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Message from the Director



Greetings from the Director's Office! My name is Dr. Will Laegreid and as of January 30 I am the new Director of the Wyoming State Veterinary Laboratory. I come to Wyoming from the University of Illinois but grew up in eastern Washington and couldn't be happier to be back in the West, in "God's

country." As I assume my new duties I am grateful to inherit a solid organization with excellent staff and facilities, due in large part to the efforts of Interim Director Dr. Todd Cornish and the previous Director, Dr. Don Montgomery (both of whom remain as pathologists at the WSVL). I hope you will join me in thanking them for their service to the WSVL and the State of Wyoming.

Looking to the future, my goal is to support the WSVL faculty and staff as they maintain and build on the legacy of sound professional practice and compliance with the requirements for AAVLD accreditation. We are currently improving our laboratory information and quality control systems, to ensure high standards of accuracy and provide the best possible service to our clients. I would like to hear

any suggestions or comments (good and bad) about the WSVL, so please email me or contact the lab by phone if you would like to contribute your thoughts. Our mission remains to provide *"accessible, timely, accountable, and accurate diagnostic services, animal disease research, & education to veterinarians, students, others interested in animal health, and the people of Wyoming."*

Another new face in the WSVL is that of Katie Talbott. Katie joined the lab in February as a senior office assistant. She will be working with current staff Marjorie Jaeger and Tammy Bartlett on duties including accessioning and billing. Katie brings a wealth of experience from her former position in a private veterinary practice and we are very pleased that she has joined the WSVL staff.

Cache Valley Viral Infection as a Cause of Abortion with Skeletal and Brain Malformations in a Wyoming Sheep Flock

The WSVL has had several cases of abortion and congenital malformations in lambs from one small flock from central Wyoming this year. Dissections revealed arthrogryposis and hydrocephalus/hydranencephaly with some lambs additionally having scoliosis and facial malformations. Differential diagnoses considered were poisonous plants (e.g. skunk cabbage, lupins and poison hemlock), spider lamb syndrome (hereditary chondrodysplasia) and Cache valley virus infection. Spider lamb syndrome was ruled out based on microscopic evaluation of growth plates. Plant poisoning could not initially be excluded

due to inability to evaluate plants in late winter on pasture. Serum neutralization testing of ewes demonstrated positive antibody titers to Cache Valley Virus (CVV, *Bunyaviridae: Orthobunyavirus*). Antibody testing performed at another laboratory in one lamb (that had not received any colostrum) also revealed antibodies to CVV confirming *in utero* infection.

CVV is a mosquito-borne arbovirus first isolated from mosquitoes in the Cache valley of Utah in 1956. It is now recognized to be present throughout North America. There is serological evidence for infection in sheep and goats, horses, pigs, cattle

and wildlife ruminant species. Most infections in sheep are subclinical and go unrecognized, but infection in the first trimester of pregnancy can result in fetal death, mummification, or fetal malformations that include arthrogryposis, scoliosis, hydrocephalus/hydranencephaly, microcephaly, and cerebellar and muscular hypoplasia. Lambs born alive are typically weak and do not survive. The diagnosis of CVV can be challenging because the virus is gone at the time malformed fetuses are noted. The detection of antibodies in the serum of a dead lamb or fetus is diagnostic of in-utero infection. Ewes also become antibody

“...detection of antibodies in the serum of a dead lamb or fetus is diagnostic of in-utero infection.”

Cache Valley Virus, *continued*

positive and are thought to be immune to future infections. There are no vaccines available for prevention of CVV infections, and methods to protect sheep are limited. They include limiting breeding of naïve ewes during mosquito season, or as much as possible protecting ewes from mosquitoes during the first trimester of pregnancy.

Jonathan Fox Pathologist
Myrna Miller, Virologist



An aborted lamb infected in utero by Cache Valley Virus showing severe skeletal malformations and arthrogryposis. (with permission, <http://w3.vet.cornell.edu/>).

Suspected Post-Vaccinal Canine Distemper

There are multiple strains of canine distemper virus that can potentially cause illness with high mortality in dogs and some wildlife species. Infections are typically multi-systemic. Cases with neurological disease are characteristically demyelinating with a fairly stereotypical topographic brain distribution. One virus strain, Snyder Hill, is different. Instead of demyelination, the virus is neuronotropic with intranuclear inclusion bodies in neurons and neuronal necrosis. Attenuated strains of the Snyder-Hill virus have been one of several used in modified-live vaccines. During the last few years, outbreaks of canine distemper causing widespread outbreaks of disease in wild carnivores with respiratory disease, neuronotropic encephalomyelitis, high morbidity, and high mortality have been reported from Europe.

Canine distemper virus infections following administration of modified live virus vaccines have been recognized for a

number of years. Cases, however, are sporadic and our current understanding is incomplete.

Clusters of cases were recognized nationwide during the early 1990's and several cases were identified by this pathologist during this time frame. Cases typically occurred in younger-aged dogs; onset of clinical signs was usually between 10 and 14 days following vaccination. Clinical signs of systemic illness including conjunctivitis, rhinitis, and pneumonia were typically absent; disease was limited to involvement of the central nervous system (CNS). Even here, the tonic-clonic symptoms typical of distemper virus CNS infection, i.e. 'chewing-gum' seizures, were absent. Instead, the earliest clinical signs were more related to changes in behavior including aggression in an otherwise affectionate dog.

The microscopic lesions of post-vaccinal canine distemper in the brain are also unlike those typically observed in naturally

occurring infection with 'street'-virus. Noteworthy are difference in topography and character of the lesions. Lesions commonly involve the thalamus and mid-brain and, instead of white matter tropism with demyelination, are neuronotropic. Characteristic of post-vaccinal canine distemper are large intranuclear inclusions in neurons accompanied by severe neuronal necrosis in affected areas of the brain stem.

The WSVL recently received a case from an 11-month-old male crossbred dog with neurological signs beginning approximately 14 days after distemper vaccination. Microscopic brain lesions were typical of post-vaccinal canine distemper as described above and are similar to the disease caused by Snyder Hill viral strains (Figure 1).

Fluorescent antibody (FA) staining was positive for canine distemper and the virus was isolated on canine peripheral blood mononuclear cells. Addi-

Post-Vaccinal Canine Distemper *continued*

tional virological studies are in progress, hopefully providing a better understanding of the relationship between vaccination and development of virulent brain infection. If you have cases with similar clinical signs 10 to 14 days following vaccination, please notify the WSVL.

D.L. Montgomery, Pathologist
Myrna Miller, Virologist
Jackie Cavender, Virology

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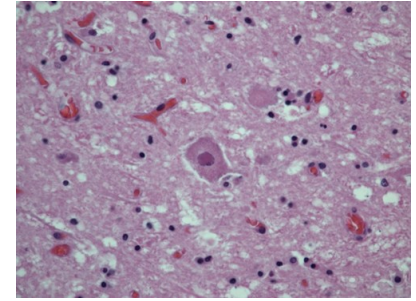


Figure 1. Red nucleus in a dog with suspected post-vaccinal canine distemper virus infection. Note the large intranuclear eosinophilic inclusion in the centrally located neuronal cell body.

Equine Ionophore Poisoning

A quarter horse gelding was presented to the clinic with hind limb ataxia, elevated heart rate, and marked discomfort while standing. The horse was treated symptomatically while lab work was initiated and improved enough to be released the following day; however, a mare in the same herd had died over night. The affected horses were part of a group of 30 which were maintained on grass pasture supplemented with cake. Several, including the mare, had been ridden the day before the onset of signs, and none were noticed to be ill. The veterinarian visited the ranch to do a necropsy on the dead horse and observed at least one other horse showing signs similar to the first case. This horse was treated symptomatically but became recumbent, grinding his teeth and in apparent pain, and eventually, after 5 days, had to be euthanized. Necropsy of the first horse to die was unremarkable except for diffusely petechiated lungs.

Samples were submitted to the WSVL for testing "at the discretion of the lab" with a tentative diagnosis of ionophores, mycotoxins, or botulism. Microscopic lesions consisted of intense pulmonary congestion,

diffuse splenic congestion, and scattered renal tubular necrosis. Sections of myocardium were unremarkable. Microbiology isolated only common contaminants; serology and virology were negative for EHV-1, EVA, and virus isolation on rabbit kidney cells was unrewarding.

Samples of the range cake were analyzed for monensin, lasalocid, salinomycin and narasin by an outside lab, which reported they contained 220 ppm monensin. Analysis of a separate sample by the WSVL Toxicology lab identified 250 ppm. The operator of the feed mill that produced the cake insisted that the cake couldn't be contaminated as "we don't even keep monensin on the premises", and requested a split sample for his own lab. Subsequent analysis of other dietary components (i.e. mineral) did not detect any monensin (<10 ppm), but stomach contents from the first dead horse did contain 1.4 ppm.

This case points up one of the big problems in the diagnosis of monensin poisoning from real world samples. The equine single oral LD50 of monensin is commonly cited as being between 1-3 mg/kg, although in our experience the minimum

lethal dose is probably closer to 0.1-0.5 mg/kg. Using the concentration determined in our lab (250 ppm) a horse would have to eat 0.2 – 1 kg of cake to achieve even a minimum lethal dose, or 2-6 kg to ingest a LD50. However, accidental contamination of a feedstuff, by definition, results in very heterogeneous concentrations, and the sample that is collected after the event may not be representative of what the horse ate. In any group of livestock fed as a group, some will get more than their share and some will get less. The metabolism of monensin is rapid and sufficiently complex that it is difficult or even impossible to extrapolate from stomach or tissue concentrations to a total dose.

The lesions (myocardial necrosis) of ionophore intoxication in horses are characteristic, but not pathognomonic, and may not be present in animals which die peracutely. Clinical chemistry, e.g. elevated "cardiac" isoenzymes or troponin levels are strongly suggestive, but, again, not positive proof. Thus, the clinician must often rely upon the traditional triad of clinical signs (sudden onset of weakness, ataxia, pain), lab work (pathology,

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Equine Ionophore Poisoning, *continued*

clinical pathology, EKG or ultrasonography) suggesting myocardial damage, and the presence of more than background amounts of ionophore in feedstuffs or tissues. Other agents which might produce cardiac damage such as yew, coyotillo, white snakeroot or rayless goldenrod, vitamin E/selenium deficiency and infectious diseases which cause ataxia and recumbency, should be ruled out.

Treatment is symptomatic and usually unrewarding. Mildly affected horses may recov-

er, but the owner should be warned of the possibility of chronic, sub-clinical, myocardial damage that may affect suitability for riding months after apparent recovery.

Merl Raisbeck, Toxicologist

FROM THE WYOMING DEPARTMENT OF HEALTH: Methicillin-Resistant *Staphylococcus aureus* (MRSA)

In 2011, there were nine MRSA animal patients seen by Wyoming veterinarians. In addition, veterinarians saw five animals with infections due to other methicillin-resistant staphylococcus species. In one canine patient there was an exact DNA fingerprint match (using pulsed-field gel electrophoresis) with a human case where there had been a history of direct contact between the canine and human. These cases and additional cases seen so far in 2012 have led to questions regarding measures that can be taken to reduce transmission of drug resistant strains of bacteria within veterinary hospitals.

There have been few published epidemiological studies looking at the colonization and transmission of MRSA and other drug resistant strains of bacteria in animals. Therefore caution needs to be exercised in adapting the recommendations for preventing transmission in human hospitals to veterinary hospitals. However, careful attention to infection control practices is considered by many to be the best method of transmission control in either setting^{1, 2}.

Infection control should include:

- hand washing and surface disinfection between patients
- changing of gloves between patients
- use of facial protection and protective outerwear when indicated
- isolating patients with suspected communicable diseases
- covering of any open wounds in patients
- cleaning and sterilization of instruments and other equipment

Culturing clinic staff or environmental surfaces in the management of drug-resistant organisms in human healthcare settings is generally only indicated as part of an epidemiological investigation or when there is epidemiologic evidence that either may be a source of ongoing transmission². This would seem like a reasonable approach for veterinary hospitals.

Detailed guidelines for infection control in veterinary clinics can be found in the Compendium of Veterinary Standard Precautions for Zoonotic Disease Prevention in Veterinary Personnel and in the Model Infection Control Plan for Veterinary Practices. Both of these documents are at the NASPHV website at www.nasphv.org.

Free consultation on zoonotic diseases can be obtained by calling the Wyoming State Public Health Veterinarian at 307-777-5825 or by calling the Public Health 24/7 All Hazards Response Line at 1-888-996-9104.

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