

# 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 32

July 2016

Number 2

## **Was The Eurasian H5 HPAI Invasion Successful?**

During the fall of 2014, a highly pathogenic avian influenza (HPAI) virus was detected in North America. This Eurasian clade 2.3.4.4 H5N8 virus quickly spread through wild waterfowl populations in the Pacific Flyway and reassorted with North American influenza viruses creating HPAI H5N2 and H5N1 subtypes. By March 2015, HPAI H5N2 viruses were detected in the Mississippi Flyway and the largest HPAI outbreak ever recorded in poultry in the United States was gaining momentum. This outbreak affected more than 48,000,000 birds in 248 commercial and backyard poultry flocks at a cost exceeding \$5 billion.

This was the first time that HPAI viruses were detected in wild birds in North America, and wild birds certainly played a part in the dissemination of these viruses within and perhaps to North America. The long-term significance of this was unknown and with the eradication of these HPAI viruses from domestic poultry in North America by June 2015, the possibility of a wild bird reservoir was a major concern. This question recently was addressed in a paper in *Proceedings of the National Academy of Sciences (PNAS)* entitled "The Enigma of the Apparent Disappearance of Eurasian Highly Pathogenic H5 clade 2.3.4.4 Influenza A Viruses in North American Waterfowl." (<http://www.pnas.org/content/early/2016/07/19/1608853113.full>)

The publication describes a collaborative study between researchers at SCWDS, Saint Jude Children's Research Hospital, The Ohio State University, and the many agencies and wildlife scientists who work with us. In this study, results were evaluated from over 100,000 wild birds sampled from 1973 through 2013 and more than 23,000 birds tested before, during,

and after the 2014-2015 outbreak. There was no evidence that HPAI viruses were present in North American wild birds, and this is consistent with all published literature. There also was no evidence that these viruses have persisted in wild birds following successful eradication in domestic poultry. These results are consistent with surveillance conducted as part of the Interagency Strategic Surveillance Plan for Avian Influenza in Migratory Birds (USDA, USFWS, USGS) in which more than 45,000 birds have been tested since June 2015.

These results are extremely encouraging, but they are not conclusive, and hence the use of "Apparent" in the title. However, the results do indicate that if these viruses have persisted in North American wild birds, the prevalence is extremely low and the current risk to poultry appears to be nonexistent or negligible. However, the most important message in this publication relates to our lack of understanding of the mechanisms that allow or prevent the successful introduction of "new" influenza viruses into North America. Wild birds, especially ducks, are infected annually with multiple influenza virus subtypes of low pathogenicity (LPAI), but we currently do not understand how subtype diversity is maintained, if and how these subtypes compete, or if different subtypes or strains are equally fit to be maintained in this complex system. Historically, Eurasian viruses have had limited success in establishing in North American wild bird populations, but we currently do not understand why.

Support for wild bird surveillance is readily available when outbreaks occur, but we generally fail to see the value of such work between outbreaks. Understanding the mechanisms behind potential introductions and the outcomes of such introductions can only be accomplished with long-term research before,

Continued...

during, and after such events. This is a global problem and the need to understand these events and relationships is evident by: 1) an increasing diversity of LPAI and HPAI viruses becoming endemic in domestic poultry worldwide; 2) increased documentation of human infections (some fatal) associated with these poultry viruses; and 3) the ability of some of these viruses to infect and move with migratory wild birds. It is possible that the events of 2014-2015 represent a vision of the future that should not be ignored.

(Prepared by Dave Stallknecht)

### **CWD Research Update 2016**

Chronic wasting disease (CWD) continues to challenge wildlife managers for multiple reasons; however, it is the complex interaction of the agent-host-environmental factors that greatly complicates CWD epidemiology and confounds its management. Numerous basic and applied research projects have been completed or are underway in order to better understand this growing wildlife health challenge. Here we provide a few of the many recent developments in CWD and prion research.

Prior to the confirmation of CWD in a reindeer (*Rangifer tarandus*) from Norway earlier this year, two studies investigated the susceptibility of this species to CWD. In one study, groups of reindeer fawns were injected intracranially with CWD inoculum from affected white-tailed deer (WTD), mule deer, or elk. Two years after inoculation, six naïve reindeer were housed with inoculated reindeer, or in pens adjacent to them. The CWD prion was detected in 5/6 of the sentinel reindeer, two of which developed clinical disease, suggesting reindeer are susceptible to CWD through natural transmission routes. These results were presented by Moore et al. at the 2015 meeting of the American College of Veterinary Pathologists [http://www.ars.usda.gov/research/publications/publications.htm?SEQ\\_NO\\_115=317903](http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=317903).

The results above confirmed an earlier study by Mitchell et al. that demonstrated reindeer susceptibility to CWD following oral inoculation (<http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0039055>). In this study, six

reindeer were inoculated orally with brain tissue from CWD-affected WTD or elk. Two of three animals exposed to the WTD inoculum developed clinical disease and had detectable prions in tissues. The third reindeer exposed to WTD brain, and the three reindeer challenged with the elk inoculum, did not develop CWD or accumulate detectable prions in tissues. The results of these studies and the recent wild reindeer case in Norway have potentially enormous wildlife management, cultural, and economic consequences. Numerous concerns exist about the impacts of CWD in this species, including ongoing conservation concerns over declining wild populations, the high cultural value of the species to circumpolar aboriginal peoples, and the potential for migratory reindeer populations to facilitate spread of the disease.

Dr. Mary Wood, a wildlife veterinarian with the Wyoming Game and Fish (WGF) Department, presented preliminary findings of a three-year study evaluating an experimental CWD vaccine in elk during a WGF Commission meeting last November. Thirty-eight elk were divided into vaccinated or control groups that were co-mingled and naturally exposed in CWD prion-contaminated enclosures. No protective effect was observed in vaccinated elk. In fact, vaccinated elk developed clinical disease more rapidly than non-vaccinated elk and survival time was shorter. One complication in the study was the potential exposure of elk to CWD prions when they were temporarily housed in a potentially contaminated environment prior to vaccination. The study has not yet been published, but Dr. Wood's presentation can be viewed at <https://www.youtube.com/watch?v=bnlk4kW3fFw>.

Potential transmission modes, including the role of environmental contamination in CWD epidemiology, have been investigated in numerous studies. Henderson et al. recently quantified urinary and salivary shedding of prions by CWD-exposed WTD using a new prion detection technique (real-time quaking-induced conversion) that appears to have greater sensitivity than other techniques <https://jvi.asm.org/content/early/2015/06/26/JVI.01118-15>. White-tailed deer were exposed to CWD orally, by aerosol, or by contact with

fomites, and saliva and urine samples were collected from each animal at intervals of three months or less. Results varied but prions were detected in saliva as early as three months and in urine as soon as six months post exposure. Once detected, prion shedding persisted throughout the course of infection. Saliva was a more consistent route of detectable shedding, with CWD prion detected in about 50% of samples collected from deer exposed by aerosol or oral routes. Researchers also found little difference in shedding patterns between the more susceptible 96G/G genotype and the more “resistant” 96G/S genotypes. (It should be noted that no WTD, mule deer, or elk have been found to be completely resistant to CWD, although some genotypes are associated with markedly prolonged incubation periods prior to development of clinical signs.) All deer were monitored throughout the study by CWD testing of tonsil and recto-anal mucosa-associated lymphoid tissue (RAMALT) biopsies, and all deer exposed by oral or aerosol routes were positive by one or both tests by nine months post exposure. Based on their findings, the researchers were able to predict the magnitude of potentially infectious prions shed in the saliva and urine and concluded the infectivity of saliva was approximately tenfold that of urine. For instance, they estimated the LD<sub>50</sub> for cervidized transgenic mice to be contained in 1 mL of saliva or 10 mL of urine from an infected deer: a 100-kg deer will produce about 1 liter of urine daily, which would contain an estimated 100 cervidized mice LD<sub>50</sub>. These findings improve our understanding of the degree to which a CWD-infected deer may contaminate the environment over the course of infection, which may span months or years.

A recent study (Pritzkow et al. *Cell Reports* 11: 1168-1175, 2015) demonstrated that plants can bind, retain, uptake, and transport prions in an experimental setting. Wheat grass roots and leaves that were exposed to prion-positive brain homogenate efficiently bound prions (including CWD prions). When these CWD-contaminated plants were fed to naïve Syrian hamsters, the hamsters developed disease. The researchers also found that prion sprayed on the surface of wheat grass remained attached to the leaves for at least 49 days. Finally, using barley grass

grown in soil contaminated with prion-infected brain homogenate, it was demonstrated that small amounts of prion can be detected in the stems, and rarely in leaves, suggesting the plant was able to take prions up from the soil. This study suggests that plants could play a role in the transmission of CWD; however, additional research is needed to better understand this potential relationship.

The potential human susceptibility to CWD has been investigated for more than twenty years. Although long-term monitoring of people consuming venison in CWD-endemic areas has not identified human cases, laboratory research into the zoonotic potential of CWD remains active. Genetically modified laboratory mice (i.e., transgenic mice) commonly are used for research as animal models of human diseases, because their genomes have been manipulated to contain human genes and express human proteins. These are referred to as “humanized” transgenic mice, as they have been engineered to express the human prion protein. “Cervidized” strains of transgenic mice also are used for CWD research. Recently, Qing et al. intracerebrally inoculated “humanized” transgenic mice with CWD material to examine the potential transmissibility of CWD prions to humans: 2 of 140 inoculated mice expressed CWD prion in the spleen, but not in the brain. However, secondary passage of CWD prion-positive “humanized” mouse spleen led to efficient CWD transmission to “humanized” and “cervidized” transgenic mice with the development of clinical disease and pathological changes. The authors also reported that, “*a recent bioassay with natural CWD isolates in a new humanized transgenic mouse line led to clinical prion infection in 2 out of 20 mice.*” The results have not yet been published, but the authors concluded “*that CWD prion has the potential to infect human central nervous system and peripheral lymphoid tissues and that there might be asymptomatic human carriers of CWD infection.*” Certainly, more studies are needed and hunters should always use common sense and follow CDC recommendations to minimize risk of exposure to CWD. The preliminary findings of the study were presented at Prion 2015 in Ft. Collins, CO, and can be found as Abstract O18 on page 19 <https://prion2015.files>.

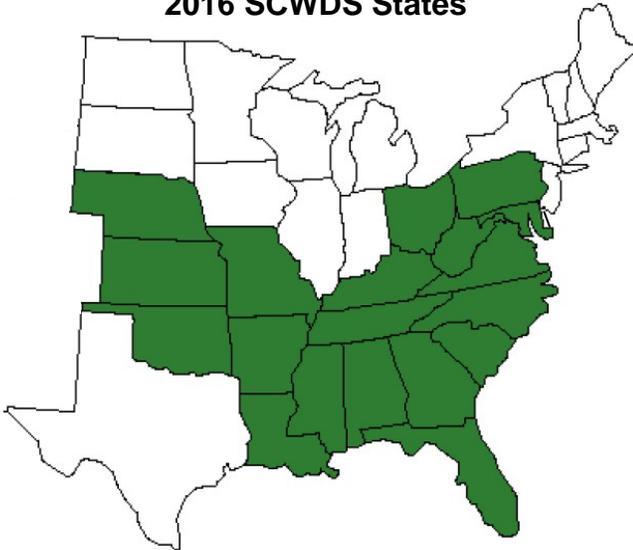
[wordpress.com/2015/05/programguide1.pdf](http://wordpress.com/2015/05/programguide1.pdf).

(Prepared by Kiley Cameron, Heather Fenton, and Mark Ruder)

## Nebraska Joins SCWDS

We are very pleased to announce that the Nebraska Game and Parks Commission (NGPC) is the newest member of the Southeastern Cooperative Wildlife Disease Study. The NGPC joined SCWDS earlier this year, bringing the total number of member states to 19. Over the years, we have worked with Nebraska biologists as they confronted wildlife mortality events, including a massive hemorrhagic disease outbreak in 2012, and we are proud of the confidence they have shown in SCWDS. We look forward to assisting NGPC biologists, managers, and administrators with the management of healthy wildlife populations.

### 2016 SCWDS States



## A Note to Our Readers

We thank you for your sustained interest in our quarterly newsletter, the SCWDS BRIEFS. 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 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: SCWDS, College of Veterinary Medicine, University of Georgia, Athens, GA 30602 or email at [brewton@uga.edu](mailto:brewton@uga.edu).

\_\_\_\_Bucukovski, J., N. Latorre-Margalef, D.E. Stallknecht, and B.L. Miller. 2015. A multiplex label-free approach to avian influenza surveillance and serology. *PLoS One* 10(8): e0134484.

\_\_\_\_Chambouvet, A., D.J. Gower, M. Jirku, M.J. Yabsley, A.K. Davis, G. Leonard, F. Maguire, T.M. Doherty-Bone, G.B. Bittencourt-Silva, M. Wilkinson, T.A. Richards. 2015. Cryptic infection of a broad taxonomic and geographic diversity of tadpoles by *Perkinsea protists*. *Proceedings of the National Academy of Sciences of the United States of America* 112(34): E4743–E4751.

\_\_\_\_Elsmo, E.J., A.B. Allison, and J.D. Brown. 2016. A retrospective study of causes of skin lesions in wild turkeys (*Meleagris gallopavo*) in

the eastern United States, 1975-2013. *Journal of Wildlife Diseases* doi: 10.7589/2015-05-129.

\_\_\_\_Fenton, H., and M.J. Yabsley. 2016. Wildlife disease investigations: process and cases. *North American Veterinary Conference Proceedings*. Chapter 533.

\_\_\_\_Fernandes, A., H. Fenton, S. Martinson, M. Desmarchelier, and S. Ferrell. 2015. Absolute polycythemia in a bald eagle (*Haliaeetus leucocephalus*). *Journal of Zoo and Wildlife Medicine* 45: 958-960.

\_\_\_\_Gleim, E.R., L.E. Garrison, M.S. Vello, M.Y. Savage, G. Lopez, R.D. Bergaus, and M.J. Yabsley. 2016. Factors associated with tick bites and pathogen prevalence in ticks parasitizing humans in Georgia, USA. *Parasites and Vectors* 9(1): 125.

\_\_\_\_Gonzalez-Astudillo, V., S.M. Hernandez, M.J. Yabsley, D.G. Mead, M.K. Keel, B.A. Munk, J.R. Fischer, M.G. Ruder, J.D. Brown, V.E. Peters, and N.M. Nemeth. 2016. Mortality of selected avian orders submitted to a wildlife diagnostic laboratory (Southeastern Cooperative Wildlife Disease Study, USA): a 36-year retrospective analysis. *Journal of Wildlife Diseases* doi: 10.7589/2015-05-117.

\_\_\_\_Guinn, K., A. Fojtik, N. Davis-Fields, R.L. Poulson, S. Krauss, R.G. Webster, and D.E. Stallknecht. 2016. Antibodies to influenza A viruses in gulls at Delaware Bay, USA. *Avian Diseases* 60: 341-345.

\_\_\_\_Kaplan, B.S., M. Russier, T. Jeevan, B. Marathe, J. Turner, E.A. Govorkova, C.J. Russell, M. Kim-Torchetti, Y.K. Choi, I. Brown, T. Saito, T. Harder, D.E. Stallknecht, S. Krauss, and R.J. Webby. 2016. Novel highly pathogenic avian A(H5N2) and A(H5N8) influenza viruses of Clade 2.3.4.4 from North America have limited capacity for replication and transmission in mammals. *M. Sphere* 1(2): pii: e00003-16; doi: 10.1128/mSphere.00003-16.

\_\_\_\_Krauss, S., D.E. Stallknecht, R.D. Slemmons, A.S. Bowman, R.L. Poulson, J.M. Nolting, J.P. Knowles, and R.G. Webster. 2016. The

enigma of the apparent disappearance of Eurasian highly pathogenic H5 clade 2.3.4.4 influenza A viruses in North American waterfowl. *Proceedings of the National Academy of Sciences of the United States of America* doi: 10.1073/pnas.1608853113.

\_\_\_\_Latorre-Margalef, N, A.M. Ramey, A. Fojtik, and D.E. Stallknecht. 2015. Serologic evidence of influenza A (H14) virus introduction into North America. *Emerging Infectious Diseases* 21(12): 2257-2259.

\_\_\_\_Levy, B., C. Collins, S. Lenhart, M. Madden, J. Corn, R. Salinas, and W. Stiver. 2016. A metapopulation model for feral hogs in Great Smoky Mountains National Park. *Natural Resource Modeling* 29(1): 71-97.

\_\_\_\_Loftis, A.D., P.J. Kelly, C.D. Paddock, K. Blount, J.W. Johnson, E.R. Gleim, M.J. Yabsley, M.L. Levin, and L. Beati. 2016. Panola Mountain *Ehrlichia* in *Amblyomma maculatum* from the United States and *Amblyomma variegatum* from the Caribbean and Africa. *Journal of Medical Entomology* pii: tjv240.

\_\_\_\_Martinsen, E.S., N. McInerney, H. Brightman, K. Ferebee, T. Walsh, W. McShea, T.D. Forrester, L. Ware, P.H. Joyner, S.L. Perkins, E.K. Latch, M.J. Yabsley, J.J. Schall, and R.C. Fleischer. 2016. Hidden in plain sight: Cryptic and endemic malaria parasites in North American white-tailed deer (*Odocoileus virginianus*). *Science Advances* 2: e1501486: 1-7.

\_\_\_\_Maxted, A., H.P. Sitters, M.P. Luttrell, A.D. Dey, K.S. Kalasz, L.J. Niles, and D.E. Stallknecht. 2016. Spring migration stopover ecology of avian influenza virus shorebird hosts at Delaware Bay. *Avian Diseases* 60: 394-405.

\_\_\_\_Mesquita, L.P., M.H. Diaz, E.W. Howerth, D.E. Stallknecht, R. Noblet, E.W. Gray, and D.G. Mead. 2016. Pathogenesis of vesicular stomatitis New Jersey virus infection in deer mice (*Peromyscus maniculatus*) transmitted by black flies (*Simulium vittatum*). *Veterinary Pathology* pii: 0300985816653172.

- \_\_\_\_Park, A.W., C. Cleveland, T.A. Dallas, and J.L. Corn. 2015. Vector species richness increases hemorrhagic disease prevalence through functional diversity modulating the duration of seasonal transmission. *Parasitology* 43(7): 874-879.
- \_\_\_\_Poulson, R.L., S.M. Tompkins, R.D. Berghaus, J.D. Brown, and D.E. Stallknecht. 2016. Environmental stability of swine and human pandemic influenza viruses in water under variable conditions of temperature, salinity, and pH. *Applied and Environmental Microbiology* 82(13): 3721-3726.
- \_\_\_\_Ramey, A.M., J.M. Pearce, A.B. Reeves, R.L. Poulson, J. Dobson, B. Lefferts, K. Spragens, and D.E. Stallknecht. 2016. Surveillance for Eurasian-origin and intercontinental reassortant highly pathogenic influenza A viruses in Alaska, spring and summer 2015. *Virology Journal* 3(1): 55.
- \_\_\_\_Ramey, A.M., J.A. Reed, P. Walther, P. Link, J.A. Schmutz, D.C. Douglas, D.E. Stallknecht, and C. Soos. 2016. Evidence for the exchange of blood parasites between North America and the Neotropics in blue-winged teal (*Anas discors*). *Parasitology Research* doi: 10.1007/s00436-016-5159-2.
- \_\_\_\_Ramey, A.M., A.B. Reeves, R.L. Poulson, D.L. Carter, N. Davis-Fields, and D.E. Stallknecht. 2016. Genome sequence of a novel H14N7 subtype influenza A virus isolated from a blue-winged teal (*Anas discors*) harvested in Texas, USA. *Genome Announcements* doi: 10.1128/genomeA.00520-16.
- \_\_\_\_Ramey, A.M., A.B. Reeves, R.L. Poulson, J. Wasley, D. Esler, and D.E. Stallknecht. 2015. Sampling of sea ducks for influenza A viruses in Alaska during winter provides lack of epidemiologic peak of infection. *Journal of Wildlife Diseases* 51(4): 938-941.
- \_\_\_\_Ramey, A.M., M.K. Torchetti, R.L. Poulson, A.B. Reeves, P. Link, P. Walther, C. Lebarbenchon, and D.E. Stallknecht. 2016. Evidence for wild waterfowl origin of H7N3 influenza A virus detected in captive-reared New Jersey pheasants. *Archives of Virology* doi: 10.1007/s00705-016-2947-z.
- \_\_\_\_Ramey, A.M., P. Walther, P. Link, R.L. Poulson, B.R. Wilcox, G. Newsome, E. Spackman, J.D. Brown, and D.E. Stallknecht. 2016. 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* 63: 194-202.
- \_\_\_\_Reeves, A.B., R.L. Poulson, D. Muzyka, H. Ogawa, K. Imai, V.N. Bui, J.S. Hall, D.E. Stallknecht, and A.M. Ramey. 2016. Limited evidence of intercontinental dispersal of avian paramyxovirus serotype 4 by migratory birds. *Infection, Genetics and Evolution* 40: 104-108.
- \_\_\_\_Robinson, G.L., G.L. Mills, A.H. Lindell, S.H. Schweitzer, and S.M. Hernandez. 2015. Exposure to mercury and Aroclor 1268 congeners in least terns (*Sternula antillarum*) in coastal Georgia, USA. *Environmental Science-Processes and Impacts* 17(8): 1424-1432.
- \_\_\_\_Rothermel, B.B., D.L. Miller, E.R. Travis, J.L. McGuire, J.B. Jensen, and M.J. Yabsley. 2016. Disease dynamics of red-spotted newts and their anuran prey in a montane pond community. *Diseases of Aquatic Organisms* 118(2): 113-127.
- \_\_\_\_Ruder, M.G., D.G. Mead, D.E. Stallknecht, M. Kedmi, E. Klement, J.D. Brown, D.L. Carter, and E.W. Howerth. 2015. Experimental infection of Holstein cows and calves with EHDV-7 and preliminary evaluation of different inoculation methods. *Veterinary Italiana* 51(4): 289-299.
- \_\_\_\_Ruder, M.G., D.E. Stallknecht, A.B. Allison, D.G. Mead, D.L. Carter, and E.W. Howerth. 2015. Host and potential vector susceptibility to an emerging orbivirus in the United States: epizootic hemorrhagic disease virus serotype 6. *Veterinary Pathology* 53(3): 574-584.
- \_\_\_\_Sapp, S.G.H., S.B. Weinstein, C.S. McMahan, and M.J. Yabsley. 2016. Variable

infection dynamics in four *Peromyscus* species following experimental inoculation with *Baylisascaris procyonis*. *Journal of Parasitology* [http:// dx.doi.org/10.1645/16-57](http://dx.doi.org/10.1645/16-57).

\_\_\_\_Sharma, P., D.E. Stallknecht, M.D. Murphy, and E.W. Howerth. 2015. Expression of interleukin-1 beta and interleukin-6 in white-tailed deer infected with epizootic hemorrhagic disease virus. *Veterinary Italiana* 54(4): 283-288.

\_\_\_\_Sikes, R.S., J.A. Bryan, D. Byman, B. Danielson, J. Eggleston, M. Gannon, W. Gannon, D. Hale, B. Jesner, D. Odell, L.E. Olsen, R.D. Stevens, T.A. Thompson, R. Timm, S. Trehwhitt, and J.R. Willoughby. 2016. Guidelines of the American Society of Mammalogists for the use of wild mammals in research and education. *Journal of Mammalogy* 97(3): 663-688.

\_\_\_\_Springer, Y.P., D. Hoekman, P.T.J. Johnson, P.A. Duffy, B.F. Allan, B.R. Amman, C.M. Barker, R. Barrera, C.B. Beard, L. Beati, M. Begon, M.S. Blackmore, W.E. Bradshaw, D. Brisson, C.H. Calisher, J.E. Childs, M.A. Diuk-Wasser, R.J. Douglass, R. Eisen, D.H. Foley, J.E. Foley, H.D. Gaff, S.L. Gardner, H.S. Ginsberg, G.E. Glass, S.A. Hamer, M.H. Hayden, B. Hjelle, C.M. Holzapfel, S.A. Juliano, L.D. Kramer, A.M. Kuenzi, S.L. LaDeau, T.P. Livdahl, J.N. Mills, C.G. Moore, S. Morand, R.S. Masci, N.H. Ogden, R.S. Ostfeld, R.R. Parmenter, J. Piesman, W.K. Reisen, H.M. Savage, D.E. Sonenshine, A. Swei, and M.J. Yabsley. 2016. Vector and pathogen sampling designs for the National Ecological Observatory Network (NEON). *Ecosphere* doi: 10.1002/ecs2.1271.

\_\_\_\_Thomas, J.M., J.E. Phillips, E.M. Bunting, M.J. Yabsley, and J.D. Brown. 2015. Molecular surveillance for lymphoproliferative disease virus in wild turkeys (*Meleagris gallopavo*) from the eastern United States. *Plos One* 10(4): e0122644.

\_\_\_\_White, C.L., M.J. Forzan, A.P. Pessier, M.C. Allender, J.R. Ballard, A. Catenazzi, H. Fenton, A. Martel, F. Pasmans, D.L. Miller, R. Ossiboff, K.L.D. Richgels, and J.L. Kerby.

2016. Amphibian: A case definition and diagnostic criteria for *Batrachochytrium salamandrivorans* chytridiomycosis. *Herpetological Review* 47(2): 207-209.

\_\_\_\_Wilson, W.C., N.N. Gaudreault, D.C. Jasperson, D.J. Johnson, E.N. Ostlund, C.L. Chase, M.G. Ruder, and D.E. Stallknecht. 2015. Molecular evolution of American field strains of bluetongue and epizootic hemorrhagic disease viruses. *Veterinary Italiana* 51(4): 269-273.

\_\_\_\_Wilson, W.C., M.G. Ruder, D. Jasperson, T.P. Smith, P. Naraghi-Arani, R. Lenhoff, D.E. Stallknecht, W.A. Valdivia-Granda, and D. Sheoran. 2016. Molecular evolution of epizootic hemorrhagic disease viruses in North America based on historical isolates using motif fingerprints. *Virus Genes* doi: 10.1007/s11262-016-1332-z.

\_\_\_\_Wong, J.K., B.R. Wilcox, A. Fojtik, R.L. Poulson, and D.E. Stallknecht. 2016. Antibodies to influenza A viruses in wintering snow geese (*Chen caerulescens*) in Texas. *Avian Diseases* 60: 337-340.

\_\_\_\_Yabsley, M.J., K. Bailey, and H.C. Adams. 2015. A novel species of *Eimeria* (Apicomplexa: Eimeriidae) from the mourning dove, *Zenaida macroura* (Columbiformes: Columbidae). *Comparative Parasitology* 82(2): 231-234.

**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](http://www.scwds.org). If you prefer to read the BRIEFS online, just send an email to Jeanenne Brewton ([brewton@uga.edu](mailto:brewton@uga.edu)) or Michael Yabsley ([myabsley@uga.edu](mailto:myabsley@uga.edu)) and you will be informed each quarter when the latest issue is available.