Wyoming State Veterinary Laboratory Newsletter – December 2006

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MESSAGE FROM THE DIRECTOR

On difficult clients

I recently heard a talk on the transmissible spongiform encephalopathies given by Dr. Jean Jewell in Dr. Bratanich's virology class. Dr. Jewell reminded upper division undergraduates of the difficult time that Dr. Stan Prusiner had when he presented data that proteins could be infectious. He made what was then a startling suggestion: that proteins might cause transmissible spongiform encephalopathies. He was laughed at for some years. But Prusiner was data-driven. He generated enough experimental data that skeptics, including myself, were persuaded. The nice thing was that he got a Nobel prize for his laboratory's work.

Unfortunately, not all clients go for evidence-based science. You occasionally meet these folks in your clinic. We also have our skeptical clients Neither of us is likely to receive a Nobel prize when we present clinical or laboratory data to a skeptic.

Recently, a producer presented the laboratory with a complex disease situation. It has a legal component, so I must be vague here. Suffice it to say that the producer was convinced the problem was due to one thing, and one thing only. He and his legal folks considered it the laboratory's job to give him supportive data. He was not a fan of the laboratory, since to date we had not given him the result he wanted. Nor did he like the D-laboratories in Colorado or Nebraska.

I duly retrieved the reports from our system. I found that - even though heavy losses had occurred - we received two (n = 2) pertinent accessions since the wreck began. One was a necropsy in a bag - the tissues were rotten. The other, also based on a field necropsy, did provide the owner with a cause of death. We corroborated this by isolating a causative agent that matched the lesions seen by the pathologist. Yet this was not what the owner wanted to hear. Therefore he did not hear it.

People, as the Jim Morrison song goes, are strange. We were then directed to do a large amount of testing to establish in Oct/Nov why his animals died back in February. And lo! We came up with some answers that explained part of the death loss. But it was still not the answer the client wanted. We could have continued to do a large amount of irrelevant testing, until such time that the client will say: Enough already, my wallet hurts - stop.

I am not sure at what point people are persuaded by facts - in our case, by laboratory results. Clearly, with this client, we failed to provide an answer he wanted to hear. All we can do

is generate results and hope the client accepts the management consequences that should flow from them.

My favorite quote in such situations is from Senator Daniel Patrick Moynihan: "Everyone is entitled to their own set of opinions, but not to their own set of facts."

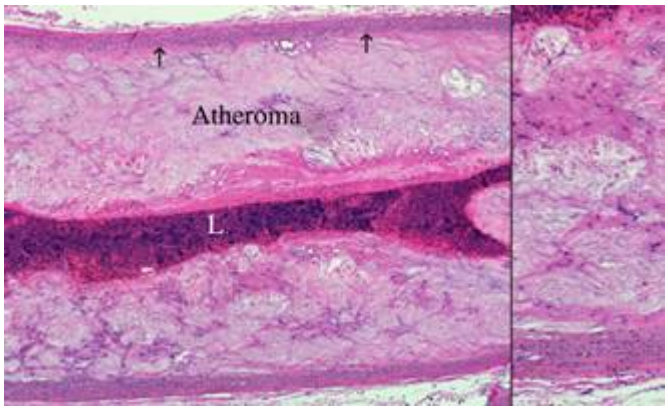
Donal O'Toole
December 12, 2006

DIAGNOSTIC CASES OF INTEREST

Sudden death in a parrot due to atherosclerlosis

Dr. Woods was presented with the carcass of a 16-year old male African Grey parrot with a history of sudden death. The bird was well cared for. According to the veterinarian who submitted the carcass, the owner was knowledgeable about birds.

Grossly Dr. Woods found marked thickening of the great vessels of the heart and of the carotid arteries, and hemopericardium. Histologically there was severe atherosclerosis.



WSVL 06A15064: Carotid artery of an African Gray parrot that died acutely. Note marked intimal thickening resulting in narrowing of the lumen of the carotid artery. Arrows point to muscle wall of artery. Everything between that and the lumen (L) is the atheroma. Image at right shows a higher magnification of atheroma, composed largely of lipid-laden macrophages and cholesterol clefts.

Atherosclerosis is common in parrots. In most cases, it is considered an incidental finding. In this animal, due to the severity of lesions, it was probably significant.

African Grey and Amazon species are particularly susceptible to atherosclerosis. In one survey, 92.4% of the former and 78.4% of the latter had evidence of atherosclerosis, although other surveys suggest a lower incidence in African Grays. Age and genetic susceptibility are established risk factors. African Grays tend to have high plasma cholesterol (8.4 ± 2.6 mmol/l) relative to other avian species. Surprisingly, diet has not yet been firmly implicated as a risk factor. There is an association of atherosclerosis plaques and chlamydia in humans, but this has not been demonstrated in parrots. Susceptible P-line chickens infected with Marek's disease (a herpesvirus) develop atherosclerosis lesions.

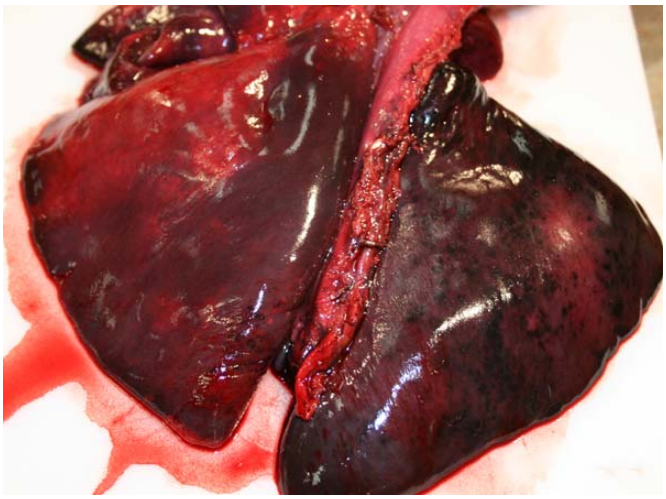
Clinical signs in parrots are generally non-specific. Sudden death (as in Dr. Woods' accession) is the most common manifestation. Signs attributed to atherosclerotic disease in parrots include lethargy, weight loss, and neurological, and circulatory and respiratory signs. Lesions occur in any small to medium arteries, and tend to affect vessels of the neck and thoracic aorta.

If you are presented with a parrot that dies suddenly, one of the rule outs should be atherosclerosis.

Bavelaar FJ, Beynen AC: 2004, Atherosclerosis in parrots. A review. Vet Q. 26(2):50-60.
Bavelaar FJ, Beynen AC: 2003, Severity of atherosclerosis in parrots in relation to α-linolenic acid. Avian Dis 47: 566 - 577.

Even big kitties get the plague

An adult mountain lion that was part of a wildlife study was found dead in the Jackson area of Wyoming. It was assumed to have been killed by wolves. Remains of the dam and her kitten were submitted to the WSVL. Dr. Cynthia Tate of the Wyoming Game and Fish Department performed the post-mortem examination. She noted congested lungs in the dam, and swollen necrotic submandibular lymph nodes in the kitten.



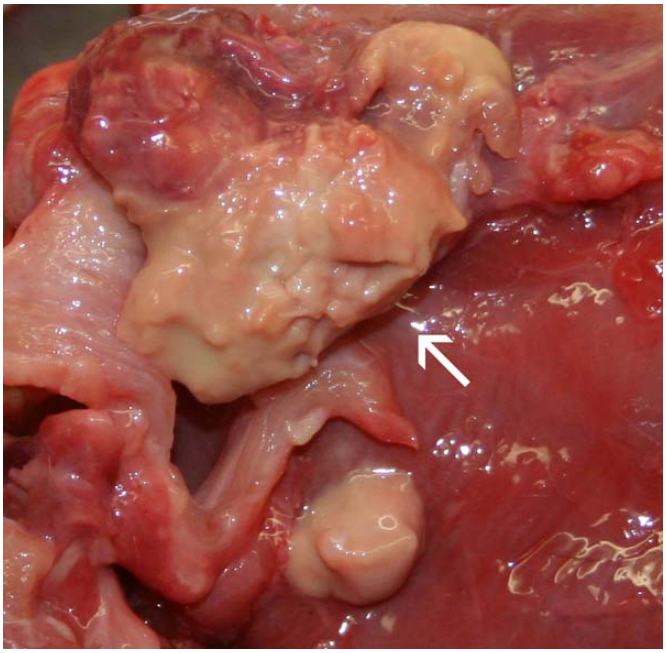
WSVL 06W15340: lungs from an adult mountain lion with plague. Although somewhat autolytic, there are areas of pulmonary hemorrhage. Histologically there was bacterial pneumonia

A direct fluorescent antibody exam was positive for *Yersinia pestis*. Subsequent cultures were positive for the organism. Histopathology confirmed the presence of lesions consistent with plague in various organs.

Since the submandibular lymph node was involved, exposure might have taken place by consuming infected rodents. Remember that, although plague can be spread to felines and other susceptible hosts by fleas, it can also be transmitted by direct contact with infected animals. Three mountain lions

have been diagnosed with plague in Wyoming in the recent past, these two and another animal that was part of the same biological survey. This indicates that plague is active in that area and that other felines may become infected. Dogs are occasionally infected but generally do not develop disease. Serological surveys on wild canines are often used to gauge the incidence of plague in an area since they are exposed, may become infected, yet survive and develop an antibody titer.

Information on plague in cats (small domestic kind) can be found in the WSVL 2005 disease updates. http://wyovet.uwyo.edu/Diseases_2005.asp



06W15341: Enlarged lymph nodes (arrowhead) from a mountain lion kitten. Histologically there was necrotizing lymphadenitis with intralesional *Y. pestis.*

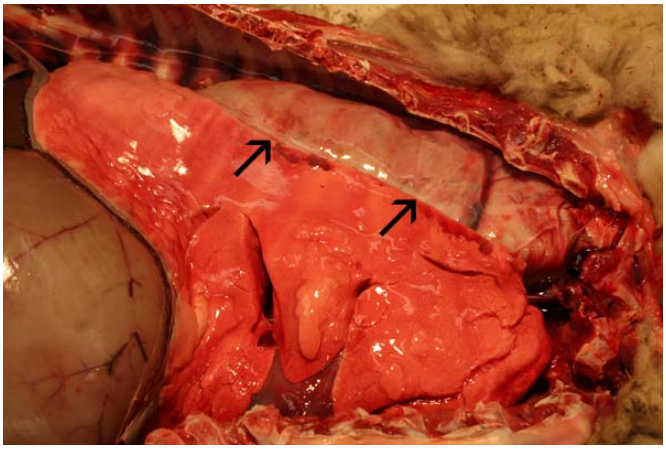
Dr. Ken Mills/Dr. Cynthia Tate

Megaesophagus in a sheep

Dr. Cornish was presented with an interesting case. A producer of black-face sheep noticed that a ewe was losing weight and "vomiting." Vomiting in ruminants is unusual. We sometimes see it in animals with rumenitis, but in such situations it is generally acute and terminal.

The 42 kg Hampshire ewe that Dr. Cornish was presented with had severe megesophagus and inhalation pneumonia.

The histological changes seen by Dr. Cornish were consistent with dysautonomia. These included neuronal degeneration and necrosis in celiacomesenteric and stellate ganglia, and in myenteric and submucosal plexuses of the abomasum, small intestine, and spiral colon. The ewe also had terminal inhalation pneumonia.



WSVL 06O16445. Marked megaesophagus (arrows) in opened thorax of a 2.5-year old Hampshire ewe.

WSVL 06O16445. Removed pluck showing megaesophagus. There is abundant feed in the opened esophagus.

These changes were suggestive of abomasal emptying defect (AED) of Suffolk and Hampshire sheep. The presence of megaesophagus is unusual however.

Some years ago, Dr. Milt McAllister and colleagues wrote an article on this disease based on cases they had seen from Wyoming and Colorado. One of the conclusions was that it is unlikely that AED is a simple homozygous recessive disorder. An untested hypothesis is that AED is a neurotoxicosis.

Donal O'Toole

Pruden SJ, McAllister MM, Schultheiss PC, O'Toole D, Christensen DE: 2004, Abomasal emptying defect of sheep may be an acquired form of dysautonomia. Vet Pathol. 41(2):164-169.

Johne's disease in a captive elk

Dr. Woods was presented with a captive yearling elk with a history of weight loss and diarrhea. Grossly, a portion of the jejunum and much of the ileum was moderately thickened. Ileocecocolic lymph nodes were large. Changes were suggestive of Johne's disease.

PCR examination in Dr. Mills laboratory confirmed the presence of *Mycobacterium avium* subsp. *paratuberculosis* (MAP). Histologically Dr. Woods diagnosed granulomatous enteritis typical of Johne's disease, with intra-lesional acid fast bacteria. Attempted culture of the organism is ongoing, but the diagnosis is Johne's disease.

At the suggestion of the United States Animal Health Association, the USDA has now established a cooperative state-federal-industry program to control Johne's disease in cattle. The goal is to educate producers about the disease, which is primarily found in dairy herds.

But John's also occurs in beef cattle - including cow-calf herds in Wyoming - as well as in captive elk, as this case indicates. At the 2006 winter meeting of the WVMA, Dr. Fred Emrich gave a presentation on the disease, and efforts in Wyoming to enroll producers in the voluntary bovine Johne's disease control program. Its long term goal is to improve management practices that minimize the impact of Johne's in herds, and to better identify and segregate test-positive and test-negative herds.

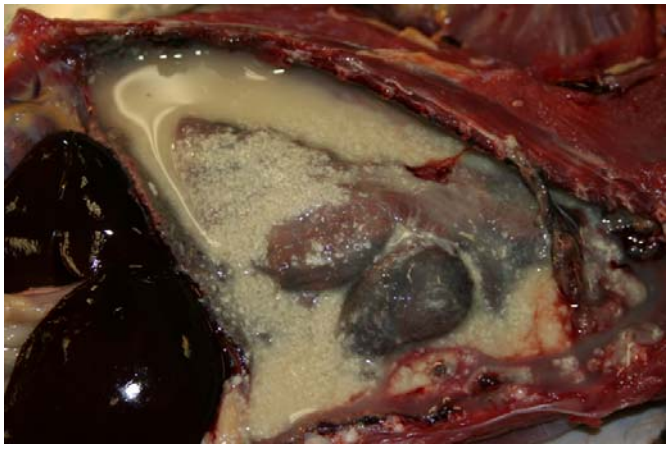
Dr. Donal O'Toole/Dr. Leslie Woods

USDA APHIS: 2005 United States Animal Health Report, p. 51.

Nocardia sp. pleuritis in a cat

Owners of a 3-year old castrated cat came home to find it breathing hard, unable to walk, and crying. It was presented to a Wyoming veterinarian as an emergency call. The clinician's tentative diagnosis was poisoning, or peracute viral disease.

Dr. Cornish was presented with a thin carcass. Pleural sacs contained ~250 ml viscous yellow purulent exudate containing "sulfur granules" (photo). Fibrin covered pleural surfaces. Lungs were atelectatic.



06F13954: Opened thorax of a cat with *Nocardia asteroides* pyothorax and pleuritis. There are distinct "sulfur granules" in the exudate.

Nocardia asteroides was grown from the pleural exudate. Histology corroborated the bacteriological findings since filamentous bacteria and associated Splendore-Hoeppli material were present. Infection was confined to the thorax. Virological findings were negative. The diagnosis was nocardial pleuritis.

Pleuritis due to *Nocardia* spp. is not exactly common - since 2000 we had one other feline case. But clinical signs and lesions in this cat were fairly typical of pulmonary nocardiosis in dogs and cats. *Nocardia* spp are common in soil, where they degrade organic matter. They are considered opportunistic organisms, and are spread by wounds, bites or inhalation. In cats, bites and scratches are involved. Treatment can be successful, but it necessitates draining the pleural fluid and prolonged administration (1 - 3 months) of sulfonamides.

Dr. Donal O'Toole

Malik R et al: 2006, Nocardia infections in cats: a retrospective multi-institutional study of 17 cases. Aust Vet J. 84(7):235-45

Notes from Bacteriology laboratory

1. Ringworm exams

WSVL has two tests to identify ringworm. The direct exam is quick but not as sensitive as a DTM culture. We have received a number of samples that were negative by the direct exam but there was insufficient sample remaining to set up a DTM.

Please send in as much sample as possible if you want the DTM culture performed.

2. Diarrhea in calves

We run an *E. coli* PCR on all diarrhea samples from calves two weeks of age or less as part of our standard diarrhea work up.

If you suspect *Clostridium perfringens* enteritis, you need to SPECIFICALLY request the PCR.

3. Johne's PCR test

The USDA has been getting increasingly interested in Johne's disease, especially in dairy states. Our state veterinarian's office now reports total number of animals tested and positive animals to the USDA, based in part on numbers generated by the WSVL.

Although less common in Wyoming, we see Johne's infected beef cattle from time to time, as well as in wildlife species (above)

The bacteriology laboratory now offers a PCR test for *Mycobacterium avium* subspecies *paratuberculosis*. Its main advantage is speed. It allows us to get away from culturing,

which typically is slow (up to 16 weeks), due to the growth characteristics of the organism.

The test was validated using archived fecal samples from 221 cows that were positive (n = 121) or negative on culture (n = 100). There were no false positives. The detection rate in the positives was 98.3% when feces had an average tube count of 3.0 cfu or more. The detection rate was lower when there were less than 3.0 cfu/tube: 73.7%.

Sample: 2 gm or more of feces
Turn-around: 1 week
Cost: $35/sample

Dr. Ken Mills

Foreign Animal Disease Diagnostic Training

Drs. Ana Bratanich (WSVL virologist) and Cynthia Tate (WGFD wildlife veterinarian based at WSVL) recently attended an intensive one-week Foreign Animal Disease Pathologist Course at Plum Island Animal Disease Center (PIADC).

PIADC is a USDA/DHS facility specializing in research and diagnosis of foreign animal diseases that represent a potential threat to the US economy. The center is on an 880 acre island off the northeastern tip of Long Island, New York. It was originally authorized and funded in response to foot-and-mouth disease (FMD) outbreaks in Mexico (1946) and Canada (1952), as an offshore location to study this disease. Designed for veterinary diagnosticians, the recent course was attended by 10 USDA employees, 10 state agency employees, and two international guests.

Diseases demonstrated were FMD, contagious bovine pleuropneumonia, rinderpest, sheep pox, heartwater, African horse sickness, African swine fever, classical swine fever, rabbit hemorrhagic disease, exotic Newcastle disease, and highly pathogenic avian influenza.

“We had clinical rounds and necropsies every afternoon. This was an invaluable opportunity to examine animals in subclinical, clinical, and postmortem stages of these foreign diseases,” said Dr. Bratanich. “There is a new awareness of these diseases as potential agroterrorism agents. Some of the diseases have the potential to infect North American wildlife as well as livestock. "

"Once a disease like this gets into wildlife populations there is a whole new set of challenges in achieving eradication,” added Dr. Tate. “Hopefully, we will not see these diseases in the US, but should they arrive on our shores, this training should enable diagnosticians like us to detect these diseases early in the effort to minimize their economic impact”.

Dr. Cynthia Tate/Dr. Nicky Bratanich

BVDV in New World Camelids

Last year Dr. Carman and her colleagues published a paper in the *Journal of Veterinary Diagnostic Investigation* about BVDV in alpaca crias. This triggered concern among alpaca owners. Our laboratory tested substantial numbers of alpacas for BVDV. We have yet to find a positive cria.

At the annual meeting of the AAVLD, Dr. Ed Dubovi presented information on the disease. The Cornell laboratory has probably looked at more animals than anyone else. His conclusions about the disease were:

1. It is rare (total of 18 acutely or persistently infected animals identified). He did not think that, in a herd without a disease problem suggestive of BVD, it was necessary to either vaccinate or to screen the entire herd for this disease.

2. Clinical signs in persistently infected crias are abortion and perinatal death; ill-thrift in young stock; diarrhea; abnormal hair coat.

3. All isolates to date type out as BVDV 1b. Since some of the affected premises were linked through the sale of animals, it is possible that cases seen in various states represent a single extended outbreak.

4. As Dr. Cornish showed occurs in cattle, crias can be infected for extended periods (up to 2 months) before clearing infection.

5. BVDV can occur in NW camelids without contact with infected cattle. Camelids appear to be able to sustain infection without the involvement of infected cattle.

Carman et.al: 2005, , J Vet Diagn Invest.;17(6):589-53
Bedenice D et al: 2006, Bovine viral diarrhea virus in alpacas from North America. Proceedings, 49th Annual Meeting, American Association of Veterinary Laboratory Diagnosticians, Minneapolis, MN. P. 30.

CYTOLOGY OR CONVENTIONAL HISTOPATHOLOGY?

IF IN DOUBT, SEND THE FIXED MASS.

OK, some of you just love cytology, and on your continuing education odyssey you’ve been advised by a clinical pathologist to ALWAYS do cytology.

But frankly, from this laboratory’s standpoint:

- When we get formalin fixed samples, we see architecture, degree of necrosis, regional variation in cellular phenotype, inflammatory response, and vascular invasion if present – all of which go into assessing the general bad-assedness of a tumor. Can’t do this with cytology.
- We can grade malignant neoplasms based on formalin fixed samples, if a validated grading system is in place. Can’t do this with most cytology samples.

- We can give you tumor margins (got it all; not got it all) with formalin fixed masses. Can't do this with cytology.
- A high proportion of cytology samples submitted to this laboratory are non-diagnostic, due to inadequate cells, smear artifact, or staining problems.
- All three pathologists here are board certified in anatomical pathology, not clinical pathology. Although we can do cytology when we have to, WE HAVE A STRONG PREFERENCE FOR FORMALIN FIXED SAMPLES.
- It often takes longer to read cytology samples than formalin fixed masses.
- When we get formalin fixed samples, we can do confirmatory immunohistochemistry as required (e.g., for T and B cells in the case of lymphoma, or Melan A for suspect melanomas). We don't do this on cytology since we have not calibrated IHC for the lighter fixation of cytology preparations.

Last but not least, cytology is comparable in cost to conventional histology, at least for small masses ($30 each). Even when a mass is large and we must take multiple levels in various planes to look at margins, cost per tumor is capped at $35.

You will provide your clients with a better product and most accurate report if, when you have a tumor for identification, you give it to us fixed rather than as cytology preparation.

Dr. Donal O'Toole

Copper deficiency in Wyoming

In the last several months we've seen cases of what look like serious copper deficiency, based on liver analysis. They highlight 3 trends we've been seeing for several years.

First, while copper deficiency is certainly not news in most of Wyoming, it has apparently become sufficiently "old hat" that producers are no longer thinking about it. In at least one case, animals were receiving no supplementation whatsoever, despite a long history of Cu deficiency on neighboring ranches. In another, the story goes that the producer was "supplementing", but couldn't tell me with what or how much. In a third, the producer had a nutritionist put together a mineral program after seeing low Cu in biopsies last year, and has seen some improvement in production, but still has a lot of cattle with very low hepatic Cu.

The second trend is cowboy necropsies. On at least two occasions in the last month, we've run small pieces of mystery meat that were submitted to the lab as liver, only to discover that they were another tissue.

The third trend is to find Cu deficiency by accident after extensive path and microbiological workups had already identified a number of other etiologies.

It shouldn't be news to most readers of this newsletter that fairly substantial portions of Wyoming produce forages that contain insufficient Cu for cattle. For a while in the early - mid '90's it seemed like Cu deficiency was the only diagnosis we made. But, as more and more producers adopted better supplementation, the number of cases fell off. Recently, we've been seeing more animals with very low tissue Cu concentrations. In some cases the owner is new to livestock production and doesn't know about trace element nutrition. More often, however, they are "supplementing" by scattering whichever trace mineral salt mix was cheapest around the pasture... whenever they remember to do so. Unfortunately, on the saline pastures typical of a lot of this state, salt consumption can be pretty erratic. Cattle's preferences can change from year to year, resulting in insufficient consumption. No supplement is worth a damn if animals don't eat it. Any worthwhile supplementation program has to include some provision for monitoring consumption and performance, and making adjustments if it fails.

The trend to do-it-yourself post mortems is especially frustrating. Totally aside from collecting the right tissue, we rely on the referring veterinarian to give us a useful description of any visible lesions, include some sort of comprehensible history and to see to it that the sample is handled and packaged without extraneous contamination. The latter is especially important with small samples like biopsies. A fly speck worth of dirt can change trace mineral concentrations 100-fold. We recently received a liver sample that was covered with multicolored granules. Seems the only container the cowboy had handy was a bucket he'd been using to haul trace mineral, so that's what he stored the liver in. Needless to say, the liver Cu concentration set a new lab record.

Copper deficiency (the disease) and the metabolism of Cu in general are frustratingly multifactorial. Uptake, distribution and elimination of the element are influenced by other dietary components and, to a lesser extent, a variety of environmental factors and even genetics. Copper deficiency rarely causes overt clinical disease under field conditions, but rather is manifested as exacerbation of other disease conditions. For these reasons it is possible to have Cu deficiency in a herd despite nominal supplementation. Conversely, it is also possible to get by with really sub-standard nutrition for years at a time and no apparent disease problems.

Given the foregoing, it should be obvious that the only reliable way to diagnose Cu deficiency is by analyzing the cow. Copper-dependent enzymes such as ceruloplasmin and superoxide dismutase (SOD) have the theoretical advantage of representing biologically available Cu, but have relatively short half-lives and aren't commonly available in most labs. Serum Cu is easy to obtain and can be readily combined into a "screen" with other trace elements such as Mo. It is definitive proof of deficiency when it is depressed.

Unfortunately, serum Cu does not become significantly depressed until other body stores have been depleted. It is relatively labile and long-standing problems may not show

up in serum if the diet has recently improved. Liver biopsies are commonly touted as the "gold standard" for determining nutritional Cu status, but collection requires a moderately invasive procedure and accurate results require careful attention to cleanliness. After a normal surgical preparation, the biopsy site should be thoroughly rinsed with copious amounts of deionized water to eliminate any residual soap or disinfectant. Disinfectants do not have to contain Cu per se to interfere with the analysis, therefore the common practice of soaking surgical instruments in a disinfectant between animals should be avoided. Absolute ethanol is an exception to the forgoing rule. Biopsy specimens should be placed in chemically clean tubes for shipment. We've had pretty good luck with conventional red top vacutainers, but ideally one should use trace element grade tubes, especially if other metals are to be run. It is also possible to rinse conventional tubes of one sort or another with dilute (5% in DI water) high purity nitric acid. Once the diagnosis has been made and remedial action is taken, its really a good idea to follow up in a year or so with production records and/or further analysis. Not all herds respond to supplemental Cu the same way and it may be necessary to go back to the drawing board 2-3 times to finally get optimum performance.

Dr. M. Raisbeck, R. Siemion

2006 WSVL/DEPT VET SCI PUBLICATIONS

Dr. Merl Raisbeck

1. Raisbeck MF, Dailey RN: 2006, Organochlorine pesticides. In: *Small Animal Toxicology* (ed. Petersen and Talcott). WB Saunders.
2. Raisbeck MF: 2006, Petroleum hydrocarbons in small animal toxicology (ed. Petersen and Talcott). Pub. W. B. Saunders.
3. Raisbeck MF: 2006, Petroleum hydrocarbons 5 minute veterinary consult. ed. Tilley and Smith. Pub. Lippincott
4. Allen JG, Colegate SM, Mitchell AA, M. F. Raisbeck MF 2005. Bioactivity-guided isolation and structural identification of toxic cucurbitacin steroidal glucosides from *Stemodia kingii*. Phytotoxin Analysis - accepted
5. Cook WE, Cornish TE, Williams EW, Brown B, Hiatt G, Kreeger TJ, Raisbeck MR 2006: *Xanthoparmelia chlorochroa* intoxication in wapiti (*Cervus canadensis*). CAB Int'l - accepted
6. Cook WE, Raisbeck MR, Cornish TE, Williams ES, Brown B, Hiatt G, Kreeger TJ: 2005. Paresis and death in elk (*Cervus elaphus*) due to lichen intoxication in Wyoming Journal of Wildlife Diseases - accepted.
7. Raisbeck MF, Siemion RS, Smith MA: 2006,. Modest copper supplementation blocks molybdenosis in cattle. J Vet Diagn Invest.18(6):566-72.
8. Dailey RN, Cornish TE, Siemion RS Raisbeck MF: 2005, Trace elements in sage grouse. J Wildlife Dis - submitted
9. Raisbeck MF, Allen JG, Colegate SM, Mitchell AA: 2006, Stemodia kingii intoxication in the mouse. In review Australian Vet Journal
10. M. F. Raisbeck, M. A. Smith and P. Talcott: Grazing reclaimed minelands in SE Idaho. Environmental Issues in Phosphate Mining, hosted by Idaho DEQ, Pocatello, ID, 4/13/06.
11. M. F. Raisbeck, R. N. Dailey, D. L. Montgomery, R. S. Siemion, M. Vasquez, J. T. Ingram (2006): Two Years Later: What killed the Red Rim Elk? pp. 1-6, Reflections, UW AES, Laramie, WY.

Dr. Don Montgomery

12. Jensen TK, Montgomery DL, et al: Application of fluorescent in situ hybridization targeting 16S ribosomal RNA and immunohistochemistry for demonstration of *Coxiella burnetii* in placentas from ruminant abortions. Acta Pathol Microbiol Immunol Scand [APMIS], November 2006 - accepted
13. Sangster CR, Stevenson CK, Kidney BA, Montgomery DL, Allen AL: Kernicterus in an adult dog. Vet Pathol, November 2006 - accepted
14. Montgomery DL: Distribution and cellular heterogeneity of bovine viral diarrhea viral antigen in the brain of persistently infected calves - a new perspective. Vet Pathol September 2006 - in review
15. O'Toole D, Taus NS, Montgomery D, Oaks JL, Li H: 2006. Intra-nasal inoculation of American bison (*Bison bison*) with OvHV-2 reliably reproduces malignant catarrhal fever Vet Pathol Nov 2006 - submitted
16. Chimalakonda AP, Montgomery DL, Nguyen JH, Lemasters JJ, Kobayashi E, Mehvar R: Attenuation of acute rejection in a rat liver transplantation model by a liver-targeted dextran prodrug of methylprednisolone. Transplantation 81:678-685, 2006
17. Baszler TV, Kiupel M, Williams ES, Gidlewski T, Montgomery DL, O'Rourke KI, Hall M: Comparison of two automated immunohistochemical procedures for the diagnosis of scrapie in domestic sheep and chronic wasting disease in North American White-tailed deer (Odocoileus virginianus) and mule deer (Odocoileus hemionus). J Vet Diagn Invest 18:147-155, 2006
18. Kreeger TJ, Montgomery DL, Jewell JE, Schultz W, Williams ES: Oral transmission of chronic wasting disease in captive Shira's moose. J Wildlife Dis 42:640-645, 2006

Dr. Todd Cornish

19. Cook WE, Cornish TE, Williams EW, Brown B, Hiatt G, Kreeger TJ, Raisbeck MR 2006: *Xanthoparmelia chlorochroa* intoxication in wapiti (*Cervus canadensis*). CAB Int'l - accepted
20. Cornish TE: 2006. Wildlife Diseases (book title). Diseases of Wild Canids (chapter title). Jane Huffman (ed.). Pennsylvania State University Press - in review.
21. Cook WE, Raisbeck MR, Cornish TE, Williams ES, Brown B, Hiatt G, Kreeger TJ: 2005. Paresis and death in elk (*Cervus elaphus*) due to lichen intoxication in Wyoming Journal of Wildlife Diseases - accepted.
22. Naugle D, Walker B, Aldridge C, Cornish TE, Cook W, Moynahan B, Halloran M, Brown K, Johnson G, Schmidtman E, Mayer R, Kato C, Matchett WR, Christianson T, Rinkes T, Creekmore T: 2005 West Nile virus heads West: survival of sage grouse declines Emerging Infectious Diseases - in review
23. Cornish TE, Cook WE, Creekmore T, Williams EW, Walker B, Aldridge C, Christianson T, Naugle D 2005**.** Fatal West Nile virus infection in greater sage grouse (*Centrocercus urophasianus*) in Montana and Wyoming Journal of Wildlife Diseases - in review
24. Dailey RN, Cornish TE, Siemion RS Raisbeck MF: 2005, Trace elements in sage grouse. J Wildlife Dis - submitted

Ms. Katie Bardsley

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Dr. Donal O'Toole

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