Notes from Our Director...

I was able to participate in a four-day African Swine Fever Outbreak Laboratory Response Course at the Plum Island Animal Disease Center on July 29th-August 1st. The course was offered by the USDA National Animal Health Laboratory Network (NAHLN) and sponsored by the US Department of Homeland Security (DHS). Participants spent three days learning the principles of the FEMA Incident Command System Level 300 directed specifically towards laboratory emergency response. The course included a simulated response exercise that included representatives from USDA, DHS, FADDL, NVSL, and NAHLN member laboratories. Participants were also invited to enter into the biocontainment section of the Center for a necropsy presentation of swine under study that were infected with African Swine Fever to learn more about the clinical presentation, progression, necropsy lesions, and laboratory methods used to detect the disease. The training finished with a live video with representatives from USDA who are now in China observing the ASF outbreak.

Our laboratory participated in two African Swine Fever Exercises the week of September 23rd. The first day involved a joint exercise with the USDA, the North Carolina Department of Agriculture’s Veterinary Division, including the State Veterinarian, and Industry Representatives. We worked through the procedures for communication, collection, transport, testing, and reporting/notification of an African Swine Fever suspect. The following days the laboratory was involved in an APHIS-NAHLN African Swine Fever disease outbreak scenario with mock laboratory submissions related to the simulated outbreak which included samples (simulated and virtual) to test and report or message.

Our IT staff along with laboratory section supervisors and management are working to enhance our website by adding a searchable test catalog. We’re hoping it will be available on our website in the next few months!

We will also be adding Diagnostic Plans to our website to provide guidelines and currently available tests to assist veterinarians with working up challenging diagnostic cases. Check out our first set of Bovine Diagnostic Plans here! https://www.ncagr.gov/vet/ncvdl/BovineDiagnosticPlans.html

In July we hosted several USDA-APHIS representatives and officials from Thailand’s Department of Livestock Development. They were interested in visiting a NAHLN laboratory and observing our poultry testing capabilities.
We are always looking to add to our diagnostic testing capabilities. We have added an avian Reovirus PCR to our test catalog and continue work on a calf diarrhea PCR panel among others. If there are tests you would like to see us incorporate please let us know.

Jim Trybus, DVM, DAVCP

Can Animals get Legionnaires’ Disease?

A recent outbreak of Legionnaires’ disease linked to North Carolina Mountain State Fair has sickened more than hundred people and became a serious public health concern. Legionnaires’ disease, caused by the bacterium Legionella, can cause severe respiratory disease in people at increased risk such as elderly, smokers, people with chronic lung disease or in people with weakened immune systems. However, most healthy people exposed to Legionella do not become sick.

Legionellae are bacteria that are ubiquitous in natural aquatic ecosystems. They can grow and multiply in human made building water systems such as cooling towers and hot tubs that are not properly maintained. It becomes a health concern once water containing Legionella spread in droplets small enough for people or animal to breathe in.

Legionnaires’ disease is not considered to be a concern for animals, nor is transmission from animals to people thought to be a risk. Animals are generally considered NOT to be infected with the bacterium Legionella. So, while it seems unlikely that animals can get Legionnaires’ Disease, there is one isolated report of this bacterium found in respiratory organs of calves. Experimental infections, however, showed the mouse as the only animal species where Legionella infection can become established.

Contamination with Legionella bacteria is theoretically possible in animals. If respiratory symptoms appear in animals that appear similar to Legionnaires’ disease in people after exposure to contaminated water, please consult with your local veterinarian. Animals are more prone to get serious infections such as leptospirosis upon exposure to contaminated water which needs to be ruled out.

Since Legionnaires’ disease is only found in humans, most veterinary diagnostic labs are not equipped for testing for the bacterium Legionella. However, most veterinary diagnostic labs could be able to facilitate testing with local public health labs if there is a strong suspicion of Legionnaires’ disease in any animal species and your local veterinarian could certainly help with that.

Anil Thachil, BVSc, PhD, DACVM
Erysipelas in a Pot Belly Pig

By: Elise Lavie, DVM

An 8-year-old, 35 kg, black, female micro Pot Belly Pig in heavy body condition (BCS 4/5) and with mild to moderate post mortem autolysis presented for necropsy following a 12-hour history of suspected dystocia. She began showing signs of labor around 6 pm and was found deceased the next morning at 6 am without having delivered any piglets.

Gross necropsy was relatively unremarkable. White froth filled the trachea and mouth. Locally extensive petechial hemorrhages were present around the coronary vessels. The proximal tracheal mucosa was dark red, as was the stomach mucosa along the greater curvature. The cervix was open, and the uterus contained nine normally formed, nearly mature piglets. Meconium was present in the amniotic fluid of some piglets indicating fetal distress. No further gross abnormalities were present.

Histopathology revealed a multifocal, moderate fibronsuppurative and mild eosinophilic myocarditis as well as mild, multifocal necrofibrinous splenitis.

Aerobic culture of the liver, lung, spleen, and uterus resulted in heavy growth of *Erysipelothrix rhusiopathiae* from all organs.

Initially, the cause of death was unclear. Calcium deficiency can cause uterine inertia and can also impair cardiac function. There did not appear to be a physical cause for the suspected dystocia. Petechiae can be the result of septicemia but are also a common perimortem change. Histopathology indicated the need for bacterial culture. A bacterial infection was the favored cause for the myocarditis due to the finbrinosuppurative component, and the splenitis was deemed likely due to a systemic bacterial infection.

Erysipelas is most commonly recognized as Diamond Skin Disease due to the dark red to purple, rhomboid vasculitis lesions that form on the skin, however multiple less commonly recognized diseases are seen with *Erysipelothrix rhusiopathiae* infection including acute septicemia. Pigs with acute *E. rhusiopathiae* septicemia frequently die suddenly without any clinical signs or skin lesions. They may also be febrile, lethargic, reluctant to stand, anorexic, may vocalize excessively when handled, and some may walk with a stilted gait on their toes. Both acute forms of disease (septicemia and Diamond Skin Disease) can be accompanied by abortions. Chronic forms of the disease also exist that result in arthritis and vegetative valvular endocarditis.

Clinical disease in groups of pigs is typically sporadic, but outbreaks of the disease can occur. Stress can induce disease in pigs exposed to the bacteria. Infected pigs excrete the bacteria into the environment from nasal secretions, saliva, urine, and feces. It can live for weeks in the environment. The bacteria is spread through ingestion of contaminated feed, water, and feces or through open wounds/abrasions in the skin. Treatment of acute forms of the disease is possible through the administration of specific antibiotics, typically penicillin, however treatment of chronic forms of Erysipelas is not usually effective. Prevention is possible through vaccination and good hygiene/sanitation.

There is a small potential for human infection with *E. rhusiopathiae*, most commonly small, localized skin lesions however the potential for endocarditis and septicemia in people does exist.
Figure 1: Typical diamond/rhomboid skin lesions due to acute E. rhusiopathiae infection.

Epizootic Hemorrhagic Disease in Deer

By: Alison Tucker, MA, VMD, DAVCP

In a group of adult captive whitetail deer, over a 3-day period in late August of a previous year, one adult was found dead, with no premonitory signs, by a pond and two other deer, observed standing within the pond, were subsequently found dead beside the pond.

An adult female whitetail deer was submitted for post-mortem examination in good nutritional condition and with mild post-mortem decomposition. The tongue was swollen and was tinged dark blue. The glossal epithelium did not have ulcers. The head was mildly swollen with subcutaneous edema. There were multiorgan petechial and ecchymotic hemorrhages on serosal surfaces within the abdomen and ruminal, abomasal and intestinal mucosa were dark red. There was pulmonary, pericardial and epicardial edema with petechial hemorrhages throughout the myocardium.

Microscopic examination of tissues identified multiorgan endothelial hypertrophy with vessel wall necrosis. Affected tissues also had microvascular thrombosis, perivascular edema and hemorrhage, and areas of tissue necrosis. In the tongue there was submucosal necrosis and hemorrhage and the epithelium had intercellular edema and pustules.

The working diagnosis was acute Epizootic Hemorrhagic Disease (EHD) which was supported by later identification of a reovirus by virus isolation at Rollins Laboratory and further characterization of that virus by RT-PCR for EHD at the National Veterinary Services Laboratories.

Figure 1: Abdominal visceral serosal hemorrhages
Epizootic Hemorrhagic Disease is an important disease of cervids. It is caused by a group of closely related Orbiviruses of the family Reoviridae. The virus is present in North America, Australia, Africa and Asia. At least seven serotypes of EHD are recognized. Serotypes 1, 2 and 6 are present in North America and there can be recombinants of these present as well. Transmission in North America is by *Culicoides sonorensis*, a biting midge, and outbreaks are caused by exposure to the midge rather than by horizontal transmission between deer. This vector and these viruses have a historical geographic range of 40°N–50°N and 35°S, although recent reports indicate the range is extending northward into Canada.

The disease can present with sudden death or can be a chronic debilitating disease with damage to the distal limbs and hooves as well as ulcers in the omasum and rumen. The incubation period is 5 – 10 days and in acute cases the disease progression from onset of clinical signs to death is approximately 8 – 36 hours. Viral infection of endothelial cells results in vascular damage, which in turn results in the tissue necrosis, hemorrhage and edema that are the hallmarks of acute infection. Clinical signs can include fever, anorexia, lethargy, stiffness, respiratory distress, edema of head and neck, oral ulcers and erosions, hemorrhage and dehydration. In acute cases animals commonly seek water or are found dead near a water source. Insect control is recommended but definitive preventive protocols are not available and treatment is symptomatic.

Blue tongue virus is a closely related Orbivirus and there is overlap in host species and clinical signs. In addition, the clinical signs of EHD can be similar to those seen with foot-and-mouth disease, vesicular stomatitis, and malignant catarrhal fever. Because of this overlap in hosts and clinical signs, outbreaks can result in a foreign animal disease investigation.

A Case of Sudden Death...

By: Stacy Robinson, DVM

A 6-month-old Angus steer calf was found dead in the owner’s pasture at noon. The calf was last seen alive the evening before at about 9 pm and appeared healthy at that time. This was the second calf on the farm that had been found dead recently. The first calf was similar in age to the second calf and was found dead in the same pasture 2 days prior. The cattle do not receive any vaccines. Their diet consisted of pelleted cattle feed, fescue hay and pasture. Adult cows appeared healthy.

At necropsy the calf was in normal hydration status and good body condition. A few red patches were in the myocardium and epicardium of the ventricles of the heart. Black patches of necrosis and hemorrhage were in the skeletal muscle of the left hip and right hip regions and in the proximal medial aspect of the hind legs. Mild emphysema was identified in a few areas of the affected skeletal muscle. A rancid butter odor was also detected in the affected skeletal muscle.

Figure 1: Necrotizing and hemorrhagic myositis with emphysema in affected skeletal muscle

Figure 2. Necrotizing and hemorrhagic myositis
Histopathology confirmed that in the red and black affected skeletal muscle and cardiac muscle there were coalescing regions of myofiber degeneration and necrosis. Myofibers were also effaced and displaced with hemorrhage and inflammation.

Morphological Diagnosis: Myositis and myocarditis, necrohemorrhagic, acute to subacute, moderate

Heart and skeletal muscle were positive for *Clostridium Chauvoei* by florescent antibody test

**Diagnosis:** Blackleg

Blackleg is caused by the bacterium, *Clostridium chauvoei* which is a gram- positive, spore-forming anaerobic bacilli. The bacterium is found in the intestinal tract of cattle and sheep and it survives in the soil for a long while. Ingestion of the spores is reported to be the most likely route of exposure in cattle. Spores are disseminated to the muscles and they may remain dormant in skeletal muscle until the muscle is damaged such as from trauma. Damaged muscle has reduced oxygen which is the perfect environment for vegetative growth from the spores. When this occurs, the bacterium produces exotoxins. Blackleg usually occurs in cattle that are in good body condition and that are less than 3 years of age. Six months to two years of age is reported to be the most common age of cattle affected with the disease. It also occurs most commonly in the summer and fall months.

Clinical signs often seen with blackleg include sudden death, swelling in the legs, lameness and fever. Crepitus/Gas bubbles can be felt under the skin of some affected animals. Usually the cattle die within 24 to 36 hours after the onset of these symptoms. Muscles in the hip and brisket areas and the tongue, heart, and diaphragm are common areas where the lesions of blackleg may be found. It is especially important that animals suspected of having blackleg are submitted for necropsy as soon as possible after death since it becomes more difficult to differentiate between blackleg and postmortem change both grossly and microscopically the longer the animal is deceased.

Positive fluorescent antibody test for *Clostridium Chauvoei* in muscle with microscopic evidence of necrotizing and hemorrhagic myositis helps to confirm the diagnosis. Anaerobic bacterial culture of affected muscle for *Clostridium chauvoei* may also be performed.

A good vaccination protocol is typically preventative. The most common fact in the history of all the cases seen at the laboratory is that the owner is not vaccinating against blackleg.

**References:**

https://www.merckvetmanual.com/generalized-conditions/clostridial-diseases/blackleg


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