Hiding in Plain Sight: The Asian Longhorned Tick

It appears that *Haemaphysalis longicornis*, the Asian longhorned tick, is here to stay. This exotic Asian tick species first was confirmed in the United States in November 2017 following its collection from a domestic sheep and its caregiver in Hunterdon County, New Jersey (SCWDS BRIEFS, Vol. 33, No. 4). Tick surveillance was not performed over the winter when the tick was dormant, but surveys by SCWDS, Rutgers University, USDA-APHIS-Wildlife Services, and other organizations were initiated in April 2018 to determine if the tick had overwintered. The longhorned tick was detected in the environment in and around the sheep paddock in Hunterdon County, and it also was found on wild opossum, raccoon, and white-tailed deer collected in the area in April and May 2018. These findings confirmed that the tick had overwintered and is utilizing wildlife hosts.

As of August 31, 2018, *H. longicornis* has been confirmed on at least one type of host (wildlife, domestic animal, and/or human) in nine states (Arkansas, Connecticut, Maryland, New York, New Jersey, North Carolina, Pennsylvania, Virginia, and West Virginia). The list of confirmed hosts in the U.S. is growing and now includes cattle, domestic goats and sheep, horses, cats, dogs, white-tailed deer, opossums, raccoons, and humans. The list of affected states and hosts undoubtedly will grow in the coming weeks and months. SCWDS has developed a website with an interactive map of *H. longicornis* distribution that is updated weekly. The map can show reports by state, year, and host, and can be accessed at [https://plot.ly/dashboard/SCWDS:40/present#/](https://plot.ly/dashboard/SCWDS:40/present#/).

There are two native *Haemaphysalis* species in the United States: *H. leporispalustris* (rabbit tick) and *H. chordeilis* (bird tick). *Haemaphysalis chordeilis* is a poorly understood Nearctic species that is reported sporadically on passeriform and galliform birds and rarely on mammals (rodents, bovids, and cervids). In contrast, the rabbit tick is broadly distributed in the New World and commonly utilizes rabbits, other mammals, and ground-dwelling birds as hosts.

Over the past few months, it has become apparent that native *H. leporispalustris* ticks have complicated detection of the longhorned tick in the U.S. Re-examination of archived ticks indicates *H. longicornis* were misidentified as *H. leporispalustris* for a number of years. When specimens originally identified as *H. leporispalustris* following their 2010 collection from white-tailed deer in West Virginia were re-examined in 2018, the ticks were correctly identified as *H. longicornis*. This currently represents the first detection of *H. longicornis* in the U.S., and it has become clear that the introduction of *H. longicornis* is not recent: This exotic tick likely has been hiding in plain sight for quite some time. Many laboratories now are going back through their archived *H. leporispalustris* specimens to confirm their identification.

There are some life history traits of *H. longicornis* that enhance its invasive potential. It has a broad native and introduced geographic range, and nymphs and adults are able to enter diapause (suspected development) or overwinter in colder environments. Further, *H. longicornis* has a wide host range including humans, cattle, dogs, horses, and small ruminants as well as a wide variety of wild and feral species including carnivores, cervids, feral pigs, rodents, and...
several bird species. Finally, some populations of *H. longicornis* are capable of asexual reproduction (parthenogenesis), in which adult female ticks can produce fertile eggs without contact with male ticks. Therefore, a single female tick introduced to a new area can start a population.

Parthenogenetic specimens were introduced in Australia and New Zealand where they became the primary vector of *Theileria orientalis*, a cattle pathogen that has had a significant economic impact on New Zealand’s cattle industry. *Haemaphysalis longicornis* is a competent vector of numerous other human, domestic, and wild animal pathogens including other *Theileria* spp., *Anaplasm* spp., *Ehrlichia* spp., *Bartonella* spp., *Borrelia* spp., *Rickettsia japonica*, and several human viruses. Exactly how *H. longicornis* will interact with pathogens and hosts in North American ecosystems is an important question, and SCWDS, along with many other organizations, is actively engaged in research to better understand the situation.

In addition to this intensive effort on the ground, SCWDS is working closely with wildlife rehabilitation facilities in areas with confirmed *H. longicornis* presence in order to sample a diversity of wildlife species. We also are seeking help from our cooperative members with regional surveillance for this tick. Thus far, white-tailed deer have proven to be a good host and are the primary focus of our surveillance, but we also are seeking samples from elk and black bear and are asking biologists to opportunistically collect ticks from these species when available. This includes road-killed animals, nuisance removals, disease investigations, research projects, depredation permits, and any other opportunity to collect samples.

This summer we distributed dozens of tick collection kits to agency personnel in SCWDS member states. We have been receiving tick submissions and already have identified *H. longicornis* on white-tailed deer from several states. We encourage you to continue sampling ahead of hunting season, because the ability to detect this tick on animals will cease in some areas as colder months arrive and the ticks enter diapause. We will send more collection vials, data sheets, and labels to your agency as needed. It is our hope that this cooperative effort will provide valuable information regarding the distribution of this exotic tick. (Prepared by Angela Ives, Virginia-Maryland Regional College of Veterinary Medicine, Mark Ruder, and Michael Yabsley)

**Heartland Virus Studies at SCWDS**

Heartland virus (HRTV) is a phlebovirus suspected to be transmitted by the lone star tick, *Amblyomma americanum*. The virus first was recognized in 2009 as a cause of febrile disease with thrombocytopenia (low platelets) and leukopenia (low white blood cell count) in humans. To date, HRTV infections have been documented in 30 humans in nine states, mainly in the Midwestern and southern United States; one case was fatal. Antibodies against HRTV have been detected in a variety of domestic and wild species, including white-tailed deer, moose, raccoons, and coyotes, from regions with human cases. Experimental HRTV infections have been attempted in raccoons, goats, chickens, rabbits, hamsters, and immunosuppressed mice. None of these species developed detectable viremia or pathologic changes, with the exception of the immunosuppressed mice.

SCWDS is continuing to conduct surveillance for *H. longicornis*. Just a few months ago, our primary objectives were to determine if *H. longicornis* could overwinter in New Jersey and become established in wildlife. However, those questions were answered this spring, and we have now transitioned to evaluating the wildlife host range and geographic distribution of the tick. Our surveillance effort involves intensive localized surveys as well as a regional approach. SCWDS biologists, in cooperation with New Jersey Division of Fish and Wildlife, USDA-APHIS-Wildlife Services, and other agencies, conducted intensive surveys of mammalian and avian wildlife in May and July 2018 at New Jersey locations with confirmed *H. longicornis*.

**Figure 1.** Nymph (left) and adult female *H. longicornis* (James Gathany, CDC)

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To better understand the potential role of white-tailed deer in the epidemiology of HRTV, a group of researchers with SCWDS and the University of Georgia, College of Veterinary Medicine’s Department of Pathology experimentally inoculated five white-tailed deer fawns with HRTV and monitored them for clinical disease, viremia, virus shedding, and seroconversion. None of the animals showed clinical signs, and there was no detectable viremia or virus shedding. Two wild-caught fawns entered the study with pre-existing HRTV neutralizing antibodies, which are thought to be maternally-derived and to provide evidence that wild white-tailed deer in Georgia are exposed to HRTV. All animals showed minimal immune responses against HRTV.

This study indicates that white-tailed deer are unlikely to be a primary mammalian host for HRTV; however, they could play a role in virus transmission by harboring large numbers of ticks, because it appears that the virus is transmitted primarily from tick to tick by co-feeding (i.e. an infected tick feeding next to an uninfected tick transmits the virus via saliva).

To further examine the potential of using white-tailed deer as an indicator of HRTV circulation, and to determine whether HRTV exposure in white-tailed deer occurred prior to identification of the first human case in 2009, sera collected during SCWDS deer herd health evaluations between 2001 and 2015 were analyzed for HRTV neutralizing antibodies. Of 783 serum samples tested, 57 (7.3%) were positive for HRTV-neutralizing antibodies. Seropositive samples were more likely to come from deer with moderate to heavy tick burdens at necropsy. Seropositive samples were obtained from deer in six of the nine states where human cases of HRTV-associated disease have been documented (Arkansas, Georgia, Kentucky, North Carolina, Oklahoma, and Tennessee) as well as three states with no recognized human HRTV infections (Florida, Louisiana, and South Carolina). Seropositive samples were identified every year except 2004, indicating HRTV likely was circulating in affected regions prior to recognition of the first human case. Overall, this study indicates that white-tailed deer in the southeastern U.S. have been exposed to HRTV as early as 2001 and that the presence of seropositive animals correlates roughly with reported HRTV-associated disease in humans.

Further investigation of the role of white-tailed deer in harboring and spreading HRTV is likely to involve continued surveillance of populations where A. americanum is endemic. If seropositive animals are detected in a previously naïve region, or if increased numbers of seropositive animals are found, this may be an indicator to local public health professionals of the possibility of human infections. (Prepared by Lorelei Clarke, UGA Department of Pathology)

**Tick-Associated Red Meat Allergy**

When tick season is in full swing, many of us think about tick-borne pathogens and how to avoid them. But there is another reason to take appropriate precautions to avoid ticks that is not related to pathogens: Red Meat Allergy. Although considered rare, this allergy may develop in some people following a tick bite. The allergy is caused by hypersensitivity to “Alpha-Gal” (galactose-alpha-1, 3-galactose), a sugar molecule. While attached and feeding, a tick secretes compounds, such as vasodilators and anticoagulants, into the host to facilitate the feeding process. One of the compounds a tick may release is Alpha-Gal, and the immune system of a small percentage of people recognizes it as a foreign molecule and develops antibodies to it. If sensitized, people may then mount an allergic response to non-primate mammalian meat (beef, pork, lamb, venison) that contains Alpha-Gal. The reaction may be immediate or may take several hours to develop. The most common clinical signs are hives, shortness of breath, vomiting, and diarrhea. Severe reactions can involve multiple body systems and cause death.

The number of people suffering from this allergy is unknown due to the lack of a diagnosis, misdiagnosis, or a failure to report cases. However, numbers of cases appear to be increasing. The allergy first was diagnosed in the late 2000s and since then has been reported in numerous countries. Red Meat Allergy most often is associated with bites from the lone star tick (*Amblyomma americanum*) in the United States, *Ixodes ricinus* in Europe, and *Ixodes holocyclus* in Australia. It is not known if people fully recover from this allergy, and the severity of reactions in some individuals has decreased over time; however, additional tick bites apparently can cause a return of severe reactions.
Diagnosis can be complicated but a history of onset of an allergic reaction following ingestion of red meat is highly suggestive. The gold standard for confirming food allergies is a food trial, but this can be dangerous due to the unpredictable severity of the allergic reaction. Some physicians use a skin prick test and/or a basophil activation test to confirm an allergic reaction to Alpha-Gal, and there are commercial laboratories that have tests for antibodies against Alpha-Gal.

Treatment of tick-associated Red Meat Allergy is similar to those for other food allergies. Patients tend to respond well to anti-histamines and epinephrine. Once an allergy is confirmed or suspected, people should avoid the consumption of beef, pork, lamb, and venison. The current concern for public health is mostly localized to the eastern U.S. and especially in the Southeast where lone star tick densities are highest. However, the association of Alpha-Gal allergies in foreign countries due to other tick species suggests that bites from additional tick species in the U.S. could cause the allergy.

Researchers from Colorado and Wyoming recently published the results of a ten-year study on the susceptibility of beef cattle to CWD when challenged with an oral dose or co-housed in outdoor research facilities where CWD was enzootic. One group of calves was given a single oral dose of brain homogenate from CWD-infected mule deer and was monitored for a decade for clinical signs. Two other groups were co-housed in paddocks for a decade with deer or elk known to be infected with CWD, as well as with “sentinel” deer or elk that were initially uninfected, but later became infected, thus demonstrating CWD transmission within the paddocks. Clinical signs did not appear in any cattle during the study, and the CWD prion was not found in the central nervous system or lymphoid tissues when they were tested postmortem. These results differ from those of previous susceptibility studies of cattle in which the animals were intracerebrally inoculated with brain homogenate from elk, mule deer, or white-tailed deer with CWD. The authors of the recent study concluded that “the risks of CWD transmission to cattle following oral inoculation or after prolonged exposure to contaminated environments are low.” The paper can be accessed at http://www.jwildlifedis.org/doi/abs/10.7589/2017-12-299. The first author is Elizabeth Williams, who died with her husband Tom Thorne in a tragic accident in 2004. Tom and Beth were our mentors, colleagues, and friends and we still miss them.

Researchers at the University of Wisconsin recently published results of an investigation of mineral licks as environmental reservoirs of CWD prions. Soil, water, and fecal samples were collected from man-made and naturally occurring mineral licks in southern Wisconsin where CWD is enzootic in wild deer. The samples were analyzed by the protein misfolding cyclic amplification (PMCA) technique, which allows sensitive detection of prions in environmental samples. Of eleven sites sampled, seven were positive for prion protein in the soil. Water samples from four of the nine sites that contained water tested positive for prion protein. Six of ten fecal samples that were tested produced positive results. Because prion proteins are not easily destroyed and can persist in the environment for...
years, the authors concluded that “… mineral licks can serve as reservoirs of CWD prions and thus facilitate disease transmission. Furthermore, mineral licks attract livestock and other wildlife that also obtain mineral nutrients via soil and water consumption. Exposure to CWD prions at mineral licks provides potential for cross-species transmission to wildlife, domestic animals, and humans. Managing deer use of mineral licks warrants further consideration to help control outbreaks of CWD.” The full publication can be found at https://doi.org/10.1371/journal.pone.0196745.

Another field study, which was conducted by personnel with the Colorado Division of Parks and Wildlife and the National Park Service, evaluated a test and cull strategy for reducing CWD prevalence in a naturally infected mule deer herd in Estes Park, CO, and adjacent Rocky Mountain National Park. Tonsil biopsies were performed annually for five years on an estimated 48-68% of adult deer in the population, and animals that tested positive by immunohistochemistry were removed. Prevalence of CWD in males was lower during the last three years of the study than in the previous period; however, prevalence among females was comparable before and after culling. The authors indicated that “a more intensive and sustained effort or modified spatial approach might have reduced prevalence more consistently in both sexes” and stated that “elements of this approach could potentially be used to augment harvest-based disease management.” They also acknowledged that application of a test and cull strategy may be limited by cost and labor, property access, and animal tolerance to repeated capture. The publication is available at https://www.ncbi.nlm.nih.gov/pubmed/29667874.

Researchers with the National Institutes of Health’s Rocky Mountain Laboratories recently published results of a long-term study on CWD susceptibility of cynomolgus macaques (Macaca fasciculatus) that were inoculated orally or intracerebrally with brain homogenates from CWD-infected deer and elk (https://jvi.asm.org/content/early/2018/04/19/JVI.00550-18). Cynomolgus macaques are genetically similar to humans and previously have been used for prion disease research. In the present 13-year study, there was no clinical, pathological, or biochemical evidence that CWD was transmitted to macaques. These results are in contrast to preliminary results from a Canadian study that were presented at a scientific conference last year. In the Canadian study, which was ongoing and unpublished at the time, cynomolgus macaques had developed clinical disease and CWD prion accumulation was detected in central and peripheral nervous system tissues (SCWDS BRIEFS Vol. 33, No. 3). Differences in the experimental design, including the strains of the CWD inoculum, ages of the study animals and other variables, could account for the different results of the two studies; however, full assessment will be more feasible when the Canadian study is published following its completion. (Prepared by Claire Gossett, Mississippi State University College of Veterinary Medicine)

Avian Paramyxovirus-1, 2018

On May 18, 2018, the USDA-Animal and Plant Health Inspection Service announced that virulent (formerly ‘exotic’) Newcastle Disease (VND) was confirmed in a flock of backyard chickens in Los Angeles County, California. Since the initial case, VND has been confirmed in 136 additional backyard poultry flocks in four southern California counties. This is the first occurrence of VND in domestic poultry in the United States since 2003. Fortunately, there has been no spillover to the commercial poultry industry.

Virulent Newcastle Disease is a reportable disease that poses a significant risk to the U.S. poultry industry. The disease is highly contagious and pathogenic; birds may die before exhibiting clinical disease affecting the respiratory, digestive, and/or nervous systems. The economic significance can be enormous: birds can die from VND or be destroyed in disease eradication operations, and there may be substantial economic loss when other countries ban importation of U.S. poultry and poultry products.

Virulent Newcastle Disease is caused by Avian Paramyxovirus-1 (APMV-1) in the genus Avulavirus in the family Paramyxoviridae. The ND viruses are divided into three pathotypes: lentogenic, mesogenic, and velogenic, which reflect increasing virulence; the VND viruses are velogenic. Clinical disease is most severe in chickens, guinea fowl, peacocks, pheasants, pigeons, and quail, while severity is variable in passerines and psittacines. Mortality in unvaccinated commercial chickens can approach 100%. Transmission among commercial poultry is via direct contact with infected birds, fecal
material, or aerosol over a short distance, or indirectly via contact with contaminated people, vehicles, and equipment.

Two severe VND outbreaks have occurred in U.S. poultry in the past. In October 2002, VND was confirmed in backyard flocks in California. The disease spread to commercial flocks and to other states. More than 19,000 premises in four states were quarantined during the outbreak, which ran well into 2003 and appeared to be due to separate introductions of two distinct VND viruses in California and Texas. Much of the viral transmission was due to human-assisted movement of backyard poultry and game fowl. More than 3 million birds were depopulated on 2,206 premises. Many countries banned importation of poultry and poultry products from affected counties, states, or the entire U.S., and the outbreak cost more than $160 million to eradicate.

A severe VND outbreak occurred in commercial poultry in southern California during 1971-1973. A total of 1,341 infected poultry flocks were identified, and about 12 million birds were destroyed at a cost of $56 million. SCWDS and USDA-APHIS conducted surveillance during the outbreak to determine if wild birds were spreading the virus. Nearly 10,000 wild birds from 71 species were tested with only four birds (0.04%) testing positive. The crow and three house sparrows were captured in affected poultry premises. Imported pet birds were found to be the source of the VND virus in this outbreak.

Other types of ND virus are found worldwide, and infections have been found in at least 236 species of wild birds. Most wild birds do not carry velogenic strains that cause clinical disease in chickens; notable exceptions are cormorants, pigeons, and doves. These species can carry ND virus strains that affect the wild hosts and have the potential to cause VND outbreaks in domestic poultry. Clinical VND, often characterized by a weak neck, unilateral wing paralysis, and other neurological signs, has been observed in wild double-crested cormorants (Phalacrocorax auritus). A small VND outbreak in domestic turkeys in the 1990s in the Upper Midwest was due to viral introduction from double-crested cormorants, and VND has been observed fairly regularly in wild cormorants in the U.S. since then. In fact, mortality has been occurring in the summer of 2018 in double-crested cormorants in the Great Lakes and Northeast. The National Wildlife Health Center (NWHC) has isolated APMV-1 from affected birds. Genetic characterization of the viruses is pending; however, based on previous investigations in cormorants, it is believed the causative virus likely is VND virus.

The NWHC also has been receiving reports of APMV-1-associated mortality of Eurasian collared doves (Streptopelia decaocto) in Texas, Utah, and Washington. Results of genetic characterization of the virus isolates also are pending; however, it is believed that the causative agent will be identified as Pigeon Paramyxovirus-1 (PPMV-1). This virus was introduced into the U.S. in the 1980s and has been associated with mortality events in Eurasian collared doves and rock pigeons (Columba livia) since then. Some strains of PPMV-1 are virulent in chickens.

With the exception of the VND outbreak in the 1990s in domestic turkeys on pasture (they now are raised in turkey houses), wild birds have not been identified as the source of previous VND outbreaks in U.S. poultry. However, the presence of velogenic ND viruses in wild birds necessitates appropriate biosecurity for domestic poultry in order to prevent virus introduction. The USDA has published recommendations that include stringent biosecurity practices on poultry farms to prevent exposure to diseases like Newcastle. Recommendations include isolation of birds from other birds and human visitors; decontamination/disinfection of clothing, shoes, tools, equipment, vehicles, and cages; observation of birds for signs of disease; and reporting of unusual mortality or clinical disease.

Properly cooked poultry is safe to eat and no human infections have been associated with consuming poultry products. Mild, self-limiting conjunctivitis has occurred in persons in direct contact with sick birds, but infection is easily prevented by using personal protective equipment and good hygiene practices.

More information on VND in poultry can be found at the USDA-APHIS website (https://www.aphis.usda.gov/aphis/ourfocus/animalhealth). Visit the website of the NWHC (https://www.usgs.gov/centers/nwhc) for more information on ND viruses in wild birds. (Prepared by Hannah Creech, UGA College of Veterinary Medicine, and Joe Corn)
Sarcocystosis in a Wild Turkey

A wild turkey was found dead in Baxter County, Arkansas in April of 2018. The turkey was obtained by the Arkansas Game and Fish Commission and submitted to SCWDS for necropsy and diagnostic testing. Gross examination revealed severe emaciation. Skeletal and cardiac muscles contained numerous pale yellow, parasitic cysts that were approximately one-half centimeter long and most notable in the heart (Figure 1), pectoral muscles, and legs (Figure 2). Microscopic evaluation revealed that these areas consisted of degenerated and necrotic protozoan cysts. The cysts incited inflammation that infiltrated many skeletal muscles, including those of the breast, thighs, esophagus, and ventriculus (gizzard), as well as the heart muscle.

A sample of heart muscle was tested at SCWDS for *Sarcocystis* spp. DNA via polymerase chain reaction. Sequence analysis confirmed the parasites were a *Sarcocystis* species that was genetically similar to, but distinct from, *Sarcocystis rileyi* and *S. atraii*. Both of these *Sarcocystis* spp. are parasites of waterbirds, but there is a paucity of sequences available from wildlife species, so relationships within this group are difficult to interpret.

This case is characteristic of sarcocystosis (also called “rice breast”), which can be caused by numerous *Sarcocystis* species in a variety of birds. The condition is more commonly observed in waterfowl, such as mallards, and *S. rileyi* is the most common species reported in waterfowl. The disease also has been reported in chukars and ring-necked pheasants, and rarely, in wild turkeys. This is only the fourth case diagnosed at SCWDS among 881 wild turkeys examined since 1975. The other cases originated in Arkansas, Georgia, and West Virginia, but none of these were genetically characterized, so it is unknown if all cases were caused by the same parasite species.

Birds often serve as intermediate or transport hosts for parasites in this genus; the life cycle continues when the parasites are consumed by the definitive carnivorous hosts, including raptors. The disease also is reported in domestic poultry.

Outwardly visible health effects of this infection in birds are rare, even with high intensity infections. However, when severe, as in the present case, these infections can be debilitating and increase the likelihood of predation or starvation.

Sarcocystosis is not believed to have significant population impacts on wild birds and the disease is not transmissible to humans. However, severe infections in waterfowl and other birds are visible and often raise concern among hunters as to the safety of the meat. While it may appear unpalatable, ingestion poses no human health risks.

The diagnosis of sarcocystosis in a wild turkey highlights our incomplete understanding of the *Sarcocystis* species that infect various game bird groups and the corresponding overlapping carnivore species that may perpetuate transmission in a given region. SCWDS would like to thank Dr. Jenn Ballard and the Arkansas Game and Fish Commission for submission of this case, which revealed a classic disease in an uncommonly affected avian species. (Prepared by Nicole Nemeth and Mark Ruder)
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.