Wyoming State Veterinary Laboratory Newsletter – October 2009

University of Wyoming
Department of Veterinary Sciences
1174 Snowy Range Road
Laramie
WY 82070
http://wyovet.uwyo.edu/

Main office/Director
Phone: 307 742 6638
800 442 8331 (Toll-free Wyoming only)
Fax: 307 721 2051

To phone laboratories or faculty directly
307 742 6681 + EXT. BELOW

Mail Room	122
Virology Lab	162
Bacteriology Lab	132
Parasitology Lab	182
Toxicology Lab	233
Clinical Path Lab	182
EM Lab	151
Regulatory Serology	143
Diagnostic Serology	163
Necropsy Office	121
Dr. Merl Raisbeck	231
Dr. Chaoqun Yao	181
Dr. Ken Mills	131
Dr. Jonathan Fox	167
Dr. Shannon Swist	211
Dr. Don Montgomery	104
Dr. Todd Cornish	191
Dr. Donal O'Toole	204
Dr. Lee Belden	766 2134
Dr. Gerry Andrews	766 3139
Dean Frank Galey	766 4133

http://uwadmnweb.uwyo.edu/VETSCI/

MESSAGE FROM THE DIRECTOR

This has been a very busy summer and early fall for all of us here at the WSVL. Construction of the new addition began in June and a lot of progress has been made in just a short amount of time; the building is beginning to take shape (see photos below).

July 2009. The new addition is a big hole in the ground with some foundation poured.

Oct 2009. All of the foundation is poured, structural steel has been erected, and the cinder-block walls are going up.

I will try to include some new photos of the building addition in subsequent Newsletters. A

few minor delays have been created by wind, rain, and snow but hopefully the addition will be enclosed soon. According to our current construction schedule, phase 1 including most of the new addition will be completed in April or May and final completion (to include some renovation of the existing building) will be in August or September 2010.

INTERESTING CASES FROM WSVL AND OTHER TIDBITS

Pentobarbital Poisoning in Two Dogs
A cautionary tale

In March of this year we confirmed pentobarbital poisoning in two dogs from the Rawlins area and a family of cats from Gillette. The reported clinical signs were typical of pentobarbital poisoning, i.e. ataxia, apprehension, confusion and excitement, followed by lethargy, coma and death. The source of pentobarbital was never identified in the cats, but the owner of the dogs, a WGFD biologist, back tracked the dogs to a horse carcass on a neighbor's property. Samples taken from the dead horse by Dr. McFarland contained pentobarbital concentrations that varied from less than 20 ppm to more than 4000 ppm. There was no obvious trend to concentration in any specific tissue with the exception that 1) skin didn't contain quantifiable amounts and 2) the identity of many tissues was a guess as they were extremely desiccated.

Interestingly, the horse had been euthanized more than two years before the dogs were poisoned, and was pretty well decomposed. After the dog's owner explained what had happened the horse owner recounted losing two of her dogs with similar signs, one shortly after the horse was put down, and one more than two years later (3 months before the dogs in question were poisoned). The horse owner's second dog had been deposited in the same gulley as the horse, and tissue samples from its carcass contained pentobarbital, suggesting that it, too, had been poisoned by the carcass.

Pentobarbital relay toxicity has been reported in a number of species as a result of consuming the flesh of animals euthanized with pentobarbital and veterinarians have been fined for accidental poisoning of wildlife by euthanized carcasses that owners failed to bury. Little is known about the environmental persistence of pentobarbital in the euthanized carcass. Pentobarbital, presumably from municipal sewage, was reported to be relatively stable at low concentrations in natural waters such as lakes and streams. It also resists degradation during rendering and was detected at very low concentrations in a FDA pet food survey, presumably as the result of rendering euthanized horses. Although this report is not a controlled experiment, it does demonstrate that pentobarbital may persist at toxic concentrations for at least 2 years in a carcass under Wyoming conditions. Veterinarians should be especially careful to insist that animals they euthanize either be incinerated or buried deeply enough that scavengers can't unearth them.

Amanda Kaiser & Warner McFarland
Carbon County Veterinary Clinic
Roger Siemion and Merl Raisbeck
Toxicology Section, WSVL

Copper Poisoning in Sheep

Two lambs were submitted for necropsy from the same farm. The first, a 7 month Hampshire wether had reportedly been "a little off" for several days, and treated with Nuflor a week prior to presentation. He seemed to get better but became lethargic again on Saturday, was treated with Draxxin, and again seemed to improve. On Wednesday morning he was again lethargic and salivating and died that afternoon. The second, a Hampshire ram lamb, was fine on Sunday, but went off feed and developed a nasal discharge Monday night. By Wednesday, he

could hardly rise and he died Wednesday evening.

Necropsy findings in both animals were similar. Lungs were congested. Kidneys were diffusely much darker than normal and one animal had dark red urine suggestive of hemoglobinuria. There were two key microscopic changes that suggested copper poisoning. Both sheep had hemoglobinuric nephrosis – the presence of hemoglobin within renal tubules that is sometimes associated with secondary degenerative changes. This occurs secondary to intra-vascular hemolysis because hemoglobin is small enough to be filtered through glomeruli. Both sheep also had liver damage characterized by some loss of periacinar hepatocytes and cholestasis. One liver had detectable copper accumulation as determined using a copper specific stain.

The definitive diagnosis of chronic copper poisoning was based upon lesions of intravascular hemolysis and liver copper analysis.

The wether had been pastured with a band of ewes for the last 3 months and, in addition, received 4 lbs/day of alfalfa-based 19% crude protein pellets. The ram was penned with a Suffolk ram and getting alfalfa hay in addition to the pelleted ration. The ewes in the pasture, and a band of ewe lambs receiving the same alfalfa, were asymptomatic. Environmental samples were solicited to determine the source of the copper. Water contained negligible amounts of copper, the alfalfa 7 ppm, and "old" and "new" pellets 29 and 22 ppm Cu respectively. Upon quizzing the owner again, it was determined that the "old" pellets were near the bottom of the pile and were actually what had been fed for the last 2-3 weeks.

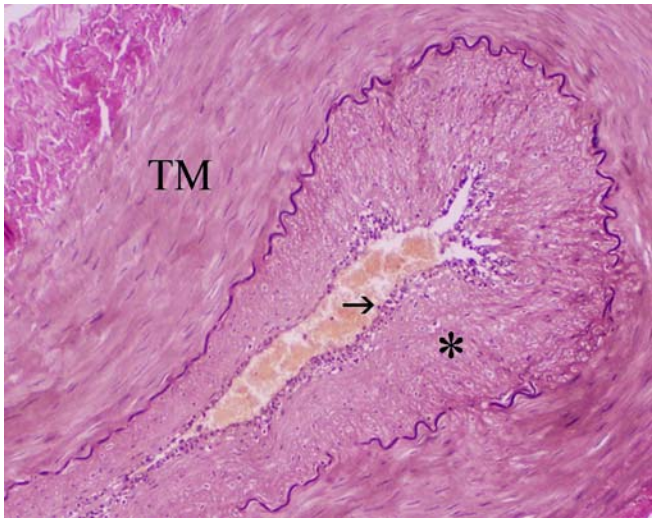
"Chronic" copper poisoning is the acute manifestation of prolonged accumulation of copper by the ovine liver as a result of the species' limited ability to excrete the element. At some point, the amount of copper in the liver exceeds the binding capacity of hepatic copper binding proteins and free (ionic) copper is released into the cytoplasm causing necrosis. As the cell dies, the remaining stores of bound copper are released in a massive cascade into the bloodstream. Free copper is toxic to virtually all cells, but red blood cells are among the most sensitive and intravascular hemolysis results. Stressors that put an extra load on the liver such as pregnancy or hepatotoxic plants may precipitate this process. Clinical signs range from lethargy and anorexia to jaundice and recumbency or sudden death. During the accumulation phase, which may last from a couple of weeks to several months, the animal is asymptomatic.

The Salt Institute recommends 7-11 ppm total dietary copper for sheep, although chronic copper poisoning has occurred at concentrations as low as 10 ppm in circumstances with unusually low dietary sulfur and molybdenum. The NRC (Mineral Tolerance of Animals, 2005) describes 15 ppm dietary copper as the "maximum tolerated concentration" in sheep with the caveat that high concentrate diets and certain genetics may predispose to copper accumulation and poisoning at lower concentrations. In Wyoming, ovine copper poisoning is rather rare as most forages are naturally deficient in copper, often high in molybdenum, and sulfur is a common water contaminant. Poisoning is usually the result of feeding supplements which contain excessive copper. Although many producers get away with using cattle supplements for sheep, this case illustrates the fact that copper concentrations that would be considered nowhere near excessive for cattle, may in fact, poison sheep.

Elaeophora schneideri in a Moose Calf

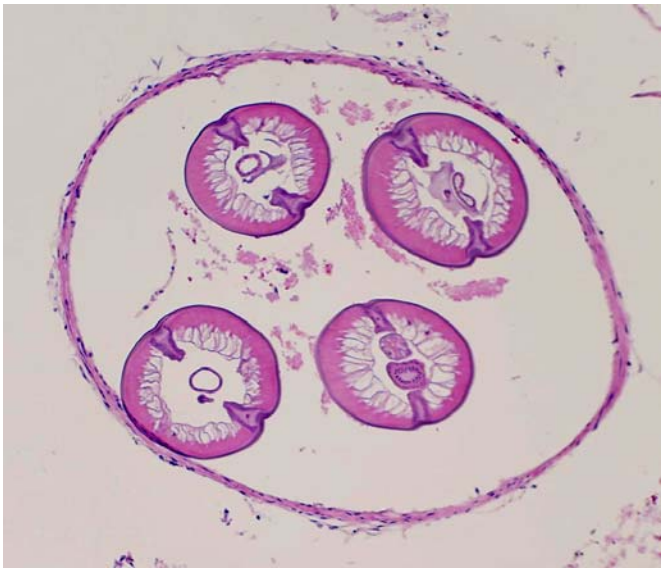
As some of you will know, there has been a decline recently in moose populations in

Wyoming. To better understand this phenomenon, the Wyoming Game and Fish Department asks that successful hunters bring heads of harvested moose to their regional offices or field check station for sample testing. The department is also willing to collect moose heads from taxidermists after they are caped. Dr. Cynthia Tate with the WGFD examines the heads following hunter harvest, and from moose showing signs of disease in the field. A disease the WGFD is particularly interested in is elaeophorisis due to *Elaephora schneideri*, the carotid worm. This parasite is well adapted to mule deer, in which infection is asymptomatic. But in some other big game species, particularly moose and elk, it can cause disease. Elaeophorosis was first documented as an endemic infection in moose in southwestern Montana in 1971 and subsequently recognized in Colorado. Adult and larval nematodes restrict blood flow in the cephalic arterial system, including letomeningeal arteries and in rete mirabile cerebri. This leads to central blindness and to necrosis of the brain, ears and nose. A characteristic lesion in carotid arteries and its major branches is thickening of the inner vascular lining.



Moderate fibrointimal hyperplasia (*) in tunica intima of branch of carotid artery of moose infected with Elaephora schneideri. The diffuse endarteritis (arrow) predisposes animals to thrombosis. TM: tunica media.

A moose calf was found moribund in September 2009 and was euthanized. A set of tissues was collected by Dr. Tate and examined microscopically. Histologically, there was moderate multifocal edema and necrosis throughout the brain. This was attributed to thrombosis in leptomeningeal vessels. Several vessels contained larval *Elaephora schneideri.* Other changes were granulomatous meningitis, and the presence of proliferative fibrointimal plaques in arteries. The presumptive cause of illness was elaeophorosis, with vascular injury leading to cerebral edema and necrosis.



Intravascular Elaephora schneideri larvae in thin-walled leptomeningeal vessel from moose calf with cerebral edema, granulomatous meningitis, and fibrointimal plaques in arteries.

The WGFD survey should result in a better understanding of the prevalence of this disease, among others, in moose, and whether it contributes to population declines. If you are contacted by hunters and asked about disease in moose, particularly if one is blind, displaying neurological signs or has necrosis of ears or muzzle, please contact Dr. Tate or other personnel in WGFD.

1. Madden DJ, Spraker TR, Adrian WJ: 1991, *Elaeophora schneideri* in moose (*Alces alces*) from Colorado. J Wildl Dis 27: 340-341.

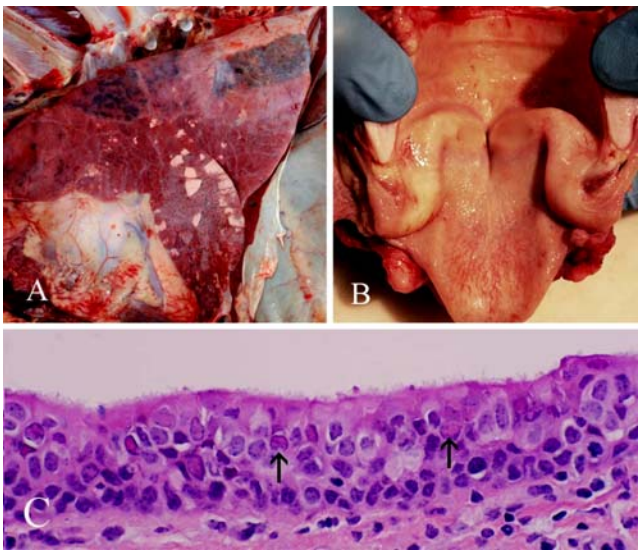
2. Worley DE, Anderson K, Creer KH: 1972, Elaeophorosis in moose from Montana. J Wildl Dis 8: 242-244.
3. Worley DE: 1975, Observations on epizootiology and distribution of *Elaeophora schnelderi* in Montana ruminants. J Wildl Dis 11: 486-488.

Cynthia Tate, WGFD
Donal O'Toole, WSVL

High Death Loss in Light-Weight Backgrounded Calves

A producer experienced heavy death loss in light-weight calves coming into a local feedlot. Losses began one week after entry with 85% morbidity and 12% mortality. The veterinarian found pulmonary abscesses at necropsy and submitted samples to his laboratory (not WSVL) for microbiology. *Histophilus somni* was isolated. The veterinarian was puzzled since he does not routinely see histophilosis in backgrounded calves. He contacted the WSVL as an article had recently been published on histophilosis. We asked him to submit samples from the next calf that died with typical clinical symptoms in order to rule out cardiac histophilosis. He was also asked to submit larynx, since there are reports that this is where *H. somni* establishes itself before septicemia develops.

The veterinarian necropsied a calf with respiratory signs. Grossly he found lungs he considered consolidated, but they floated in formalin. Formalin-fixed heart and larynx were submitted for histology. No gross or microscopic lesions were found in heart, including papillary muscles where *H. somni* classically sets up house. The larynx was grossly unremarkable, but histologically there were multiple viral-type inclusions found in laryngeal epithelium. Infectious bovine tracheitis due to BHV-1 is well recognized as an important co-factor in bovine respiratory disease complex. The limited laryngeal cellular necrosis and numerous inclusion bodies are not features typical of IBR infections. Immunohistochemical staining for BHV-1 was performed on laryngeal sections and no antigen was detected leading us to believe that some other, less pathogenic herpesvirus might be involved.



A. Lungs at necropsy showing mottling and discoloration, but no clear cut pneumonia. B. Grossly unremarkable larynx. C. Intranuclear herpesvirus-type inclusions (arrows) in arytenoid fold epithelium of larynx.

The WSVL will be doing a survey with a Wyoming veterinarian this year to establish the importance of histophilosis in backgrounded and feedlot cattle. Specifically, we wish to establish how many cattle with cardiac lesions have evidence of concurrent thrombotic meningoencephalitis (TME) due to *H. somni*, and whether upper respiratory tract lesions are present. A pre-veterinary student with a well-known Wyoming veterinary surname, Matt Asay, will be working with me, EPSCoR and Hatch funding to get this done.

O'Toole D, Allen T, Hunter R, Corbeil LB: 2009, Diagnostic exercise: Myocarditis due to *Histophilus somni* in feedlot and backgrounded cattle. Vet Pathol 46(5):1015-1017.

Donal O'Toole, WSVL

Anterior Mediastinal Hemorrhage and Hemothorax in a Dog with Anticoagulant Rodenticide Poisoning

A six-year-old Australian shepherd was submitted to a veterinary clinical with a history of “ain’t doing right”. Other than a mild neutrophilia detected on blood work, physical examination was unremarkable. The next day the dog developed severe respiratory distress and died.

On postmortem examination, major lesions were limited to subcutaneous hemorrhages and marked hemorrhage into the anterior mediastinum progressing to hemothorax. Anticoagulant rodenticide poisoning was suspected and liver analysis revealed brodifacoum.

This case seems straight forward but there is another side to the story. A syndrome of acute anterior mediastinal hemorrhage has been recognized in dogs less than two years of age but occasionally older. These hemorrhages are thought to develop in the thymic remnant and some have an underlying demonstrable cause such as anticoagulant rodenticides but other cases are idiopathic. Some cases may occur from traction on collars or harnesses that tear blood vessels in friable thymic tissue. In one case, two six-month-old Shetland sheepdog littermates were affected and no poisoning or trauma could be documented.

Coolman BR et al: Severe idiopathic thymic hemorrhage in two littermate dogs. JAVMA 205(8):1152-1153, 1994

Todd Cornish, WSVL
Merl Raisbeck, WSVL

FROM THE WYOMING DEPARTMENT OF HEALTH

Deer Associated Parapoxvirus

In early 2009, a deer hunter from Virginia and a deer hunter from Connecticut both developed nodular lesions on fingers after they had cut themselves while dressing the white tailed deer they had each shot. Parapoxvirus was later cultured from the wounds from both individuals. If the hunters did contract parapoxvirus from their deer, this would represent the first indirect evidence where viral culture was performed suggesting that wild deer in the United States are infected with parapoxvirus. Investigations are currently taking place in several states to look for the parapoxvirus in deer and also perform surveillance among hunters for any deer associated parapoxvirus infections.

Three of the most common parapoxviruses that infect animals are contagious echthyma (orf) in sheep and goats, and psuedowcowpox and bovine papular stomatitis both in cattle. The parapoxvirus cultured from the two deer hunters most closely resembles pseudocowpox virus but it appears to be a novel strain. Animals with parpoxvirus will typically develop blisters or scabs around the mouth, nostrils, eyelids or ears. Animals can also have asymptomatic infections. In humans, parapoxvirus infections develop at the site of virus penetration into the skin and manifest as papules, nodules or pustules that typically take one to two months to resolve. Parapoxvirus lesions in people can resemble cutaneous anthrax and fungal infections. Anyone developing suspicious skin lesions should contact their health care provider.

References:

Personal Communication. Amira A. Roess, PhD, MPH., Epidemic Intelligence Service Officer, Poxvirus and Rabies Branch, Centers for Disease Control and Prevention.

Zoonoses and Communicable Diseases Common to Man and Animals. Third Edition, Vol. 2, No. 3. (1 January 2003), pp. 64-66, 80-83, 242-245. by Acha, N. Pedro, Szyfres, Boris.

Karl Musgrave, DVM, MPH
State Public Health Veterinarian
Wyoming Department of Health
6101 Yellowstone Road, Suite 510
Cheyenne, WY 82002
karl.musgrave@health.wyo.gov
Cell: 307-421-8591
Office: 307-777-5825
Fax:307-777-5402