

SCWDS BRIEFS

A Quarterly Newsletter from the
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HPAI Update

Highly pathogenic avian influenza (HPAI) H5 viruses continue to infect large numbers of domestic poultry flocks in the mid-western United States and continue to be detected in wild birds. Currently H5N1, H5N2, and H5N8 virus infections have been confirmed in 222 domestic poultry flocks involving more than 47,000,000 birds, all of which have died or will be euthanatized in disease eradication efforts. Infections also have been confirmed in 73 wild waterfowl and raptors as well as in a small number of captive raptors.

Unfortunately, we have gained little ground in understanding this outbreak since it began in December 2014. The presence of these viruses in such large numbers of domestic birds over an extensive geographic area makes understanding viral reservoirs and transmission events increasingly important and complicated. An example of this is the isolation of HPAI H5N2 from a Cooper's hawk in Minnesota. Although infection probably occurred as a result of predation of another wild bird, the precise source of this infection cannot be determined and could have been a domestic or wild bird. Although this is a noteworthy event, especially related to raptor health, it contributes little to our understanding of the current HPAI situation.

There are two overarching goals to pursue as this outbreak continues: The first and foremost goal is the total eradication of these HPAI viruses from domestic flocks. With the momentum of this outbreak, novel control measures may be needed in the short-term but eradication needs to remain as the ultimate objective. The second goal is to better understand the epidemiology of these viruses in wild bird populations. In the short-term, it is critical that we identify which species are infected as well as when and where

they are exposed. Although increased surveillance of wild birds will help us obtain this information, research is needed to interpret and fully understand the field data. Research also is needed to provide answers to more complex and important long-term questions about the mechanisms that allow for successful invasion, the possible maintenance of these types of viruses, and the outcome of such introductions in wild bird populations.

Scientists in government and academia already are engaged in these influenza research efforts through the USDA-Animal Plant and Health Inspection Service, U.S. Geological Survey, NIH Centers of Excellence for Influenza Research and Surveillance, and state wildlife agencies. We have a solid understanding of the epidemiology of avian influenza viruses of low pathogenicity in wild birds in North America, and this understanding, coupled with existing technical expertise and extensive data, virus isolates, and genetic sequence inventories, should allow us to move forward quickly. However, "quickly" is a very subjective term, especially under outbreak situations, because much of this work is either just getting started or is in the final stages of planning. SCWDS will provide research updates as information becomes available.

We do not know the conditions that allowed these viruses to enter North America, if these viruses will persist in our wild bird populations, if individual viral genes from these viruses will integrate into the North American gene pool, or how prevalent or diverse HPAI H5 viruses may become. However, it is important to note that research related to these questions has importance far beyond the current outbreak: More and more LPAI and HPAI influenza viruses are being introduced worldwide and are becoming endemic in domestic bird populations.

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Some of these (H9N2 and H5N1) have persisted in domestic bird reservoirs for decades. In many parts of the world, contact between domestic and wild birds is not restricted thus allowing for frequent viral and genetic exchanges. Our current HPAI H5N8 introduction is a product of such a scenario, and it may not be the last “new” virus that we see enter North America.

For current information on HPAI viruses in the United States, visit the USDA-APHIS website at http://www.usda.gov/wps/portal/usda/usdahome?contentidonly=true&contentid=avian_influenza.html. (Prepared by David Stallknecht)

Another New Flu

An ongoing outbreak of canine influenza virus (CIV) that has affected an estimated 1,300 dogs in Illinois, Indiana, and Wisconsin has caught the attention of pet owners, animal health professionals, and the national media. This outbreak, which began in March of 2015, originally was believed to be caused by the H3N8 influenza A virus subtype that has existed in horses in the United States and elsewhere since 1963 and apparently jumped to dogs (initially in greyhounds) in the U.S. in 2004. The H3N8 virus has been circulating since then, primarily in dogs in kennels and shelters. However, investigators at the University of Wisconsin and Cornell University determined that the current outbreak is caused by a novel H3N2 virus that now has been detected in dogs in Alabama, California, Georgia, Massachusetts, Michigan, Minnesota, New Jersey, New York, and Texas.

Dogs infected with H3N8 or H3N2 may have no clinical signs or may develop severe lethargy, anorexia, fever, and upper respiratory disease that occasionally is fatal. Transmission occurs via aerosol droplets or direct contact with contaminated objects and has occurred primarily in animal shelters, kennels, dog day care centers and other locations where dogs are in close contact. Infections with CIV H3N2 have not been documented in wildlife in North America, and neither H3N2 nor H3N8 CIVs have ever been reported in humans.

Genetic analysis of H3N2 viruses from the current CIV outbreak revealed that they are most closely related to Asian H3N2 viruses first

detected in dogs in 2006 in South Korea. The CIV H3N2 is believed to have originated as an avian influenza virus that was transmitted and adapted to dogs. Since then CIV H3N2 also have been circulating in dog populations in China and Thailand. The CIV H3N2 also has been reported to cause respiratory illness in cats in Asia. Detection of CIV H3N2 this spring in the upper Midwest suggests recent introduction of the virus to this area, but how or when the virus arrived in the U.S. is unclear.

The H3N2 and H3N8 CIVs are antigenically different, and current serological assays for H3N8 may not detect antibodies against H3N2. An H3N2-specific serologic assay is under development and is available at a few diagnostic labs. Diagnosis may be made by virus isolation or a polymerase chain reaction (PCR) assay. The CIV vaccine currently available for dogs protects against H3N8 but it is unknown whether it will provide appropriate protection against H3N2; however, efforts are underway to develop an H3N2 vaccine.

Chicago veterinarians have reported a decline in the number of cases of canine influenza since mid-April and believe media coverage of the “dog flu” outbreak may have slowed the spread of the virus by educating owners on ways to minimize the risk of exposure to their dogs. The U.S. outbreak continues to be monitored with current information available at the websites of several institutions including Cornell University: <https://ahdc.vet.cornell.edu/news/civchicago.cfm>. (Prepared by Kevin Niedringhaus)

Avian Cholera and More!

On March 10, 2015, hundreds of dead ducks and geese were found dead in and around a pond on a private hunting reserve in western Kentucky. The mortality event was investigated by Kentucky Department of Fish and Wildlife Resources biologists, who sent several mallards, ring-necked ducks, and snow geese to SCWDS for postmortem examination.

Gross examination revealed the birds were in good nutritional condition with abundant body fat. Lesions included multiple pale, pin-point foci throughout the liver (Figure 1) and multifocal hemorrhage in visceral organs. Microscopic

lesions included multiple foci of hepatic and splenic necrosis that contained vast numbers of bacteria and were surrounded by inflammatory cells. *Pasteurella multocida* was isolated from the liver of all six birds, confirming the diagnosis of avian cholera. It all appeared straightforward and very typical of avian cholera, but there was more to the story: We isolated two influenza viruses that later were identified as highly pathogenic avian influenza (HPAI) H5N2 by the USDA-APHIS National Veterinary Services Laboratories.

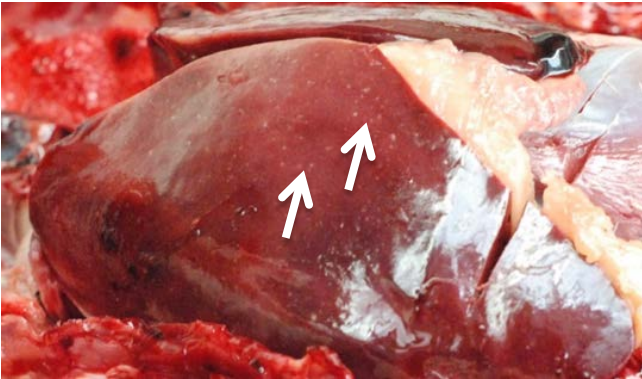


Figure 1. Pin-point foci of liver necrosis (arrows)

Pasteurella multocida is a gram-negative bacterium that affects numerous species of wild and domestic birds and mammals (see the following article on the recent Saiga antelope die-off). Humans are susceptible and infections often are associated with animal bites, especially from cats. Avian cholera typically is caused by *P. multocida* serotypes A:1 or A:3, and it is considered one of the most important diseases affecting wild waterfowl. It has a worldwide distribution and may impact common eider populations. Avian cholera outbreaks occurred this past winter and spring in Idaho, Kentucky, Missouri, Nevada, Pennsylvania, Virginia, and other states with mortality estimates ranging from 25 to tens of thousands of birds. In Idaho, snow geese were reported to have fallen from the sky in an outbreak that affected thousands of birds.

Avian cholera occurs in multiple clinical forms based on host factors with infections ranging from peracute to chronic. Ducks and geese typically are found dead, and mortality associated with fowl cholera often occurs during epornitics involving hundreds to thousands of birds, although smaller die-offs also occur. Gross necropsy findings of avian cholera often include

good body condition and multiple, small white foci scattered throughout the liver and/or spleen.

The role of reservoir hosts of *P. multocida* is not fully understood, but outbreaks most likely are sparked by asymptomatic carriers. Studies have pointed to species, such as the lesser snow goose as one potential reservoir, and some outbreaks have been linked to their arrival at a site. Transmission occurs most frequently via direct contact between birds. Indirect means of transmission include ingestion of contaminated water, inhalation of aerosolized droplets, and potentially via mechanical transmission by ticks, poultry mites, lice, and flies. Infected birds may shed vast numbers of *P. multocida*, and carcass removal has been used to decrease bacterial loading in the environment and to prevent scavenging.

The potential role of weather in avian cholera epidemiology is unclear. Increased precipitation has been associated with higher disease prevalence in some instances, while drought conditions can lead to crowding that can facilitate disease transmission and exacerbate outbreaks. Winter and spring are the predominant seasons for outbreaks, but waterfowl concentrations then may make mortality events easier to detect. Avian cholera has been reported at various locations during all months of the year, and the true seasonal prevalence across populations has yet to be fully discerned.

Unfortunately, we cannot provide epidemiological information regarding HPAI in wild birds like we can for avian cholera. Prior to 2002, HPAI had been documented in wild birds on a single occasion in South Africa in 1961. Beginning in 2002, HPAI H5N1 caused morbidity and mortality in domestic poultry, wild birds, and captive exotic birds in Asia, eventually spreading to Europe and North Africa. This virus also infected humans and killed some of them.

The first detection of HPAI in wild birds in North America occurred in late 2014 and to date, Eurasian H5N8, and HPAI viruses with Eurasian hemagglutinin H5 and North American neuraminidases N2 and N1 (which differs from the completely Eurasian H5N1) have been confirmed in wild and/or domestic birds in several states. Fortunately, no human health issues have

been associated with any of the HPAI viruses recently detected in North America. As stated in the last issue and in the first article in this issue of the SCWDS BRIEFS, we currently do not know enough about HPAI epidemiology to predict its spread and potential for establishment in wild birds in North America or its impacts on wild bird populations. In fact, we are uncertain how it may impact the health of individual wild birds as we isolated H5N2 from these birds that apparently died from avian cholera, as well as from two snow geese in which we could find no other potential cause of death. (Prepared by Kali Standorf from the University of Florida College of Veterinary Medicine and Heather Fenton)

Saiga Antelope Die-Off in Kazakhstan

A massive die-off that began in early May 2015 has killed more than 134,000 Saiga antelope (*Saiga tatarica tatarica*) in Central Kazakhstan according to an official report submitted by the country's Ministry of Agriculture to the World Organisation for Animal Health (OIE). Mortality began on May 5, grew in intensity until it peaked on May 15-16, and subsided by early June 2015. The report documented four separate outbreaks with mortality ranging from 10,294 to 61,203 antelope per population, and the overall morbidity/mortality rate was 88%.

The Saiga antelope is regarded as critically endangered, the category at highest risk for extinction, and the recent mortality event is feared to represent a significant threat to the future of this species. Female Saiga antelope gather in large herds each year in early May to calve. Conservationists were on-site in Central Kazakhstan by May 10 this year to observe calving success, as well as the condition of the calves and adults, and this put them in position to observe the mortality event. They reported animals with weakness, depression, ataxia, diarrhea, hypersalivation, and dyspnea. Affected antelope often collapsed and remained recumbent until death, which occurred within hours of the first clinical signs. The mortality event was investigated by local, regional, and national veterinary services as well as the Royal Veterinary College in London, the Food and Agriculture Organization of the United Nations, the Research Institute for Problems of Biological Safety, and others. Sample analysis by the

National Reference Center of Kazakhstan's Ministry of Agriculture resulted in isolation and identification of *Pasteurella multocida*, the causative agent of hemorrhagic septicemia (HS). However, the possible roles of other factors, including weather conditions and coinfections with viruses or other pathogens, continue to be investigated.

Hemorrhagic septicemia is a major disease of cattle and water buffalo (*Bubalus bubalus*) in parts of Asia, southern Europe, and the Middle East, and occasional outbreaks have been reported in sheep, goats, and swine. The only outbreaks reported in the United States occurred in bison (*Bison bison*) on three occasions between 1911 and 1965. The Asian serotype (B:2) and the African serotype (E:2) of *P. multocida* are not known to infect people, although other serotypes have been reported in humans and numerous other species. Asymptomatic cattle and buffalo may carry the bacteria in the nasopharynx, and transmission occurs via direct contact with infected animals or fomites, as well as by ingestion or inhalation. Stressors, such as crowding and poor food supply, are thought to increase susceptibility to infection and clinical disease.

Most cases of HS in cattle and buffalo are acute or peracute and begin with fever, dullness and reluctance to move. Hypersalivation and serous nasal discharge develop along with edematous swelling of the pharyngeal region that may spread to the brisket. Respiratory distress develops and is followed by recumbency and death, usually within 6-24 hours of the first signs. Some cases may be protracted over five days before death, but chronic cases have not been observed. Affected animals, especially buffalo, rarely recover, and the case fatality rate approaches 100%. More recently, HS has been seen as a secondary complication of foot and mouth disease in cattle and buffalo.

The catastrophic Saiga antelope die-off of 2015 has caught the world's attention. Similar mortality events were observed in recent years on a much smaller scale, but the cause(s) were not identified. In 2010, approximately 12,000 antelope died in western Kazakhstan, and a smaller event there in 2011 killed 900 animals. In 2012-2013, two die-offs in the same area as

the 2015 event killed approximately 1,900 antelope. The impacts of all of these die-offs on Saiga antelope conservation, particularly the 2015 outbreaks that killed half of the global population, will be closely monitored for years to come. (Prepared by John Fischer)

Antibiotic Resistance in Geckos

The emergence of antibiotic-resistant bacteria is a growing public health concern and has serious implications for human and veterinary medicine. Our global economy facilitates the international movement of humans, livestock, produce, and wildlife as well as their potentially antibiotic-resistant bacteria. Humans and livestock are known reservoirs for antibiotic-resistant bacteria; however, we know little about the prevalence of antibiotic-resistant bacteria harbored by wildlife and, to our knowledge, nothing has been reported for wild-caught reptiles collected for the pet trade.

The United States imported over 1.5 billion live animals between 2000 and 2006. During this period, 69% of these animals originated from Southeast Asia, an area known for high rates of antibiotic resistance in bacteria cultured from humans. Wild animals imported to the United States from Southeast Asia often endure stressful transport conditions. These stressful conditions often lead to physiological changes within the host, most of which have been linked to immunosuppression, increased mutations and exchange of antimicrobial resistance genes among enteric bacteria, and an increased rate of pathogen shedding.

Tokay geckos naturally occur from northern India to the Indo-Australian Archipelago. Many geckos captured for distribution in the international exotic pet trade come from peri-domestic settings, where they might have acquired antibiotic-resistant bacteria via exposure to human or livestock waste. Tokay geckos can be aggressive and often are released by their owners into the environment. Consequently they now are considered an established, breeding, invasive species in the United States (Florida, Hawaii, and Texas) and in other countries (Belize and Martinique), creating a unique potential for the introduction of resistant bacteria from Southeast Asia into new regions and hosts. In order to

evaluate any potential risk imported geckos could present, SCWDS conducted a study to investigate 1) the antimicrobial resistance patterns of lactose-fermenting enteric bacteria from individually-housed imported Tokay geckos immediately upon arrival and, 2) how antimicrobial resistance patterns might change after manipulating their density - mimicking stressful transport and distribution practices.

We cultured bacteria from the feces of geckos shortly after their arrival at SCWDS from Indonesia to identify "baseline" commensal flora, as well as approximately six months later after they had been caged in groups at low, medium, or high densities. From the 110 geckos we studied, we recovered 189 isolates of bacteria in the family *Enterobacteriaceae*, which is a large family of gram-negative bacteria of the intestinal tract that includes pathogenic and harmless, commensal organisms.

These bacteria were resistant to antibiotics that commonly are used in Southeast Asia, and specifically in Indonesia (chloramphenicol, aminopenicillins and tetracyclines). We found relatively high rates of resistance among *Citrobacter* spp., *Enterobacter* spp., *Klebsiella* spp., and *Serratia* spp. to these and other commonly-used antibiotics in the USA, such as cephalosporins. Interestingly, the prevalence of *Klebsiella* spp. resistant to cefoxitin was much lower in individually housed geckos (4%) compared to animals in combined groups (50%). There also was a low prevalence of resistance against other antibiotics, such as aminoglycosides and fluoroquinolones often used to treat severe gram-negative bacterial infections in the USA.

Using a conservative definition of multi-drug resistance (resistance to three or more classes of antibiotics) we found four multidrug-resistant isolates of *Enterobacter* spp. Two of these isolates were resistant to four classes of drugs, including one quinolone and two aminoglycosides, which often utilize plasmid-mediated resistance mechanisms and are commonly reported in SE Asia. Future work should follow to understand whether close contact between pet owners and imported reptiles harboring antibiotic-resistant bacteria results in the exchange of these organisms, and

if the movement of antimicrobial resistant bacteria from imported Tokay geckos impacts the gastrointestinal flora of domestic animals or native wildlife, particularly in areas in the USA where the Tokay gecko has become established. (Prepared by Chrissy Casey and Sonia Hernandez)

TWS/AAWV Position on Wild Sheep Disease Risk

The Wildlife Society (TWS) and American Association of Wildlife Veterinarians (AAWV) recently adopted a Joint Issue Statement on “*Domestic Sheep and Goats Disease Transmission Risk to Wild Sheep.*” The policy was adopted in response to the threat that respiratory pathogen transmission from domestic sheep and goats to bighorn sheep (*Ovis canadensis*) and thinhorn (Dall) sheep (*O. dalli*) poses to once abundant wild sheep populations. Wild sheep numbers were devastated through the early 1900s by unregulated hunting, disease, competition with domestic livestock for forage and space, and habitat fragmentation. The statement cites incidences of pneumonia-related wild sheep die-offs associated with the presence of domestic sheep and goats as well as controlled research studies that confirmed transmission of *Mannheimia hemolytica* and *Mycoplasma ovipneumoniae* to wild sheep from domestic sheep.

Policy elements regarding the risk of disease transmission from domestic sheep and goats to wild sheep are to:

- Accept that science consistently has demonstrated respiratory disease transmission from domestic sheep/goats to wild sheep upon contact or proximity.
- Recognize this disease transmission is a significant risk factor for wild sheep population restoration and conservation.
- Recognize effective spatial and temporal separation of wild and domestic sheep/goats is the only available solution to prevent or minimize disease transmission and promote cooperative strategies to achieve separation, reduce disease transmission, and mitigate disease outbreaks.

- Recognize co-mingling of domestic and wild sheep may result in continued loss of wild sheep from disease and that wildlife managers may need to cull infected wild sheep herds to reduce disease transmission risks.
- Recognize some wild sheep populations may harbor pathogens detrimental to other wild sheep. Translocation can spread the pathogens and should occur only when disease risk is low and conservation benefits are high.
- Emphasize the need to develop strategies to address wild sheep populations chronically infected with significant respiratory pathogens.
- Emphasize importance of monitoring herd health following translocations or disease events.
- Promote increased communication and cooperation among all stakeholders.

The entire statement, as well as other TWS Position Statements, can be found at: (<http://wildlife.org/position-statements/>). (Prepared by John Fischer)

Muskox Mortality in the NWT

In July 2012, approximately 100 muskoxen (*Ovibos moschatus*) were found dead or dying on Bank’s Island, Northwest Territories (NWT), Canada. This mortality event was the largest of several summer muskox die-offs in northern Canada that have been attributed to systemic infection with the bacterial organism *Erysipelothrix rhusiopathiae*.

Erysipelothrix rhusiopathiae is a gram-positive bacillus that can persist for weeks or months in the environment. The bacterium commonly is found in decomposing nitrogenous waste and in aquatic environments where it often is associated with the mucus covering the skin of fish. It can infect a variety of species, including many domestic and wild mammals and birds, as well as humans. In non-human animals, the disease associated with *E. rhusiopathiae* infection is commonly referred to as ‘erysipelas.’ Systemic infections in domestic turkey, sheep, and swine

can cause huge economic losses, and vaccines are available for these species.

Human infections with *E. rhusiopathiae* most commonly occur via occupational handling of pork or fish containing the bacterium and result in a mild skin disease known as 'erysipeloid.' (The unrelated human disease known as 'erysipelas' is caused by *Streptococcus* spp.) Erysipeloid often occurs on the hand and is characterized by localized skin pain, discoloration, and inflammation that develop after contamination of an open wound. Although these infections often are self-limiting, erysipeloid sometimes leads to sepsis and endocarditis. Public health concerns have been raised regarding the safety of wild meat during the recent erysipelas outbreaks in the NWT because local Inuit communities rely on the harvest of wild game for subsistence.

Although infection with *E. rhusiopathiae* is rare in wild artiodactylids, it was associated with a mortality event among moose in 1989 in Algonquin Provincial Park, Ontario, Canada. Affected moose were emaciated, had marked hair loss and heavy infestations of the winter tick (*Dermacentor albipictus*) suggesting the possibility of tick-associated transmission of the bacterium. The method of transmission in muskoxen is unknown, but potential routes being considered include direct contact with open wounds, contamination of water sources, and contact with potential vectors or reservoir species.

Erysipelas in muskox is considered a potentially emerging disease in the Arctic, and scientists are

trying to identify factors that may have facilitated large-scale mortality events. Climate change is being considered as a potential driver of the emergence of this disease in muskoxen. Although currently unknown, it is hypothesized that muskox, as well as other species adapted to northern climates, may be more susceptible to infectious diseases in the face of changing environmental conditions. In Norway, an unusually hot and humid summer was regarded as a possible contributing factor to the development of a 2006 pneumonia outbreak that killed a large proportion of an introduced muskox population. Changing environmental conditions also have been shown to shorten the time necessary for completion of the life cycle of a muskox lungworm (*Umingmakstrongylus pallikuukensis*) and to facilitate expansion of the geographic distribution of the parasite. No evidence currently exists for arctic warming as a cause for the recent *E. rhusiopathiae* infections, but the possibility of an association warrants further study.

It is unclear how *E. rhusiopathiae* was introduced to the Bank's Island muskox population. Ongoing studies by scientists at the University of Calgary and international wildlife health centers have determined that the bacterial strain from the 2012 mortality event may have been present in muskox populations since the early 1990's. Although the population impacts of erysipelas-associated mortality are unknown, some local guides and outfitters indicated they would not provide muskox hunts after 2014. (Prepared by Julia Hill, University of Georgia College of Veterinary Medicine and Heather Fenton)

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