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 atherosclerosis**

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.

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 atheroscleromatous
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
atheroscleromatous 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.

26(2):50-60.
Bavelaar FJ, Beynen AC: 2003, Severity of atherosclerosis in parrots in

**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.

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

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.

These changes were suggestive of abomasal emptying defect (AED) of Suffolk and Hampshire sheep. The presence of megesophagus 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


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. Ileoceccolic 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 Johne'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

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**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.

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Dr. Donal O'Toole

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**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.


### 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.
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 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 ‘90s 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...
Dr. Merl Raisbeck


Dr. Don Montgomery


Dr. Todd Cornish


Ms. Katie Bardsley


Dr. Gerry Andrews


Dr. Ken Mills


37. Willford J, Mills K, Goodridge LD: Evaluation of Three Commercially Available ELISA kits for Detection of Shiga Toxigenic Escherichia coli- Food Microbiology and Safety-in revision

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