Wyoming State Veterinary Laboratory
Newsletter
April 2004

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MESSAGE FROM THE DIRECTOR

Lichen poisoning in elk

Those of you who follow the news will be aware that the WSVL and the Wyoming Game and Fish Department investigated the deaths of a large number of elk in the Red Rim area, southwest of Rawlins. The publicity the die-off received is a reminder of strong national public interest in the health of Wyoming's wildlife – and the laboratory's diagnostic role so that wildlife managers can prevent future losses.

This issue of the newsletter contains a summary of the investigation and where we are at the moment. The tentative diagnosis is lichen toxicosis. The causative lichen is *Xanthoparmelia chlorochroa*, a lichen found throughout Wyoming (initial press releases called it *Parmelia*, an old name for the organism). The laboratory made the decision that WGFD would take the lead in the field investigation and in coordinating press releases. Dr. Walt Cook, a WGFD veterinarian and former PhD graduate student of Dr. Beth Williams, was the conduit through whom field and laboratory information flowed. Walt did a top-notch job, particularly given the interest of the media and concerned members of the public.

WGFD biologists asked the question whether ground lichen that is abundant in the Red Rim area where losses occurred might play a role in the deaths. Dr. Merl Raisbeck used his connections with the veterinary diagnostic toxicology community to solicit ideas and suggestions. One colleague remembered an obscure reference to ground lichens being toxic. Dr. Raisbeck tracked down the reference, which was in a publication put out by the Wyoming Agricultural Experiment Station in 1953. In it was a report that cattle and sheep could be intoxicated by ground lichens. The clinical signs (bright, alert, recumbent animals) fit those in the elk. On that basis, Dr. Terry Kreeger exposed three elk in the Sybille facility to *Xanthoparmelia chlorochroa.* Two of the elk developed clinical signs similar to those seen in the affected elk; the results of histological examinations are pending.

These studies need to be expanded and refined. Drs. Raisbeck and Cook are planning to do additional studies to get a better understanding of how and why ground lichen intoxicates ruminants. Other than the 1953 AES report, there are no reports in the peer-reviewed literature of poisoning with *Xanthoparmelia*. Lichen have, until now, been considered harmless. Such studies have broader implications than the Red Rim die off. Cattle and sheep eat lichens, particularly late in winter when rations are short, so there are implications for the livestock industry. *Xanthoparmelia* and other ground lichens are used in the health food industry, so it may also have ramifications for human health.

Donal O'Toole

ELK DIE OFF ON RED RIM, SOUTHEASTERN WYOMING

Background: Recumbent adult elk were found on or shortly before 6 February 2004. New cases of the syndrome continued to be seen by wildlife managers until late March 2004. All were in the Red Rim wildlife habitat management area, a 40 - 50 square mile area southwest of the town of Rawlins, WY just east of the Sweetwater-Carbon county line (WGFD elk hunting area 108). Most affected animals were adult females; affected calves are underrepresented. This is a largely cow-calf herd, which is presumed to explain the paucity of affected bull elk. A herd of 400 healthy elk was seen feeding in the area in late January. Losses are assumed to have begun between 26 January and 6 February 2004. On 31 December 2003, a recumbent elk was found 0.5 mile south of the Ferris RR Crossing, 1 mile west of Rawlins, approximately 11 miles east of the Red Rim outbreak. This animal fits the case definition (below) and may be the first one affected in the outbreak. Estimate of total losses are 310 elk. Losses have now ceased. The affected herd has moved out of the Red Rim area. No other species, including resident cattle and pronghorn, were affected. Press releases on the investigation are posted on the Wyoming Game and Fish Department (WGFD) web site: http://gf.state.wy.us/

Case definition: Affected elk display progressive weakness and are generally found down, typically in sternal recumbency, and unable to rise. Elk are afebrile, with normal pulse and respiration. They display normal mentation. Affected elk do not survive, even if offered food and water. Animals are generally in fair-to-good nutritional condition. Findings at necropsy are minimal: some elk have muscle pallor, particularly of the thigh, corresponding to acute myopathy histologically. Many affected elk have moderately distended large bowels (small colon and rectum) that are filled with feces of normal consistency. Discolored (red-brown) urine was found in the vicinity of many affected elk. The bladders of affected elk contained urine of normal color.

Attempted treatment: Four elk with typical clinical signs were collected in the field and transferred to the WSVL, where Dr. Cook treated them for up to 17 days. Elk were treated with intravenous fluids, and injections of vitamin A, D, E and selenium. None responded. All were euthanized.

Disease investigation: A disease investigation was begun as soon as the large scale of the outbreak was recognized and was coordinated by Dr. Walt Cook of WGFD. Field data were collected by WGFD personnel, who conducted a feeding trial using ground lichen. Laboratory work was performed at the Wyoming State Veterinary Laboratory (WSVL), whose personnel did necropsies and collected samples in the field. WGFD personnel recorded dates on which individual affected elk were found, as well as sex, age and GPS locations. Data were entered on GIS maps, overlaid with geographic and vegetation data. These are currently being analyzed.

Data indicating lichen intoxication:

WGFD employees noted that elk in the Red Rim area were eating lichen (*Xanthoparmelia chlorochroa*). Digested lichen was present in the rumen contents of affected elk at necropsy. Three captive adult elk held at WFGD's Sybille facility were offered a diet of lichen between 15 – 25 March 2004. Two of three displayed clinical signs of weakness 7 and 10 days after exposure respectively, and were euthanized and examined post-mortem. The remaining elk ate little of the lichen diet, showed no clinical signs and was returned to a normal diet after 10 days. Histological examination of tissues is ongoing. Additional experimental studies will be undertaken by personnel from the WGFD and the WSVL. A large volume of lichen was collected from the site where elk were affected and will be fed to animals to define the pathogenesis and toxicity of the lichen.

Lichen in the diets of elk, pronghorn and livestock: Lichens are composite, symbiotic organisms composed of members from as many as three kingdoms. The dominant partner is a fungus (kingdom Fungi), which cultivates partners that manufacture food by photosynthesis, either algae (kingdom Protista), or cyanobacteria (blue-green algae) (kingdom Monera). The lichen implicated in the current episode is *Xanthoparmelia chlorochroa* (Tuck.) Hale (Syn.: *Parmelia chlorochroa*) ("tumbleweed shield lichen"). Information on lichen as a food item used by elk is limited. Elk are reported to eat *Bryoria trichodes*, spp. *Americana*, *Ramalina menziesii*, and *Usnea* sp. (*U. barbata* and *U. plicata*, cited, are misidentifications). Marcum, 1980, reported an average of 3% volume of lichens occurring in 33% of rumens sampled from a western Montana herd in Oct.-Nov., 1972. Most forage studies of elk do not list lichens. They are probably used as occasional winter forage, especially at times of stress. Roosevelt elk (*C.e. rosevelti*) eat tree lichens during winter, especially at higher elevations; they are important in the diet and are taken from as far up as the animals can reach. Reports of intoxication with *Xanthoparmelia* spp. are sparse. Poisoning of "laboratory animals" fed "an emulsion of usnic acid extracted from lichen identified as *Parmelia molluscula* Ach is mentioned in two personal communications (OA Beath, Wyoming Agricultural Experiment Station 1960; WT Huffman, Salina Utah Experiment Station 1955) in Kingsbury's text *Poisonous Plants of the United States and Canada*. The remaining fraction, devoid of usnic acid, was not toxic. The lichen contained approximately 1.6% of usnic acid, which was extracted with carbon disulfide. The species identification is incorrect as *Parmelia molluscula* does not occur in North America (TL Esslinger: *A cumulative checklist for the lichen-forming, lichenicolous and allied fungi of the continental United States and Canada*, version #8, posted 17 July 2002). Bulletin 324 of the Wyoming Agricultural Experiment Station (*Poisonous Plants and Livestock Poisoning*; July 1953) has a brief section on intoxication produced by ground lichen identified as *Parmelia molluscula.* It stated that it was not a serious menace to livestock, and that it caused poisoning in winter when forage was scarce. The lichen was toxic year-round. Although the report states that "the nature of the poison and

symptoms produced have been investigated quite thoroughly," Bulletin 324 is the only publication where the investigations are recorded. Signs consisted of "lack of coordination in movement of the hindlegs," with mild depression and inability to move either forelimbs or pelvic limbs in more severe cases. The authors stated that no lesions or characteristic pathology developed. Unlike sheep, cattle continued to eat and drink while recumbent. Subcutaneous injections with strychnine sulfate were stated to be beneficial for affected cattle, but not sheep. The authors concluded that the toxic principal affected either the spinal cord or cerebrum. A toxic dose was reported to be 1% of an animal's weight for 5 days, or a single dose of 3.6% of an animal's weight. There is no record of the *X. chlorochroa* occurring in elk. Other species in the Red Rim area, such as pronghorn, are reported to eat lichens including *X. chlorochroa.* Domestic sheep grazed some of the same ranges and were thought to compete with antelope for the same lichens. Wildlife biologists with the Bureau of Land Management and the USDA Forest Service in Nevada and New Mexico used the presence of *X. chlorochroa* as an indicator of excellent antelope range for a number of years. They considered it "fair" forage in spring, and "poor" forage in summer.

April 4, 2004

Beath OA, Gilbert CS, HF Eppson, Rosenfeld I: 1953, Poisonous plants and livestock poisoning. Bulletin of the Agricultural Experiment Station, University of Wyoming, pp. 12 – 16.
Kingsbury JM: 1964, *Poisonous Plants of the United States and Canada*, pp. 86 – 87, Prentice-Hall, New Jersey

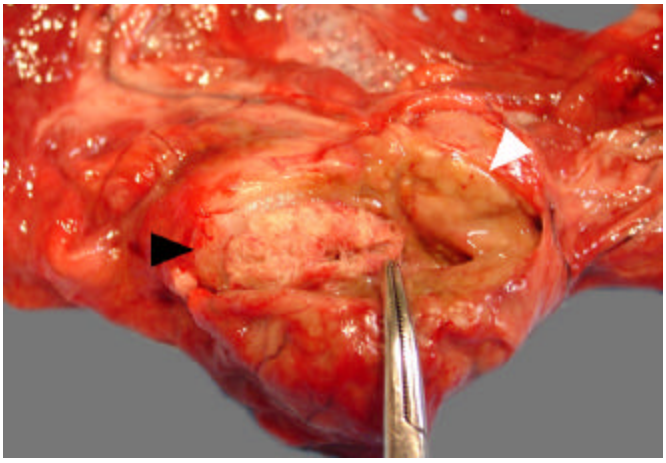
RECENT CASES

Peritonitis associated with 6 gauze sponges in a dog

A veterinarian in eastern Wyoming was recently presented with a 93 lb St. Bernard bitch that had not gained weight since overohisterectomy was performed out-of-state a year earlier. The dog had developed lethargy and periodic bouts of vomiting. Palpation revealed intra-abdominal masses. An exploratory laparotomy revealed multiple masses around the bowel. One large abscess compressed the lumen of the large intestine.

The laboratory received a 13.0 x 8.0 x 2.5 cm length of large bowel with attached opened abscess, and a 32 x 15 x 2.8 cm mesenteric mass. The bowel was focally stenotic. The histological lesions consisted of severe diffuse chronic fibrinous/sclerosing and pyogranulomatous peritonitis, and severe transmural pyogranulomatous entertis.

The dog survived surgery, put on weight and began to act like the young dog she was.



Gauze-induced peritonitis in a St. Bernard dog, presumably a sequel to leaving 6, 4 x 4 inch gauze sponges during an overohisterectomy one year earlier. Black arrowhead: sponge. White arrowhead: wall of abscess.

There is a good reason that surgeons count their surgical sponges. Retained gauze sponges induce florid foreign body reactions, as in this case. They are sufficiently well known to have their own medial term: gossypiboma (L: *gossypium* – cotton). Chronically retained sponges break down to semi-liquid material consisting of small fragments that cannot be removed in one piece. There is a surprisingly rich literature of this iatrogenic complication of surgery: gauze sponges have been left in the brain, nasal passages, pericardial sac, limbs (after bone surgery), abdominal cavity, and pleural sacs. In addition to the gainful employment these cases give to our legal brethren, gauze sponges may be associated with the subsequent development of neoplasia. The most common complication is a large foreign body reaction that is be mistaken for a neoplasm.

Kumar PV et al: 2001, Fine needle aspiration cytology of foreign bodies presenting as cystic abdominal masses. A report of three cases. Acta Cytol. 45(2):245-248.
Pardo AD et al: 1990, Primary jejunal osteosarcoma associated with a surgical sponge in a dog. J Am Vet Med Assoc. 1990 Mar 15;196(6):935-8.

Ureaplasma abortion in a beef herd in northeastern Wyoming

A producer in northeastern Wyoming had 5 abortions in his 300 cow herd. The typical presentation was abortion in the last month of pregnancy with placental lesions, hepatic discoloration and hemolyzed serum. The placenta was generally retained, with subsequent metritis. All ages of animals in the herd were affected.

We received an excellent set of samples and a good history. In the lung there were peribronchial aggregates of lymphoid cells, with mild bronchointerstitial pneumonia. There was a necrotizing placentitis with mineralization and vasculitis. Stains for bacteria were negative. Based on the lesions, Dr. Montgomery requested PCR testing for *Ureaplasma*, a method recently adapted to the WSVL by Amy in the bacteriology laboratory, and the lung tested

positive. Bacterial culture was negative. No viruses were isolated. Serology ruled out leptospirosis.

This is the first time the WSVL has made a tentative diagnosis of *Ureaplasma* abortion in Wyoming. Ureaplasma is a small bacterium resembling *Mycoplasma*, but distinct due to its ability to hydrolyze urea. In addition to abortion, it has been associated with vulvitis, endometritis, salpingitis, and infertility. Abortion is associated with placental necrosis and placentitis, and fetuses have helpful peribronchiolar lymphoid aggregates with pneumonitis ("cuffing pneumonia). Infection of normal cattle with this organism is common, and it can be isolated from the reproductive tract. In a recent study using vaginal swabs from healthy cattle, *U. diversum* was positive by culture in 35.7% (60 samples) and by PCR in 52.9% (89 samples). Treatment is with oxytetracycline.

Vasconcellos Cardoso et al: 2000, Detection of Ureaplasma diversum in cattle using a newly developed PCR-based detection assay. Vet Microbiol. 72(3-4):241-250.

Outbreak of canine distemper in stray dogs in Lander-Riverton area

March 27, 2004: The Wyoming State Veterinary Laboratory confirmed a diagnosis of canine distemper (CD) in an 8-month old unvaccinated dog from Lander. The dog presented with suggestive clinical signs of CD, including conjunctivitis, rhinitis, muscle fasciculations and neurological signs including ataxia. Findings at necropsy were of severe bilateral purulent conjunctivitis, hyperkeratosis of all foot pads, thymic atrophy, and pulmonary edema. Histological lesions were typical of CD, and included demyelination and malacia in cerebellum and brainstem, with typical CD inclusion bodies. The diagnosis was corroborated by positive fluorescent antibody staining, and the presence of typical nucleocapsids when samples were examined by electron microscopy. Attempted virus isolation is ongoing.

One of the veterinary clinics in Lander has seen 5 other dogs with similar clinical signs, although a diagnosis was not confirmed in these animals. The clinic reports that stray dogs on the Wind River Indian Reservation have signs of CD, and that some of these dogs are being shot to control the disease. A litter of puppies acquired in Riverton and transported to Cody developed clinical signs of CD. Samples from these animals are currently being tested for CD. The presence of neurological signs and history is strongly suggestive of the disease.

The disease: Canine distemper is a **highly contagious**, systemic, viral disease of dogs that occurs worldwide. It is relatively uncommon in Wyoming, but it occurs in wildlife and in unvaccinated or very young dogs. The disease is characterized by diphasic fever, leukopenia, gastrointestinal and respiratory catarrh, and frequently pneumonic and neurologic complications. CD occurs in *Canidae* (dogs, coyotes, and foxes), *Mustelidae* (eg, ferret, mink and skunk) and *Procyonidae* (eg, raccoon); fatal infections have been found in large exotic cats (e.g. lions). It causes epizootic periodically in some free-ranging coyote, skunk and raccoon populations in Wyoming.

Laboratory diagnosis: In dogs with multisystemic signs, conjunctival, tracheal, vaginal or other epithelium, or the buffy coat of the blood can be examined by immunofluorescent assay, supplemented where appropriate by negative stain electron microscopy (on feces) at the Wyoming State Veterinary Laboratory. These samples may be negative when the dog is showing only neurologic manifestations or when circulating antibody is present (or both). The diagnosis can then be made by serologic demonstration of serum neutralization titers. Post-mortem diagnosis requires submission to the WSVL of a carcass or a wide range of fresh and fixed tissues, focusing on those tissues where the virus is likely to be found: brain, stomach, lung, kidney, bladder, conjunctiva and eye. The WSVL has a specific immunohistochemical test for use on formalin fixed tissues to confirm a diagnosis of CD.

Treatment: This is directed at limiting secondary bacterial invasion, supporting the fluid balance and overall well-being of the dog, and controlling nervous manifestations. Antibiotics, electrolyte solutions, protein hydrolysates, dietary supplements, antipyretics, nasal preparations, analgesics, and anticonvulsants can be used. No one treatment is specific or uniformly successful. Dogs may recover completely from systemic manifestations, but good nursing care is essential. Despite intensive care, some dogs do not make a recovery. Treatment for neurologic manifestations of distemper is unsuccessful. If the neurologic signs are progressive or severe, the owner should be appropriately advised.

Control: The most effective form of control is **VACCINATION**. Puppies are vaccinated with MLV vaccine when 6 weeks old, and at 2- to 4-week intervals until 16 weeks old. Measles virus is in the same family as canine distemper virus (CDV) and it can induce immunity to CDV in the presence of relatively high concentrations of maternal distemper antibody. An MLV measles vaccine and a combination MLV measles-MLV CDV vaccine is available. These vaccines must be administered intramuscularly. Pups 6-7 weeks old should receive the measles or combination vaccine and at least two additonal doses of MLV distemper vaccine when 12-16 weeks old. Many varieties of attenuated distemper vaccine are available and should be used according to manufacturers' directions and with the advice of a veterinarian. Annual revaccination is suggested because of the breaks in neurologic distemper that can occur in stressed, diseased, or immunosuppressed dogs. On rare occasion commercial vaccines may induce iatrogenic CD.

Idiopathic thymic hemorrhage in young dogs

A practitioner submitted tissues from a 6-month-old female Labrador retriever with a history of polyuria / polydipsia, persistent lipemia and glucosuria. The dog died peracutely. Necropsy performed by the submitting veterinarian indicated massive thoracic hemorrhage as the

immediate cause of death. Testing at WSVL for a variety of anticoagulant compounds was negative. Histopathology on submitted tissues revealed diffuse hepatocellular fatty change, indicative of a metabolic disturbance that would support a clinical diagnosis of diabetes mellitus. A source or cause of the massive thoracic hemorrhage was not identified. In the absence of trauma and no analytical evidence of anticoagulant poisoning, idiopathic thymic hemorrhage was suggested.

Acutely fatal hemorrhage of the thymus is a sporadic disease predominantly of young (< 2-year old) dogs. The cause of the syndrome is unknown. We see approximately 1 case/year at WSVL. Dogs generally present with signs of acute blood loss, such as pale mucous membranes and weak pulse. The clinical course is usually short (<24 hours), although some histological studies suggest some dogs have subclinical episodes prior to a fatal hematoma. Dyspnea may occur when mediastinal hemorrhage progresses to hemothorax. It is assumed that the thymus loses tissue support as it involutes, and vessels dilate and rupture spontaneously. Gross findings consist of cranial hemomediastinum with no evidence of bleeding in sites other than thorax. There is one report of surgical intervention with successful removal of the hematoma, without recurrence, and another of successful medical treatment. German shepherd dogs were over-represented in two case studies. A similar disease occurs in people. Diabetes is not a component of this syndrome in dogs but may have been an underlying disease process.

Klopfer U, Perl S, Yakobson B, Nobel TA: 1985, Spontaneous fatal hemorrhage in the involuting thymus in dogs. . J Am An Hosp Assoc 21 (2) 261-264.
Glaus TM, Rawlings CA, Mahaffey EA, Mahaffey MB: 1993, Acute thymic hemorrhage and hemothorax in a dog. J Am An Hosp Assoc 29 (6) 489-491
Coolman BR, Brewer WG, D'Andrea GH, Lenz SD: 1994, Severe idiopathic thymic hemorrhage in two littermate dogs. J Am Vet Med Assoc 205 (8), 1152-1153 and 206 (2): 156-157
Van der Linde-Sipman JS, Van Dijk JE: 1987, Hematomas in the thymus of dogs. Vet Pathol 24: 59 – 61

Salmonella infection in Wyoming foals

We have recently had two cases of bloody diarrhea in foals from which we isolated *Salmonella*. The differential on both cases included *Clostridium* because of hemorrhage in the intestine. There were differences in that one case involved only a short intestinal segment, whereas in the other it was much more extensive. The variation in clinical presentation has been seen with entertoxemia in calves and probably led to the field diagnosis of Clostridium. Bottom line - confirm the diagnosis because as you all know once you have *Salmonella* in a facility it is difficult to eliminate. Early diagnosis and isolation will help keep the bacterial numbers from getting out of hand.

Dr. Ken Mills

Equine Herpesvirus-1 Associated Abortion Storm

We have recently been investigating a serious abortion storm resulting in loss of many near term and term foals. The herd was vaccinated for influenza, equine encephalitis, and West Nile virus and treated with ivermectin 4 days before abortions started. Necropsy and microbiology on three foals indicated equine herpesvirus 1 (EHV-1) as the cause of abortion. Mild pulmonary edema and multiple pale foci of necrosis were scattered throughout the livers of the aborted foals. Large intranuclear inclusion bodies and foci of necrosis were found in many organs when the tissues were examined microscopically; these inclusions are a tip-off that viruses are behind the fetal deaths. The virology laboratory isolated EHV-1 from the tissues of the foals; no other pathogens were identified.

Equine herpesvirus 1 is probably the most commonly diagnosed cause of infectious equine abortion. Abortions usually occur after 7 months of gestation and typically near term. Individual mares in a herd may abort or there may be epidemic loss of foals. The virus causes respiratory tract infection, viremia, and transplacental infection of the fetus; abortion may follow weeks to months following infection. Signs of respiratory disease may or may not been seen in infected horses. Residual reproductive problems are not seen with EHV-1 infection in mares.

Killed vaccines are used to protect mares and other horses on a premise against EHV-1 infection. For best protection against EHV-1 abortion, mares can be vaccinated repeatedly during pregnancy; typically in gestation month 5, 7, and 9. Management of pregnant mares can also decrease the likelihood of EVH-1 infection and abortion. Avoid mixing pregnant mares from different sources, if possible separate pregnant mares from other horses, and minimize stress to reduce the likelihood of reactivating latent viral infection.

In the last few years, there has been a lot of concern among horse owners that vaccination with West Nile virus (WNV) vaccine causes abortion or fetal deformities. There is no scientific evidence that this is the case. According to USDA, APHIS "Horse owners should be assured that the vaccine is safe, and it should be used as protection against the West Nile virus. Millions of doses of the vaccine have been used since USDA's Center for Veterinary Biologics approved its use in 2001." Although the mares on this premise were vaccinated several days prior to the start of abortions, the cause of abortions was EHV-1 infection and not WNV vaccine. Unfortunately, the aborting mares had not been vaccinated for EHV-1 and the association with WNV vaccination was coincidental. This case highlights the need to educate horse owners about the availability and necessity to use equine vaccines that protect against potentially significant viral infections of horses including EHV-1 and WNV.

Dr. Beth Williams
Pathology

Carbamate poisoning (Temik®) in Jackson area

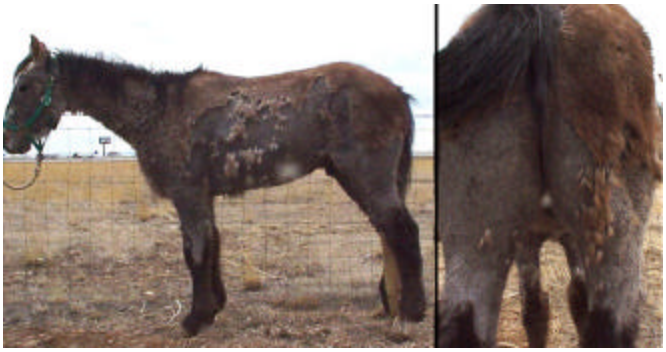
The laboratory received samples from two cases of carbamate poisoning in the Jackson area. Presentation was fairly typical of cholinesterase inhibition: hyperptyalism, tachycardia, tachypnea and miosis. Atropine and activated charcoal seemed to help clinical signs in one case. The second dog presented in worse condition and subsequently died. Blood cholinesterase was depressed in the first dog and essentially zero in the second. A hotdog full of black granules yielded aldicarb (Temik®) by TLC, a result later confirmed by GC/MS analysis. Several more suspect cases are currently being investigated.

It's interesting that the blood cholinesterase was nearly normal in the first dog a few hours after she had vomited and was treated with charcoal. In my experience, clinical signs don't usually occur until cholinesterase has been depressed to a small fraction of normal. The apparent rebound in activity illustrates the usefulness of early aggressive intervention to clean out the gastrointestinal tract. I recommend enterogastric lavage followed by activated charcoal. Atropine is useful in controlling some parasympathetic signs, but it doesn't do anything about the poison *per se*. It must be repeated every few hours to be effective. As animals become refractory to atropinization fairly quickly, it's a good idea to use only enough to control secretion so as to prolong its effectiveness.

A conundrum facing the clinician dealing with a cholinesterase inhibitor poisoning is whether or not to treat with a cholinesterase reactivator such as 2-PAM. The old dogma insisted that you must know that you're dealing with an organophosphate before using oximes such as 2-PAM. 2-PAM can potentiate the effects of at least one of the carbamates. However, to be effective the drug had to be administered within 24 hours of exposure to the pesticide, which is too early to get laboratory identification of the agent. Recent research suggests that oximes are contraindicated only with a few carbamates, and are harmless in most. The odds favor using of 2-PAM in cases of apparent organophosphate poisoning unless you are reasonably sure that you're dealing with a carbamate. 2PAM is available from most veterinary distributors in the area. Salespeople may not be aware that their company carries it since it is a low volume item.

Another problem with this type of case is the potential for legal ramifications. It's easy to lose sight of this fact in the excitement of treating an acute poisoning, and, in fact, these cases seldom actually make it to court. The few that do, however, more than justify spending extra time on paperwork. Make sure that you record all observations, any history and any contacts you have with owners, police and the laboratory. Please call to let the laboratory know that you're sending samples, and take some simple steps to maintain a chain-of-custody. The idea of chain-of-custody is to demonstrate that the sample you took made it to the analyst's bench without tampering or confusion with another sample. Contrary to urban legend, this doesn't have to be a ridiculously complicated, form-laden process like many law-enforcement agencies use. A simple procedure I've used for years is to seal a signed letter with the history (and requesting a return receipt) in the box with the samples and use similarly signed gummed labels to seal the box. The laboratory technician cuts the label to open the box, compares the signature on the (hopefully) undamaged label to the letter, makes a copy of the letter with a note about when and who received it and sends it back to you for your records.

Dr. Merl Raisbeck
22 April 2004



Pemphigus in a young horse with "sudden onset." The horse has severe diffuse crusting dermatitis of the trunk and proximal aspects of the limbs. The horse had clinical signs suggestive of laminitis.

Surveillance for BSE in adult Wyoming cattle

The USDA announced in March that it is undertaking a national surveillance program for BSE. This is in the wake of laboratory-confirmed BSE in an imported Canadian cow in WA on 23 December 2003. The surveillance is planned to occur over one year. It is in response to a recommendation made by an advisory panel to Secretary Veneman that the USA looks harder for BSE. Until now the number of cattle examined for evidence of BSE has been small (20,543 in FY 03). USDA APHIS is charged with increasing the number of animals examined to 268,500. This is calculated to detect BSE occurring at a rate =1 positive in 10 million adult cattle with a 99 percent confidence level.

The odds of finding BSE in Wyoming cattle are small. Imported dairy cattle from Canada, particularly Alberta, are rare in the state. Our dairy industry is small, and this is the population most likely to have been fed prion-contaminated meat and bone meal. There is a remote possibility that BSE may occur as a spontaneous disease of cattle. There is a report from Italy of a distinctive form of BSE characterized by a large amount of amyloid in the brain. The disease is provisionally called bovine amyloidotic spongiform encephalopathy (BASE). The Italian authors

speculate that it resembles the sporadic (spontaneous) form of Creutzfeldt-Jakob disease.

Based on a target population of 268,500 downer and old cows, the USDA estimates that 2,513 of these (1% national total) occur in Wyoming. Dr. Bret Combs and Dr. Jim Logan, representing the USDA and State veterinarian's office respectively, submitted a draft plan for the number of animals that they estimate their staff can get to in order to examine for BSE in Wyoming. Due to the size of the state and the predominance of ranching operations, their best estimate is that they will obtain approximately 240 brain samples for BSE testing. How this will be done, and where they will be tested, remains to be established. The USDA selected 7 accredited diagnostic laboratories to serve as 'high-volume" laboratories for evaluating brains for BSE. These are in CA, CO, TX, WI, WA, GA, and NY. There was consternation among other accredited diagnostic laboratories, including the Wyoming laboratory, that the USDA decided on a centralized rather than a state-based testing approach. The WSVL has considerable experience with rapid testing for BSE. Dr. Williams' research laboratory is equipped to do this. The 7 laboratories will receive federal funding for automated testing equipment. If they have an animal whose brain tests "positive" on a rapid ELISA test, they will refer samples to the National Veterinary Services Laboratory, where the test will be repeated and validated. The USDA will determine whether an animal is officially positive for BSE.

At this time I am not sure what role the WSVL will play in the one-year surveillance testing, or how we will respond to requests from you to test for BSE. The USDA has made provision for other laboratories to test on a "fee-for-service" basis. These laboratories must be approved to do the rapid test by APHIS. One criterion (test 1,000 brain samples/month) will be hard for us to meet, unless we serve as a regional laboratory for adjacent states. Many laboratory directors are unhappy with the centralized testing approach and we are unsure what impact this will have when clients have a downer cow and want us to rule out BSE and check for other CNS diseases, including rabies. Dr. Bret Combs, Dr. Logan and I will try to come up with a plan that meets both USDA requirements and allows the WSVL to offer you a service to examine the brains of clients' cattle that display signs of disease.

Regardless of where testing is done, we need your assistance in sampling and submitting brains from cattle that fit the criteria of "at-risk" animals for BSE during the coming year.. Establishing that BSE is a rare disease in the USA is critical to keep export markets open for US cattle and for the meat trade.

Casalone C, Zanusso G, Acutis P, Ferrari S, Capucci L, Tagliavini F, Monaco S, Caramelli M: 2004, Identification of a second bovine amyloidotic spongiform encephalopathy: molecular similarities with sporadic Creutzfeldt-Jakob disease. Proc Natl Acad Sci U S A. 2004 Mar 2;101(9):3065 - 3070.

BRUCELLOSIS TESTING AND TEST INTERPRETATION BY USDA

With Wyoming now in Class A status, there will be a lot of testing on ranches and at livestock markets. The USDA is required to follow the Brucellosis Uniform Methods and Rules (UM&R).

When a test reveals a *card-positive, rivanol-negative* animal, that **individual** is quarantined. Remaining animals that test negative are free to move.

If a test reveals a *card-positive, rivanol-positive* animal, **the entire group of cattle** is quarantined until the tittered animals are re-tested and the titer responses resolved. The owner's copy of the test along with the quarantine notice will be mailed from the Wyoming Livestock Board's office. Quarantined cattle can be "S" branded and consigned to slaughter only on an I-27 shipping permit.

Questions about test results, quarantine, re-testing, or movement restrictions can be answered by calling the Wyoming Livestock Board at 307 777 7515 or USDA Veterinary Services at 307 772 2186.

Dr. Mark Stewart
April 21, 2004

Note: hemolyzed blood samples tend to give false positive results for brucellosis. Please avoid these sorts of samples.

Brucella serology on abortions

The WSVL encourages people to get a blood sample from each aborting dam and have it checked for Brucellosis. We realize that you may not want to go to the expense of a full work-up on each abortion but a single serum sample could be sent in to rule out Brucellosis. Brucellosis testing is not charged so your costs would be minimal.

Dr. Ken Mills

Governor's Brucellosis Coordination Team

The Governor's Brucellosis Task Force was assembled by Governor Dave Freudenthal for the primary purpose of finding effective long-term solutions to the brucellosis problem in Wyoming.

The Task Force is comprised of a diverse group of individuals from both public and private sectors with varied backgrounds and interests. The Task Force also has several technical advisors and is chaired by Dr. Frank Galey, dean of the University of Wyoming College of Agriculture. The Task Force will meet at least monthly at various locations around the state. The meetings are organized to provide educational information, have focused discussions and allow for public comment.

More information can be found at
http://wyagric.state.wy.us/relatedinfo/govbrucecoordinati.htm