SCWDS BRIEFS

A Quarterly Newsletter from the

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

Phone (706) 542-1741 **FAX (706) 542-5865**

Volume 30 **July 2014** **Number 2**

Another Vesicular Stomatitis Outbreak

On May 23, 2014, the USDA-APHIS-National Veterinary Services Laboratories in Ames, Iowa, confirmed the diagnosis of vesicular stomatitis New Jersey virus (VSNJV) infection in four horses on one premises in Kinney County, Texas. Since the start of the outbreak, vesicular stomatitis (VS) has been diagnosed in 343 horses and 10 cattle on 241 premises in eight Colorado counties and eleven Texas counties.

Vesicular stomatitis is a viral disease caused by two VS virus serotypes, New Jersey and Indiana. The viruses are members of the genus *Vesiculovirus* within the family Rhabdoviridae. Clinical signs and lesions of VS infection in animals include excessive salivation, as well as blanched, raised or broken vesicles on the tongue, lips and nostrils, corners of the mouth, and the gums, teats, coronary bands, and sheath. These signs and lesions may be seen in cattle, horses and other equines, pigs, and South American camelids, such as llamas. Horses are the most seriously affected species, but animals usually recover in about two weeks if there are no complications, such as secondary infections. Humans also are susceptible to infection and may develop a flu-like illness. Although clinical disease has not been reported, serological evidence of infection has been found in many other animals including deer, pronghorn antelope, bighorn sheep, bats, raccoons, opossums, lynx, bobcats, bears, coyotes, foxes, and others.

The clinical signs of VS are similar to those of foot-and-mouth disease (FMD) in cattle, swine, and other cloven-hoofed animals. (Horses are not affected by FMD.) Consequently, VS is reportable to the World Organization for Animal Health, formerly the Office International des Epizooties (OIE). As such, identification of VS in livestock potentially can lead to economic and regulatory impacts, such as bans on importation of animals and animal products.

Historically, VS epizootics have been reported in the southeastern United States; major epizootics now appear confined to the western states. Outbreaks have been reported with increased frequency and have occurred in 2004, 2005, 2006, 2009, 2010, and 2012. Vesicular stomatitis tends to occur in late spring and affect few to multiple premises in one or two states when outbreaks are mild. However, multiple states and premises have been involved some years. For example, in 2012, the outbreak involved only 36 premises in just two states (Colorado and New Mexico), whereas the 2005 outbreak affected animals on 445 premises in nine states (Arizona, Colorado, Idaho, Montana, Nebraska, New Mexico, Texas, Utah, and Wyoming).

Vesicular stomatitis outbreaks have been difficult to stop once the virus spreads from the index premises. Reasons include the lack of a vaccine to protect animals and the multiple transmission routes the viruses can take. In studies conducted by SCWDS, we have demonstrated that VSNJV is efficiently transmitted via animal-to-animal contact. In studies conducted using swine, a natural host, the virus was transmitted to 100% of the naïve contact animals after the virus was introduced into the group via an experimentally infected pig. This finding was validated experimentally in groups of horses and cows; however, animal-to-animal contact transmission in these species was less efficient. Additionally, we have demonstrated that black flies can serve as biological and mechanical VSNJV vectors. Other biological vectors of VSV include sand flies and possibly *Culicoides* midges. Determining the relative contribution of each of these transmission routes during an outbreak is impossible. Therefore, control efforts must be

Continued…

comprehensive and should emphasize isolating affected animals away from susceptible animals, controlling populations of biting insects, using insect repellants, especially along the coronary bands and around the muzzle and snout, and carefully cleaning equipment used for milking.

The 2014 outbreak continues with new cases announced regularly. Livestock owners and veterinarians who suspect that an animal may have VS should contact state or federal animal health authorities. Additional information on the current VS outbreak can be found at the website of the USDA-APHIS. (Prepared by Danny Mead)

MCF Hits Captive Deer

Malignant Catarrhal Fever (MCF) has been confirmed as the cause of a recent captive white-tailed deer die-off at a licensed breeding facility in Alabama. Of the 149 deer housed at the facility, approximately 60 (40%) died during the outbreak. Daily death losses generally consisted of 2-3 deer of all ages, although 5-6 dead deer were found on some days.

Officials with the Alabama Division of Animal Industry, Auburn University College of Veterinary Medicine, and USDA-Animal Plant and Health Inspection Service-Veterinary Services (VS) performed necropsies and conducted an epidemiological investigation. Testing at the USDA-VS-Foreign Animal Disease Diagnostic Laboratory on Plum Island, New York, detected Ovine Herpesvirus-2 (OHV-2) in a deer that died on May 27, and the investigation shifted focus to identifying the source of the virus. The deer were housed adjacent to other species, including two aoudad sheep, one domestic sheep, some kangaroos, and a camel. The domestic sheep was a 5- or 6-month-old lamb that had been on the premises for two months before the deer mortality began on May 19, 2014. A polymerase chain reaction (PCR) test subsequently confirmed this animal was carrying OHV-2 and was the likely source of the outbreak.

Malignant Catarrhal Fever is caused by herpesviruses in the gammaherpesvirus subfamily. None of the MCF viruses infect humans. As with other herpesviruses, infection is persistent in animals that survive. Two primary epidemiological forms of MCF are recognized: The wildebeest-associated form of MCF (WA-MCF) is caused by Alcelaphine Herpesvirus-1 (AHV-1), which has been associated with devastating disease in cattle in African countries when transmitted from infected wildebeests, which serve as silent reservoirs of the virus. The WA-MCF is detected periodically in the United States, with outbreaks typically occurring in zoo collections, although a well-publicized outbreak in cattle in 2008 began when heifers were exposed to wildebeest in Texas and subsequently shipped to several states (BRIEFS Vol. 24, No. 1). Almost all wildebeest are infected with the virus.

Sheep-associated MCF (SA-MCF) has had greater impacts in North America and is transmitted from asymptomatic sheep and goats to other artiodactyls, primarily deer, cattle, and bison. Viruses associated with this form of MCF include OHV-2, Caprine Herpesvirus-2 (CpHV-2), as well as several other MCF viruses, and it is believed that nearly all domestic sheep carry OHV-2.

Sheep do not develop clinical disease when infected, and are asymptomatic when they shed the virus. Shedding of OHV-2 in nasal and ocular secretions is most frequent from post weaning-age animals (as was the case with the recent captive deer die-off in Alabama) and from ewes at parturition. White-tailed deer and other ruminants that develop clinical MCF usually are dead end hosts. Animal to animal transmission has been suspected in some situations, but there has been no firm evidence that this is possible.

The WA- and SA-MCF forms of MCF are indistinguishable in infected animals, and clinical disease typically is acute and fatal 7-8 days following infection. Clinical signs of MCF can vary and are due to the predilection of the virus to infect lymphocytes and epithelial cells. Clinical signs and lesions include fever, lethargy, inappetence, diarrhea, ocular and nasal discharge, oral and buccal mucosal erosions and ulcerations, and corneal opacity. Internally, gross findings often include hemorrhagic intestinal contents, gastrointestinal ulcerations, pulmonary congestion and edema, and small hemorrhages in the spleen, intestine, heart, liver and other organs. There may be multiple, whitish nodules in several tissues, including heart, kidney, spleen and liver. Microscopically,

Continued…

epithelial necrosis is common, and the presence of widespread necrosis of arterioles and perivascular proliferation of lymphoid cells is considered to be pathognomonic for MCF. The perivascular lymphoid proliferations correspond to the whitish nodules seen grossly.

Due to the peracute nature of the disease in white-tailed deer, a presumptive diagnosis may be based on clinical signs and/or lesions, plus evidence of exposure to a reservoir. However, aerosolized MCF viruses can travel as far as three miles, and MCF cannot be ruled out in the absence of direct exposure. Diagnostic techniques for MCF have greatly improved in past years, and because isolation of the viruses is extremely difficult, quantitative PCR testing often is used to obtain a definitive diagnosis. Multiplex real-time PCR also is helpful to differentiate which MCF virus is involved when multiple reservoir species are present.

Malignant catarrhal fever has been diagnosed in many captive wild animals, including several cervid species housed or pastured in proximity to wildebeest or sheep. However, confirmed MCF cases in free-ranging ruminants in the U.S. have been rare. Sheep-associated MCF was diagnosed in four free-ranging mule deer in the winter of 2003 in Colorado. Post-mortem lesions were consistent with MCF, and genetic sequences of OHV-2 were detected by PCR testing. Another reported outbreak in free-ranging wildlife occurred in 1985 in black-tailed deer in California, but MCF was not confirmed through detection of viral genetic material.

This is not the first MCF outbreak in captive white-tailed deer in Alabama. A smaller MCF mortality event occurred in southern Alabama in December 1991 (SCWDS BRIEFS, Vol. 8, No. 1). The affected deer came from an exotic animal auction, and 7 of 8 animals from this shipment died. It was not determined which form of the MCF virus caused the 1991 outbreak.

The recent Alabama mortality event appears to have run its course, with no additional mortalities reported since the last week of June, approximately two weeks after the domestic and aoudad sheep were removed from the facility. To prevent future cases of MCF in captive cervids, owners are advised that captive deer should not be co-housed with exotic or domestic animal reservoir species. (Prepared by Anna Fagre, MPH, Colorado State University)

A New Arbovirus in the U.S.

It has been nearly 15 years since West Nile virus was first detected and subsequently became established in North America. However, attention now is focused on Chikungunya virus (CHIKV), another exotic arbovirus that has been introduced into the United States. Although Chikungunya outbreaks had been reported for several years in countries in Africa, Asia, Europe and the Pacific and Indian Oceans, the first locally acquired cases in the Western Hemisphere were reported in 2013 in the Caribbean. In July 2014, the first locally acquired cases were diagnosed in Florida.

Chikungunya virus is an Alphavirus and is related to eastern equine encephalitis virus (EEEv), which is endemic in many parts of the eastern and northeastern United States. Unlike EEEv, CHIKV is not known to cause disease in mammals other than humans, and the transmission cycle involves only humans and mosquitoes, with no wildlife reservoir.

Chikungunya virus was first identified in Tanzania in 1953 during an epidemic of a dengue-like illness with patients suffering from fever and joint pain. Since then it has been detected in numerous countries in central and southern Africa and has spread to several other continents. In 2013, the virus was confirmed or suspected in nearly 250 patients in Saint Martin. Currently, local transmission of CHIKV has been confirmed in several other Caribbean countries and islands, including Aruba, British Virgin Islands, Guadeloupe, Haiti, Martinique, Nevis, Puerto Rico, Saint Barthelemy, Saint Kitts, Saint Lucia, and Sint Maarten.

Human infection with CHIKV is characterized by an acute febrile period of 2-5 days that begins 3-7 days after the bite of an infected mosquito. The fever usually is followed by a longer period of joint pain in the extremities. Additional symptoms can include headache, muscle pain, joint swelling, and rash. Recovery varies with age and ranges from 5-to-15 days in younger people to several months in older patients. The

Continued…

case fatality rate is less than 1%, but the joint pain can be disabling.

Aedes aegypti and A. albopictus mosquitoes are vectors of CHIKV, but neither is native to the United States. The Asian tiger mosquito, *A. albopictus,* is native to Southeast Asia but has spread to several other continents. It was found in a shipment of used tires at the Port of Houston in 1985, and since then it has spread rapidly across much of the southeastern, midwestern and mid-Atlantic United States, and up the east coast to Maine. On the other hand, *A. aegypti*, which was found in the U.S. much earlier, remains confined to small areas in southern Arizona, Florida, and Texas, and appears to have declined or disappeared in areas into which *A. albopictus* has spread.

Aedes albopictus is a competent vector of several arboviruses including EEEv, yellow fever virus, the four dengue viruses, and *Dirofilaria immitis*, the heartworm of dogs and cats. This mosquito is a container breeder and therefore is closely associated with human habitations. Females feed throughout the day, as well as in early morning and late afternoon, and humans can reduce the likelihood of acquiring arboviruses carried by *A. albopictus* by using insect repellents, such as DEET and picaridin, maintaining screens on doors and windows, and mosquito-proofing water storage vessels and containers near homes.

From 2006 to 2013, travel-associated CHIKV infection was reported in an average of 28 people per year returning to the U.S. from affected areas; however, 584 cases had been reported in 2014 through mid-August, with the majority returning from the Caribbean. According to the U.S Centers for Disease Control and Prevention, the number of cases among travelers visiting or returning to the U.S. from affected areas will continue to increase. In addition, local spread of the virus in the continental United States may be seen in areas where *A. albopictus* has become established. (Prepared by Carolyn Chen, Western University and Danny Mead)

A Note to Our Readers

We thank you for your sustained interest in our quarterly newsletter, the SCWDS BRIEFS. Our latest issue marked the beginning of the 30th year of publication. We continue to receive positive feedback from many readers, which lets us know that we are still providing items of interest to you in each issue.

One difficult aspect of putting out a publication such as the BRIEFS is maintaining the mailing list. We want to reach as many of you as we can, but can do so only if you let us know you want to be included on the mailing list, notify us of any address changes, or inform us of someone else you know who would like to be added to the mailing list. Of course, if you want to reduce the volume of mail coming into your home or office you may opt to be removed from the regular mailing list and have your name added to our email list to be informed when each new issue is posted on our website. This way, you usually can read the newsletter at least 10 days before a mailed copy would arrive. As always, if you have suggestions for improvement of the Briefs, please let us hear from you. Our goal is to provide timely information of interest to you.

Recent SCWDS Publications Available

Below are some recent publications authored or co-authored by SCWDS staff. Many of these can be accessed online from the web pages of the various journals. If you do not have access to this service and would like to have a copy of any of these papers, let us know. Many can be sent to you electronically with minimum effort; others will be mailed to you. For your convenience, please indicate requested publications, fill out the form on page 7, and check the appropriate box to receive either an electronic copy or a hard copy and return it to us: Southeastern Cooperative Wildlife Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.

Continued…

____Allison, A.B., M.K. Keel, J.E. Phillips, A.N. Cartoceti, B.A. Munk, N.M. Nemeth, T.I. Welsh, J.M. Thomas, J.M. Crum, A.B. Lichtenwalner, A.M. Fadley, G. Zavala, E.C. Homes, and J.D. Brown. 2014. Avian oncogenesis inducted by lymphoproliferative disease virus: A neglected or emerging retroviral pathogen? *Virology* 450-451: 2-12.

____Allison, A.B., D.G. Mead, G.F. Palacios, R.B. Tesh, and E.C. Holmes. 2014. Gene duplication and phylogeography of North American members of the Hart Park serogroup of avian rhabdoviruses. *Virology* 448: 284-292.

____Barton, H.D., P. Rohani, D.E. Stallknecht, J. Brown, and J.M. Drake. 2013. Subtype diversity of reassortment potential for co-circulating avian influenza viruses at a diversity hotspot. *Journal of Animal Ecology* 83(3): 566-575.

____Brown, J.D., R. Poulson, and D.E. Stallknecht. 2014. Wild bird surveillance for avian influenza virus. Book chapter in *Animal Influenza Virus*, 2nd Edition, pp. 69-81. E. Spackman (Editor). Humana Press, Totowa, New Jersey.

____Brown, J.D., D.E. Stallknecht, C. Lebarbenchon, and D.E. Swayne. 2014. Survivability of Eurasian H5N1 highly pathogenic avian influenza virus in water varies between strains. *Avian Diseases* doi: 10.1637/10741-120513.

____Brown, V.L., J.M. Drake, H.D. Barton, D.E. Stallknecht, J.D. Brown, P. Rohani. 2014. Neutrality, cross-immunity and subtype dominance in avian influenza viruses. *PLoS One* 9(2): e88817.

____Cardona, C.J., D.A. Halvorson, J.D. Brown, and M.J. Pantin-Jackwood. 2014. Conducting influenza virus pathogenesis studies in avian species. Book chapter in *Animal Influenza Virus*, 2nd Edition, pp.169-183. E. Spackman (Editor). Humana Press, Totowa, New Jersey.

____Cottrell, W.O., M.K. Keel, J.W. Brooks, D.E. Mead, and J.E. Phillips. 2013. First report of clinical disease associated with canine distemper virus infection in a wild black bear (*Ursus Americana*). *Journal of Wildlife Diseases* 49(4): 1024-1027.

____Franca, M., E.W. Howerth, D. Carter, A. Byas, R. Poulson, C.L. Afonso, and D.E. Stallknecht. 2014. Co-infection of mallards with low virulence Newcastle disease virus and low pathogenic avian influenza virus. *Avian Pathology* 43(1): 96-104.

____Fritzen, C., E. Mosites, R.D. Applegate, S.R. Lii, J. Huang, M.J. Yabsley, L.R. Carpenter, J.R. Dunn, and A.C. Moncayo. 2014. Environmental investigation following the first human case of babesiosis in Tennessee. *Journal of Parasitology* 100(1): 106-109.

____Gonzales, A.V., S.M. Hernandez, S.L. Boone, E.K. Lipps, S. Shrestha, and M.J. Yabsley. 2013. Spatial, temporal, molecular, and intraspecific differences of haemoparasite infection and relevant physiological parameters of wild birds in Georgia, USA. *International Journal for Parasitology: Parasites and Wildlife* 2: 178-189.

____Handel, A., C. Lebarbenchon, D. Stallknecht, and P. Rohani. 2014. Trade-offs between and within scales: environmental persistence and within-host fitness of avian influenza viruses. *Proceedings of the Royal Society B* 281(1787).

____Keeler, S.P., M.S. Dalton, A.M. Cressler, R.D. Berghaus, and D.E. Stallknecht. 2014. Abiotic factors affecting persistence of avian influenza virus in surface water from waterfowl habitats. *Applied and Environmental Microbiology* 80(9): 2910-2917.

____Keeler, S.P., M.J. Yabsley, H.C. Adams, and S.M. Hernandez. 2014. A novel *Isospora* species (Apicomplexa: Eimeriidae) from warblers (Passeriformes: Paulidae) of Costa Rica. *Journal of Parasitology* 100(3): 302-304.

Continued…

____Kistler, W.M., J.D. Brown, A.B. Allison, N.M. Nemeth, and M.J. Yabsley. 2014. First report of *Angiostrongylus vasorum* and *Hepatozoon canis* in a red fox (*Vulpes vulpes*) from West Virginia, USA. *Veterinary Parasitology* 200(1-2): 216-220.

____Kjos, S.A., P.L. Marcet, M.J. Yabsley, U. Kitron, K.F. Snowden, K.S. Logan, J.C. Barnes, and E.M. Dotson. 2013. Identification of bloodmeal sources and *Trypanosoma cruzi* infection in triatomine bugs (Hemiptera: Reduviidae) from residential settings in Texas. *Journal of Medical Entomology* 50(5): 1126-1139.

____Lebarbenchon, C., J.D. Brown, and D.E. Stallknecht. 2013. Evolution of influenza A virus H7 and N9 subtypes, Eastern Asia. *Emerging Infectious Diseases* 19(10): 1635-1638.

____Lebarbenchon C., M. Patin-Jackwood, W.M. Kistler, M.P. Luttrell, E. Spackman, D.E. Stallknecht, and J.D. Brown. 2013. Evaluation of a commercial enzyme-linked immunosorbent assay for detection of antibodies against the H5 subtype of influenza A virus in waterfowl. *Influenza and Other Respiratory Viruses* 7(6): 1237-1240.

____Lebarbenchon, C., R. Poulson, K. Shannon, J. Slagter, M.J. Slusher, B.R. Wilcox, J. Berdeen, G.A. Knutsen, C.J. Cardona, and D.E. Stallknecht. 2013. Isolation of influenza A viruses from wild ducks and feathers in Minnesota. *Avian Diseases* 57(3): 677-680.

____Levine, R.S., D.G. Mead, and U.D. Kitron. 2013. Limited spillover to humans from West Nile virus viremic birds in Atlanta, Georgia. *Vector-Borne Zoonotic Diseases* 13(11): 812-817.

____Lund, A., J. McMillan, R. Kelly, S. Jabbarzadeh, D.G. Mead, T.R. Burkot, U. Kitron, and G.M. Vazquez Prokopec. 2014. Long term impacts of combined sewer overflow remediation on water quality and population dynamics of *Culex quinquefasciatus*, the main urban West Nile virus vector in Atlanta, Georgia. *Environmental Research* 129: 20-26.

____Magenwirth, J., N.M. Nemeth, M.J. Yabsley, B.A. Munk, M.K. Keel, and J.D. Brown. 2013. Infestation of two brown-headed cowbirds (*Molothrus ater*) with hunchback mites (*Harpirhynchus quasimodo*). *Avian Diseases* 57(4): 818-821.

____Mascarelli, P.E., M.K. Keel, M.J. Yabsley, L.A. Last, E.B. Breitschwerdt, and R.G. Maggi. 2014. Hemotropic mycoplasmas in little brown bats (*Myotis lucifugus*). *Parasites and Vectors* 7:117.

____McGuire, J.L., E.A. Miller, T.M. Norton, B.L. Raphael, J.S. Spratt, and M.J. Yabsley. 2013. Intestinal parasites of the gopher tortoise (*Gopherus polyphemus*) from eight populations in Georgia. *Parasitology Research* 112(12): 4205-4210.

____Mehrkens, L.R., L.A. Shender, M.J. Yabsley, B.C. Shock, F.A. Chinchilla, J. Suarez, and K.V.K. Gilardi. 2013. White-nosed coatis (*Nasua narica*) are a potential reservoir of *Trypanosoma cruzi* and other potentially zoonotic pathogens in Monteverde, Costa Rica. *Journal of Wildlife Diseases* 49(4): 1014-1018.

____Mercurio, A.D., S.M. Hernandez, J.C. Maerz, M.J. Yabsley, A.E. Ellis, A.L. Coleman, L.M. Shelnutt, J.R. Fischer, and S.B. Wilde. 2014. Experimental feeding of *Hydrilla verticillata* colonized by *Stigonematales cyanobacteria* induces vacuolar myelinopathy in painted turtles. (*Chrysemis picta*). *Plos One* 9(4): e93295.

____Munk, B.A., E. Garrison, B. Clemons, and M. Keel. 2014. Antleroma in a free-ranging white-tailed deer (*Odocoileus virginianus*). *Veterinary Pathology* doi: *10.1177/ 0300985814528216.*

____Murray, H.L., M.J. Yabsley, M.K. Keel, E.J.B. Manning, T.J. Wilmers, and J.L. Corn. 2014. Persistence of *Mycobacterium avium* subspecies *paratuberculosis* in endangered Florida Key deer and Key deer habitat. *Journal of Wildlife Diseases* 52(2): 349-353.

Continued…

____Nemeth, N.M., M.G. Ruder, R.W. Gerhold, J.D. Brown, B.A. Munk, P.T. Oesterle, S.V. Kubiski, and M.K. Keel. 2013. Demodectic mange, dermatophilosis, and other parasitic and bacterial dermatologic diseases in free-ranging white-tailed deer (*Odocoileus virginianus*) in the United States, 1975-2012. *Veterinary Pathology* 51(3): 633-640.

____Pepin, K.M., E. Spackman, J.D. Brown, K.L. Pabilonia, L.P. Garber, J.T. Weaver, D.A. Kennedy, K.A. Patyk, K.P. Huyvaert, R.S. Miller, A.B. Franklin, K. Pedersen, T.L. Bogich, P. Rohani, S.A. Shriner, C.T. Webb, and S. Riley. 2014. Using quantitative disease dynamics as a tool for guiding response to avian influenza in poultry in the United States of America. *Preventive Veterinary Medicine* 113(4): 376-397.

____Phillips, J.E., D.E. Stallknecht, T.A. Perkins, N.S. McClure, and D.G. Mead. 2014. Evolutionary dynamics of West Nile virus in Georgia, 2001-2011. *Virus Genes* doi: 10.1007/s11262-014-10610.

____Ramey, A.M., R.L. Poulson, A.S. Gonzalez-Reiche, D.R. Perez, D.E. Stallknecht, and J.D. Brown. 2014. Genomic characterization of H14 subtype influenza A viruses in new world waterfowl and experimental infectivity in mallards (*Anas platyrhynchos*). *PLoS One* 9(5): e95620.

____Ramey, A.M., R.L. Poulson, A.S. Gonzalez-Reiche, B.R. Wilcox, P. Walther, P. Link, D.L. Carter, G.M. Newsome, M.L. Muller, R.D. Berghaus, D.R. Perez, J.S. Hall, and D.E. Stallknecht. 2014. Evidence of seasonal patterns in the relative abundance of avian influenza virus subtypes in blue-winged teal (*Anas discors*). *Journal of Wildlife Diseases* dio: 10.7589/2013-09-232.

____Ramey, A.M., E. Spackman, J. Yeh, G. Fujita, K. Konishi, K. Uchida, J.A. Reed, B.R. Wilcox, J.D. Brown, and D.E. Stallknecht. 2013. Antibodies to H5 subtype avian influenza virus and Japanese encephalitis virus in northern pintails (*Anas acuta*) sampled in Japan. *Japanese Journal of Veterinary Research* 61(3): 117-123.

____Ramey, A.M., P. Walther, P. Link, R.L. Poulson, B.R. Wilcox, G. Newsome, E. Spackman, J.D. Brown, and D.E. Stallknecht. 2014. Optimizing surveillance for South American origin influenza A viruses along the United States Gulf Coast through genomic characterization of isolates from blue-winged teal (*Anas discors*). *Transboundary and Emerging Diseases* doi: 10.1111/tbed.12244.

____Roellig D.M., L.A. Gomez-Puerta, D.G. Mead, J. Pinto, J. Ancca-Juarez, M. Calderon, C. Bern, R.H. Gilman, V.A. Cama; Chagas Disease Workgroup in Arequipa. 2013. Hemi-nested PCR and RFLP methodologies for identifying blood meals of the Chagas disease vector, *Triatoma infestans*. *PLoS One*: 8(9):e74713.

____Santamaria, A., J.E. Calzada, A. Saldana, M.J. Yabsley, and N.L. Gottdenker. 2014. Molecular diagnosis and species identification of *Ehrlichia* and *Anaplasma* infections in dogs from Panama, Central America. *Vector-Borne and Zoonotic Diseases* 14(5): 368-370.

____Shock, B.C., A. Moncayo, S. Cohen, P.K. Williamson, and M.J. Yabsley. 2014. Diversity of piroplasms detected in blood-fed and questing ticks from several states in the United States. *Tick and Tick-Borne Diseases* 5(4): 377-380.

____Williams, B.M., A. Berentsen, B.C. Shock, M. Teixiera, M.R. Dunbar, M.S. Becker, and M.J. Yabsley. 2014. Prevalence and diversity of *Babesia, Hepatozoon, Ehrilichia*, and *Bartonella* in wild and domestic carnivores from Zambia, Africa. *Parasitology Research* 113(3): 911-918.

PLEASE SEND REPRINTS MARKED TO:

NAME _____

E-MAIL _____

ADDRESS _____

_____

CITY _____

STATE_____ZIP_____

ELECTRONIC COPY ☐

HARD COPY ☐

SCWDS BRIEFS

Southeastern Cooperative Wildlife Disease Study
College of Veterinary Medicine
The University of Georgia
Athens, Georgia 30602-4393

Nonprofit Organization
U.S. Postage
PAID
Athens, Georgia
Permit No. 11

RETURN SERVICE REQUESTED

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

Information on SCWDS and recent back issues of the *SCWDS BRIEFS* can be accessed on the internet at www.scwds.org. If you prefer to read the BRIEFS online, just send an email to Jeanenne Brewton (brewton@uga.edu) or Michael Yabsley (myabsley@uga.edu) and you will be informed each quarter when the latest issue is available.