environments contaminated by excreta or decomposed carcasses. Their findings are as follows: "Under experimental conditions, mule deer became infected in two of three paddocks containing naturally infected deer, in two of three paddocks where infected deer carcasses had decomposed in situ approximately 1.8 years earlier, and in one of three paddocks where infected deer had last resided 2.2 years earlier." The authors concluded, "Although live deer and elk represent the most plausible mechanisms for geographic spread of CWD, our data show that environmental sources could contribute to maintaining or prolonging local epidemics, even when all infected animals are eliminated." Both articles in *Emerging Infectious Diseases* can be found at www.cdc.gov/ncidod/EID.

Two additional publications have provided information on the epidemiology of CWD in captive deer. In the September 4, 2003, issue of the journal *Nature*, in an article entitled Prion Disease: Horizontal Prion Transmission in Mule Deer, it was reported, "...horizontal transmission is remarkably efficient, producing a high incidence of disease (87%) in a cohort of mule deer in which maternal transmission was improbable, indicating that horizontal transmission is likely to be more important [than maternal transmission] in sustaining CWD epidemics." Furthermore, the authors stated, "…direct and indirect transmission of CWD can probably occur, and concentrating deer in captivity or by feeding them artificially may facilitate transmission."

In a paper published in the April 2004 issue of the *Journal of Wildlife Diseases*, entitled Epidemiology of Chronic Wasting Disease in Captive White-tailed and Mule Deer, the authors found similar patterns in both species. They concluded, "...sustained horizontal transmission of CWD most plausibly explained epidemic dynamics.... It follows that CWD epidemic dynamics in sympatric, free-ranging white-tailed deer

and mule deer in western North American ranges also may be similar."

Although some gaps remain, the contributions of seasoned CWD researchers are adding to the body of knowledge on CWD. For example, Dr. Mike Miller with the Colorado Division of Wildlife authored or co-authored all four publications listed above and Dr. Beth Williams with the Wyoming State Veterinary Laboratory co-authored three of the four publications. Research is an integral component of the CWD National Management Plan, and projects are underway to elucidate additional epidemiological aspects that are essential to our understanding of the disease and to the development of practical control measures. (Prepared by John Fischer)

Second International CWD Symposium

The Second International Chronic Wasting Disease Symposium, hosted by the Wisconsin Department of Natural Resources, will be held in Madison, Wisconsin, July 12-14, 2005. This symposium will include plenary sessions, concurrent sessions, and panel discussions on current topics important to understanding and managing chronic wasting disease (CWD) in free-ranging and farmed cervids. Session topics likely will include: CWD biology, wild deer disease ecology, farmed cervid epidemiology, CWD diagnostics, CWD control and management, USDA and state cervid herd CWD certification, and human dimensions of CWD. Field trips will be offered. Plans for the seminar are in the preliminary stages and forthcoming information will be provided at the Wisconsin Department of Natural Resources website as it becomes available (http://dnr.wi.gov/org/land/wildlife/whealth/issues/CWD/index.htm). (Prepared by Tom M. Hauge, Wisconsin Department of Natural Resources)

Crow Decoys Used in WNV Study

Surveillance of dead wild birds plays a critical role in the early detection of West Nile virus (WNV), which offers public health authorities an opportunity to advise the public to take measures to prevent exposure to the virus. In DeKalb County, Georgia, in 2002, extensive data on dead wild bird surveillance was compiled by the DeKalb County Board of Health as part of Georgia's WNV monitoring. DeKalb County is part of the metro-Atlanta area. These surveillance activities demonstrated that more WNV-positive dead birds were reported in urban areas versus rural areas. However, the larger number of WNV cases in the more highly urbanized portions of DeKalb County could be interpreted in at least two ways: It could reflect a truly higher level of WNV activity, or it could reflect a higher probability of dead bird detection and reporting associated with the higher human density. Understanding which factors are related to detection and reporting of bird mortalities is of obvious importance in regard to WNV surveillance, and, in more general terms, to the understanding of any disease affecting wildlife.

SCWDS, in collaboration with the DeKalb County Board of Health, conducted a study to assess the detection and reporting of crow carcasses within these urban and rural environments in order to determine if the previously observed WNV distribution might represent a potential reporting bias. Commercially purchased plastic crow decoys were used instead of dead birds, and two trials were conducted using 400 decoys in each trial. Decoys were labeled with reporting instructions that conformed to those used by the DeKalb County Board of Health for real dead birds and were distributed throughout designated urban and rural areas within DeKalb County along randomly selected public roadways. Decoys were monitored after 7 days and

were categorized as detected and reported, missing and unreported, or still present in the environment. Only about 10% of the total number of known "dead crows" were reported and a significantly higher proportion of decoys were reported from the urban area than from the rural area. The proportion reported from the urban area (17%) was approximately six times that of the rural area (3%). This suggests that factors such as human density and landscape differences may influence dead wild bird reporting and the perceived distribution of WNV. Our study demonstrates that passive surveillance will result in considerable underestimation of the total mortality of birds and, thus, the underestimation of the extent of WNV distribution.

A second phase of research related to use of wild birds as a surveillance system for WNV currently is being conducted by SCWDS in the vicinity of Athens, Georgia. The intent of the current phase is to gain information on the persistence and fate of actual dead birds in settings similar to those used with plastic decoys in DeKalb County. The current project consists of monitoring how long dead crows, English sparrows, and house finches remain in the environment and attempting to determine what happens to the carcasses that disappear. Motion-sensitive "trail cameras" will be used to obtain photographic evidence of which species of scavengers remove birds in both urban and rural settings. When combined, these studies will provide important information on interpreting dead bird surveillance data and on the potential for oral exposure to WNV among both avian and mammalian scavengers. (Prepared by Marsha Ward and Randy Davidson, D.B. Warnell School of Forest Resources and SCWDS)

Whitetails and STARI

Epidemiology

Although Lyme disease caused by a spirochete bacterium, *Borrelia burgdorferi*, is relatively rare in the southeastern United States, a Lyme disease-like infection referred to as Southern Tick-Associated Rash Illness (STARI) and thought to be caused by *Borrelia lonestari*, has been recognized in people in this region. For details, please see SCWDS BRIEFS Vol. 18, No. 4 and Vol. 20, No. 1. Previous work at SCWDS resulted in identification of *B. lonestari* infection in deer from throughout the South via polymerase chain reaction (PCR) and the first isolation of *B. lonestari* in tick cell culture. This isolate since has been used in an experimental infection trial to confirm that white-tailed deer may serve as a reservoir host for *B. lonestari*.

In the experimental infection trial, two white-tailed deer fawns were inoculated with culture-grown *B. lonestari* organisms and then monitored for evidence of infection. Both deer developed circulating spirochetes, which were detected by direct microscopic examination of blood smears and PCR amplification of organism from blood samples. *Borrelia lonestari* also was re-isolated in culture from both deer. In contrast, circulating spirochetes were not detected in laboratory mice that were inoculated with the culture. Antibodies to *B. lonestari* were detected in both deer and mice following experimental inoculation.

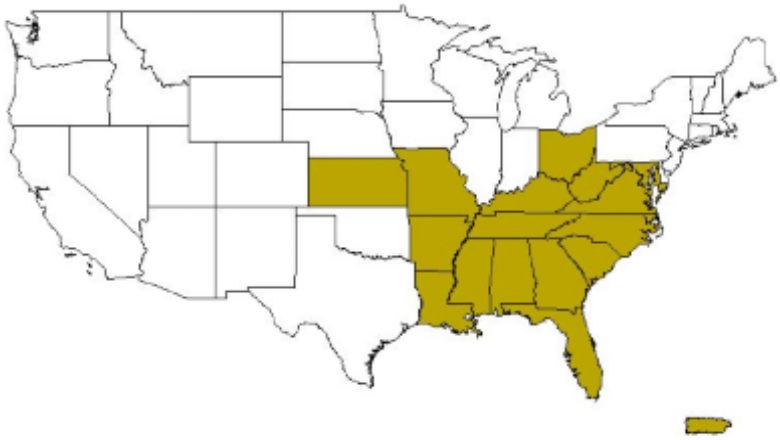
The finding that deer, but not mice, are able to support an infection with *B. lonestari* is consistent with previous data that indicate that this organism is transmitted by the lone

star tick, *Amblyomma americanum*. All motile stages of the lone star tick feed on deer, but these ticks are rarely found on rodents in nature and typically only larval stages infest rodents. Other human pathogens known or suspected to be maintained in a white-tailed deer/lone star tick cycle include *Ehrlichia chaffeensis* and *Ehrlichia ewingii*. Lone star ticks feed on deer as larvae, nymphs, and adults, thus, ample opportunity exists for a tick to acquire infection from an infected deer in one life cycle stage and transmit it to a naïve deer or a person in a subsequent feeding.

This study is important in that it describes successful experimental infection of white-tailed deer with *B. lonestari*, providing an initial animal infection model and offering support to the hypothesis that white-tailed deer play an integral role in the natural history of this organism. However, additional experimental infection trials using tick transmission are necessary to confirm that *B. lonestari* is maintained in nature in a cycle involving white-tailed deer as reservoir hosts and lone star ticks as vectors. (Prepared by Susan Little)

New SCWDS Member

The Ohio Division of Wildlife joined SCWDS as an associate member beginning July 1, 2004. Ohio is the first state to join SCWDS since 1997, when the Kansas Department of Wildlife and Parks became our first associate member. SCWDS was founded by the Southeastern Association of Fish and Wildlife Agencies (SEAFWA) with 11 member states in 1957. Over the last 47 years (we'll be celebrating our 50th anniversary in 3 years!) additional SEAFWA states have become full SCWDS members in addition to our two associate members from non-SEAFWA states. The full listing of SCWDS members and associate members now includes the fish and wildlife management agencies of Alabama, Arkansas, Florida, Georgia, Kansas, Kentucky, Louisiana, Maryland, Mississippi, Missouri, North Carolina, Ohio, Puerto Rico, South Carolina, Tennessee, Virginia, and West Virginia. We welcome Ohio to SCWDS and look forward to assisting the Division of Wildlife with the management of their wildlife populations. (Prepared by John Fischer)



SCWDS Members, July 1, 2004

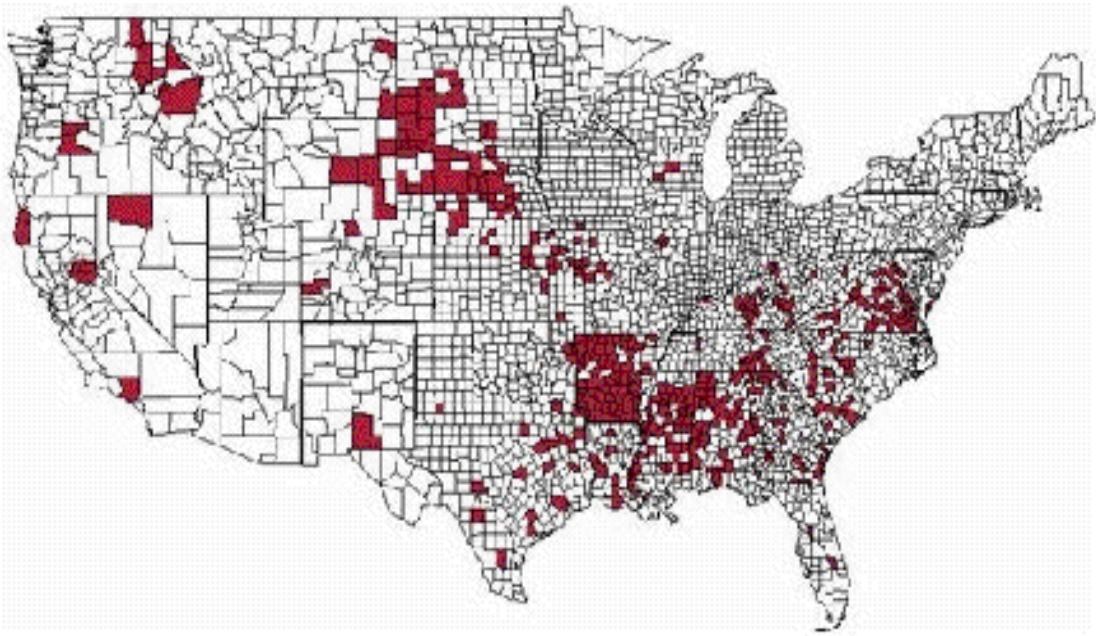
2003 HD Final Report

The Final Report of the 2003 Hemorrhagic Disease (HD) Surveillance project has been completed and distributed to all cooperators. To gather data, SCWDS corresponded with state fish and wildlife agency administrators in each state, plus individuals with most of the state veterinary diagnostic laboratories. Additional participants included personnel in the U.S. Fish and Wildlife Service and USDA's Animal and Plant Health Inspection Service. We are indebted to all respondents for their efforts to monitor our North American wild ruminants for these important diseases. A full copy of the report can be provided upon request. Also, if you wish to participate in the upcoming 2004 survey, please let us know.

During late summer and fall of 2003, suspected or confirmed HD activity was reported among wildlife from 411 counties in 32 states (see following map). Disease confirmation was made by demonstration of the causative virus or viruses in 12 states as follows: epizootic hemorrhagic disease virus (GA, ID, KS, MO, SC, TN, TX, WA, WY); bluetongue virus (KS, WA); deer adenovirus (CA); and not specified (CO, SD). As usual, several of the southern states had generalized activity that was a mix of acute losses and chronic lesions; some midwestern and western states had mortality among deer.

This surveillance project has been conducted annually since 1980 and represents one of the most comprehensive data bases for wildlife morbidity and mortality anywhere. Over the 24 years of the survey, there has been a dramatic increase in awareness and concern among wildlife managers about health issues of free-ranging ungulates, and there has been a simultaneous improvement in the diagnostic procedures available for confirming the disease agents. Nevertheless, the mortality events in which HD was suspected but not diagnosed are much too commonplace. History has shown that wildlife disease dynamics are not static, and over time *new problems are an absolute certainty*. For example, two newly recognized cervid diseases – deer adenovirus and chronic wasting disease – have emerged in wild populations since the HD survey was begun, and other diseases could emerge at any time.

We implore each of you to encourage your personnel and associates to submit specimens from any wildlife mortality event to an appropriate diagnostic laboratory. Although HD outbreaks in big game are not big news today and we may feel comfortable in dealing with them, one should have an uneasy feeling about unexplained losses. The next "new disease" could develop spontaneously in nature or it could be accidentally or maliciously introduced. Regardless, increased diagnostic efforts are the key to our timely reaction to protect our valuable wildlife resources. (Prepared by Vic Nettles)



Clinical HD Reported from Deer in 2003

New Faces at SCWDS

There have been quite a few changes in the "SCWDS Family" in recent months. Several staff members have left SCWDS for a variety of reasons, and several new employees have joined us. Caroline Duffie is starting graduate school at the University of Missouri in St. Louis. Robbie Edalgo's wife Jen has started graduate school at the University of West Virginia and they have moved to Morgantown. Clay George has taken a position as a wildlife biologist with the Georgia Department of Natural Resources. Darrell Kavanaugh accepted a job as a wildlife biologist with USDA's Wildlife Services. Lynn Lewis-Weiss's husband, Dr. Kevin Weiss, graduated from the University of Georgia's College of Veterinary Medicine, and they have moved to Charlotte, North Carolina. Nate Mechlin returned home and is a wildlife biologist with the Missouri Department of Conservation.

We will really miss all of these fine folks, but we have welcomed some new people to the staff. Brian Chandler, Jay Cumbee, Ginger Goekjian, Bill Hamrick, Sabrina McGraw, Kerri Pedersen, and Ben Wilcox have all come on board, and each one brings valuable skills and knowledge to our organization. We are happy to have them with us at SCWDS and know that you will enjoy working with them. (Prepared by Gary Doster)

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: Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602

_____Corn, J.L., D.E. Stallknecht, N.M. Mechlin, M.P. Luttrell, and J.R. Fischer. 2004. Persistence of pseudorabies virus in feral swine populations. *Journal of Wildlife Diseases* 40(2): 307-310.

_____Davidson, W.R., E.J.B. Manning, and V.F. Nettles. 2004. Culture and serologic survey for *Mycobacterium avium* subsp. *paratuberculosis* infection among southeastern white-tailed deer (*Odocoilei virginianus*). *Journal of Wildlife Diseases* 40(2): 301-306.

_____Flack, G.L., M.J. Yabsley, B.A. Hanson, and D.E. Stallknecht. 2004. Hemorrhagic disease in Kansas: Enzootic stability meets epizootic disease. *Journal of Wildlife Diseases* 40(2): 288-293.

_____Gottdenker, N.L., E.W. Howerth, and D.G. Mead. 2003. Natural infection of a great egret (*Casmerodius albus*) with eastern equine encephalitis virus. *Journal of Wildlife Diseases* 39(3): 702-706.

_____Hanson, B.A., D.E. Stallknecht, D.E. Swayne, L.A. Lewis, and D.A. Senne. 2003. Avian influenza viruses in Minnesota ducks during 1998-2000. *Avian Diseases* 47: 867-871.

______Mead, D.G., E.W. Gray, R. Noblet, M.D. Murphy, E.W. Howerth, and D.E. Stallknecht. 2004. Biological transmission of vesicular stomatitis virus (New Jersey serotype) by *Simulium vittatum* (Diptera: Simuliidae) to domestic swine. *Journal of Medical Entomology* 41(1): 78-82.

______Mecham, J.O., D.E. Stallknecht, and W.C. Wilson. 2003. The S7 gene and VP7 protein are highly conserved among temporally and geographically distinct American isolates of epizootic hemorrhagic disease virus. *Virus Research* 94(2003): 129-133.

______Munderloh, U.G., C.M. Tate, M.J. Lynch, E.W. Howerth, T.J. Kurtti, and W.R. Davidson. 2003. Tick-cell culture isolation of *Anaplasma* sp. from white-tailed deer. *Journal of Clinical Microbiology* 41: 4328-4335.

______Varela, A.S., M.P. Luttrell, E.W. Howerth, V.A. Moore, W.R. Davidson, D.E. Stallknecht, and S.E. Little. 2004. First culture isolation of *Borrelia lonestari*, putative agent of southern tick-associated rash illness. *Journal of Clinical Microbiology* 42(3): 1163-1169.

______Varela, A.S., D.E. Stallknecht, M.J. Yabsley, V.A. Moore, W.R. Davidson, and S.E. Little. 2003. Experimental infection of white-tailed deer (*Odocoileus virginianus*) with *Ehrlichia chaffeensis* by different inoculation routes. *Journal of Wildlife Diseases* 39(4): 881-886.

______Yabsley, M.J., S.E. Little, E.J. Sims, V.G. Dugan, D.E. Stallknecht, and W.R. Davidson. 2003. Molecular variation in the variable length PCR target (VLPT) and 120-kDa antigen genes of *Ehrlichia chaffeensis* from white-tailed deer. *Journal of Clinical Microbiology* 41(11): 5202-5206.

______Yabsley, M.J., V.G. Dugan, D.E. Stallknecht, S.E. Little, J.M. Lockhart, J.E. Dawson, and W.R. Davidson. 2003. Evaluation of a prototype *Ehrlichia chaffeensis* surveillance system using white-tailed deer (*Odocoileus virginianus*) as natural sentinels. *Vector Borne and Zoologic Diseases* 3(4): 195-207.

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