

# Colorado State University Veterinary Diagnostic Laboratories



---

Volume 2, Number 1

Fall 1997

**O Letter from the Director**

Here is issue #3 of LabLines! Inside, you'll find informative articles on *Clostridial perfringens* genotyping, Malignant Catarrhal Fever, antimicrobial susceptibility testing, assessing Vitamin A and E status in food animals, blue green algae poisoning, soft tissue sarcomas in dogs and cats, and polioencephalomalacia. Additional stories include *Nematodirus* in calves, sunburn, agalactia and salt poisoning in swine, and mysterious encephalitis in captive elk.

In the last few months, you may have noted a new name on your pathology reports -- Gary Mason. Dr. Mason, a Colorado native, is our new pathologist. He received his DVM from Texas A&M in 1988, and completed his pathology training at the University of Tennessee in 1997. His specialties include cattle pathology, dermatopathology, and a recent interest in fish pathology! We are very pleased to have him join us. In the office area, you may have spoken to our new phone receptionist, Jennifer Swenson. We welcome Nancy Ault back to the office after a summer of maternity leave. We also have two new word processors, Esta Moutoux and Tracie Trimarco, to get your reports to you faster.

Additions to laboratory services include:

- Preliminary fax results for biopsies allowing 24-48 hour turnaround time.
- New atomic absorption spectrophotometer allowing new tests in the chemistry/toxicology section.
- New antimicrobial susceptibility testing system.
- On-going test development in the microbiology section.

This summer, I participated in the CVMA tour with Jim Noone (CVMA President) and Joe Lory (CVMA Executive Director). It was truly pleasurable to travel through Colorado and meet many of you in person, after years of seeing your names on laboratory requests or visiting with you by phone. I am grateful to the CVMA for giving me this opportunity to visit with you.



*Barbara E. Powers*

Barb

**Powers, DVM/PhD**

Colorado State University  
Diagnostic Laboratories  
300 West Drake  
Fort Collins, CO 80523  
Phone 970/491-1281  
Fax 970/491-0320  
email: [jkammerz@vth1.vth.colostate.edu](mailto:jkammerz@vth1.vth.colostate.edu)  
<http://www.vetmed.colostate.edu/dlab>

Arkansas Valley  
Animal Disease Diagnostic Laboratory  
27847 Road 21/Rocky Ford, CO 81067  
Phone 719/254-6382 Fax 719/254-6055  
Western Slope  
Animal Diagnostic Laboratory  
425-29 Road/Grand Junction, CO 81501  
Phone 970/243-0673 Fax 970/242-0003

---

**CLOSTRIDIUM PERFRINGENS GENOTYPING**

Most domestic animals carry some *Clostridium perfringens* bacteria in their intestines. There are five genotypes of *C. perfringens* recognized -- types A, B, C, D, and E. These produce various damaging exotoxins and a conclusion that a particular isolate is the cause of a disease process may be misleading. Dr. Bob Ellis has adapted polymerase chain reaction (PCR)-based genotyping (developed by Dr. Glenn Songer at the University of Arizona) to identify *C. perfringens* genotypes to improve our understanding of what the presence of different genotypes in the intestines or feces may mean. Three or four isolated colonies are picked from the initial isolation plate and subjected to PCR. A survey during the first half of 1997 produced the following isolates:

Species	Total Typed	Genotypes
Bovine	18	15-A*, 2-C 1-A&E**
Equine	23	14-A, 3-C, 2-A&C 1-A&A+entero 1-A&C+entero
Canine	1	1-A&A+entero
Porcine	<u>1</u>	1-A&C
	43	

\*Capital letters indicate type.

\*\*Mixed genotypes.

These results agree with others in finding type A to be the most common genotype in most species. For instance, in a recent survey of piglets with diarrhea, we found *C. perfringens* type A in 64 of 77 pigs sampled regardless of clinical disease. Therefore, interpretation can be difficult even though the literature associates type A with neonatal enteritis in pigs and neonatal abomasitis in calves. Genotyping results of *C. perfringens* isolates from foals submitted to the Large Animal Clinic indicate at this time that foals with type A enteritis respond to treatment better than foals with type C enteritis. Another recent discovery is that several calves have yielded type E + enterotoxin isolates which have not been previously recorded. Note that some isolates are positive for the gene controlling enterotoxin production. This may have clinical significance in some animals.

Presumptive diagnoses are often based on isolation of numerous *C. perfringens*, observation of numerous organisms associated with lesions on histopathology, and clinical disease that COULD be related. Genotyping of *C. perfringens* isolates will increase the accuracy of diagnoses, assist in determining treatment in some instances, and will be used to improve current vaccines that are available for prophylaxis of *C. perfringens* enteritis.

C. perfringens genotyping: Submit fresh intestine or feces. Fee--\$25 (plus \$30 for aerobic/anaerobic culture).

---

**RECENT CHANGES IN ANTIMICROBIAL SUSCEPTIBILITY TESTING**

---

We improved our antimicrobial susceptibility test procedures by adopting the disk diffusion method. This gives us the capability to review and summarize trends, and increases flexibility in the selection of drugs tested.

We report results as:

S=Susceptible, suggesting that the infection may be appropriately treated with the standard systemic dosage of antimicrobial recommended for that species, unless otherwise contraindicated.

I=Intermediate, indicating antimicrobial susceptibility that approaches usually attainable blood and tissue levels, but response rates may be lower than for susceptible isolates. This implies clinical applicability in body sites in which the drugs are physiologically concentrated (eg, quinolones and beta-lactams in urine) or when a high dose of drug can be used safely.

R=Resistant, indicating that the infection is not inhibited by the usually achievable systemic concentrations of the antimicrobial and/or its susceptibility may fall within the range where microbial resistance mechanisms are likely.

We select the most appropriate agents to test that have proven clinical efficacy for treatment of systemic infections and are commonly used in the animal species from which the isolate was obtained. Agents (eg, neomycin, polymyxin, bacitracin, nitrofurazones) used for purposes other than the treatment of systemic disease (eg, prophylaxis, enteric diseases, topical treatment) are not tested because the validity of susceptibility tests for these uses has not been established.

Given the limited number of antimicrobial agents approved for use in some animal species, extra-label use is commonly practiced and we include some of these agents in our tests. When agents (e.g., chloramphenicol and fluoroquinolones in food animals) have been specifically prohibited from extra-label use, they are not tested.

We include one representative of each group of related drugs in the test panel. The following guidelines identify these groupings and aid in interpretation of antimicrobial susceptibility test results.

**Penicillin**--Represents penicillin G with a spectrum of activity directed primarily against gram-positive and some fastidious, gram-negative bacteria.

**Oxacillin**--Tests for susceptibility to methicillin, nafcillin, cloxacillin and related agents that are used specifically to treat penicillinase-producing staphylococci.

**Ampicillin**--Tests for susceptibility to amoxicillin and hetacillin which have activity against more gram-negative bacteria than penicillin, but are susceptible to beta-lactamase hydrolysis.

**Amoxicillin/clavulanic acid**--Tests the combination of

antimicrobial and beta-lactamase inhibitor.

**Ticarcillin** --Tests for susceptibility to carbenicillin, which have an expanded spectrum of activity against gram-negative bacteria, including many *Pseudomonas* spp.

**Cephalothin**--Tests susceptibility to first-generation cephalosporins, such as cephapirin and cefadroxil.

**Cefoxitin**--A second-generation cephalosporin.

**Ceftiofur**--Susceptibility results are not predictive for other cephalosporins.

**Tetracycline**--The class representative for chlortetracycline, oxytetracycline, minocycline, and doxycycline.

**Aminoglycosides**--Cross-resistance for the aminoglycosides is not expected so each drug in this class must be tested individually. Results are not reported for use in food animals as recommended by the AABP. Currently, we test susceptibility to **amikacin** and **gentamicin**.

**Erythromycin, tilmicosin** and **tiamulin**--Macrolides tested individually.

**Enrofloxacin**--The only quinolone currently tested. Results may not be predictive for other quinolones.

**Trimethoprim/sulfamethoxazole**--To represent the potentiated sulfonamides, including trimethoprim/sulfadiazine and ormetoprim/sulfadimethoxine.

**Triple Sulfa**--A representative of all non-potentiated sulfonamides.

**Pirlimycin** and **penicillin/novobiocin**--Tested as infusion products for treatment of bovine mastitis during lactation.

**Rifampin, florphenicol, chloramphenicol** and **spectinomycin**--Single class drugs that may be included on test reports.

Some drug and isolate combinations are not tested because these combinations have well-established predictable outcomes:

- **Clindamycin** is active against gram-positive and anaerobic bacteria, but not commonly gram-negative.
- Anaerobes are not tested for susceptibility as they are predictably susceptible to clindamycin, tetracyclines, most beta-lactams and chloramphenicol, except the *Bacteroides fragilis* group which is frequently resistant to beta-lactam drugs. Aminoglycosides and fluoroquinolones are not effective for the treatment of anaerobic infections. Potentiated sulfonamides provide variable clinical results with anaerobes that do not correlate with in vitro susceptibility testing.

Final selection of the agent to use in treating an animal must include many considerations in addition to susceptibility, such as microbiological, clinical, and pharmacological factors (distribution, safety, residue avoidance), as well as clinical indications, efficacy, label-approved indications for use and veterinarian-client-patient relationships.

In future issues, we will have gathered enough data to provide you with sensitivity patterns of certain bacteria.

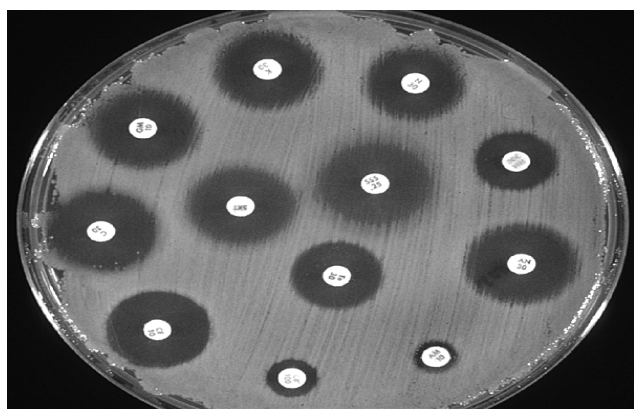
Antimicrobial susceptibility: After isolation of microbial agent. Fee--\$8.50.

---

## SOFT TISSUE SARCOMAS IN DOGS AND CATS

Barb Powers

What exactly do we mean when we make a diagnosis of soft tissue sarcoma? This includes a group of sarcomas including fibrosarcoma, peripheral nerve sheath tumors (schwannoma or neurofibrosarcoma), "hemangiopericytoma," malignant fibrous histiocytoma, myxosarcoma, and liposarcoma. With the possible exception of liposarcoma, the exact tumor type is less important than the grade of tumor. In fact, at the light microscope level, it is difficult to distinguish between schwannoma and neurofibrosarcoma, and hemangiopericytomas are likely a form of peripheral nerve tumor. The grading system we use is adopted from human medicine and comprises three grades -- 1



Ag  
a r  
p l  
t e  
d e  
m

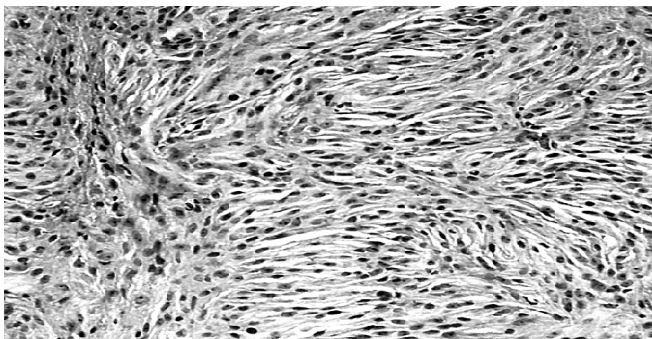
onstrating growth inhibition of a bacterial isolate by several antimicrobial agents

being the least malignant and 3 being the most malignant. Grade is based on overall tumor differentiation, mitotic rate, and amount of tumor necrosis. In a recent study of dogs with soft tissue sarcomas treated by aggressive surgery alone, grade was predictive of survival as the 3-year survival rate was about 80% for grade 1, 50% for grade 2, and 20% for dogs with grade 3 tumors (JAVMA 211; 1997, p. 1147). Grade also was predictive of tumor recurrence in soft tissue sarcomas treated with surgery and adjuvant radiation therapy.

These soft tissue sarcomas present as masses in the subcutaneous regions of the trunk, or more commonly, the extremities. At surgery, these may seem to “peel out,” however, this type of conservative surgery invariably results in tumor cells being left behind. The evaluation of margins for completeness of removal is a significant predictor for local recurrence; less than 3% of dogs with “clean” margins have tumor recurrence, while at least 30% of dogs with “dirty” margins have tumor recurrence.

Photomicrograph of a soft tissue sarcoma of peripheral nerve origin, grade 1

In cats, we have recently seen an increase in the apparent “vaccine-



associated” fibrosarcomas; locally invasive sarcomas developing between the shoulder blades or in the thigh. These sarcomas are usually grade 2 or 3, and other types of sarcomas including malignant fibrous histiocytoma and osteosarcoma can occur. These tumors have a high propensity for recurrence despite aggressive treatment. Even when we report “clean” margins, a high percent still recur.

If you have questions regarding treatment options, the Comparative Oncology Unit at Colorado State University is available for consultation. In addition, there is a funded protocol involving radiation and hyperthermia for canine soft tissue sarcoma patients in our region. Call 970-221-4535 for further information regarding treatment.

Tumor Diagnostics: Submit tissue sample in 10% formalin. Fee--\$21; plus \$1.50 prepaid mailer, \$6 for FedEx courier, OR \$5 for local courier.

---

#### **NEW AND IMPROVED TOXICOLOGY/CHEMISTRY TESTING**

**Dwayne Hamar and Cathy Bedwell**

We recently purchased a Graphite Furnace Atomic Absorption Spectrophotometer (GFAA). Graphite furnace analyses require less sample, detect lower amounts of metals, and provide more reproducible results at low concentrations of metals.

We have developed the method for blood lead analysis by GFAA and now use this for all blood lead analyses. With the GFAA, we can analyze feline and avian blood samples previously referred to another laboratory. A minimum of 0.1 ml of whole blood is needed. Whole blood, not serum or plasma, should be submitted since most of the circulating lead is associated with RBC.

We developed a method for molybdenum analysis by GFAA for all sample types. In the past, we have analyzed liver and feed samples for molybdenum using flame atomic absorption. We also have started using GFAA for serum and/or blood molybdenum whose concentrations were below the detection limit of the flame atomic absorption method.

Additionally, we are currently developing GFAA methods for mineral analyses that have been requested in the past. These include cobalt and manganese analyses on serum, blood, liver, and forage.

---

Lead levels: Submit tissues or whole blood. Fee--\$6 (tissue); \$15 (blood). Molybdenum: Submit liver, serum, blood or feed. Fee=\$8

---

#### **MALIGNANT CATARRHAL FEVER IN BISON AND CATTLE**

**Pat Schultheiss and Jim Collins**

Malignant catarrhal fever (MCF) is receiving increased attention at the Diagnostic Laboratory. We have seen a series of cases in bison and dairy cows. Although MCF is thought to be a sporadic disease, the recent cases indicate that it can be a herd problem. Diagnosis is based on finding typical clinical signs and lesions which may include corneal opacity, nasal and ocular discharge, enlarged lymph nodes, hemorrhage in the urinary bladder, and ulceration in the alimentary tract. Histopathologic lesions of vasculitis in various organs, including kidney, urinary bladder, liver, adrenal gland, intestine, lung, brain, and carotid rete are key in establishing a definitive diagnosis.

The agent of MCF has not been identified definitely but there is evidence that it may be ovine herpesvirus-2 (OHV-2). This virus has not been isolated, but its characteristics have been studied in cells from infected animals. We have developed a polymerase chain reaction (PCR) test that identifies sequences of the viral genome in tissues and blood. We are using this PCR test to investigate the correlation of infection between this virus and clinical disease. The OHV-2 virus has been found consistently in cattle and bison with MCF. A preliminary investigation has identified OHV-2 in blood of some normal cows from herds which have experienced cases of MCF, but not in herds without MCF.

More work needs to be done to identify the prevalence of this virus. Confirmation of the role of the virus as the agent of MCF will depend on transmission studies using experimental animals. We can continue to diagnose MCF based on pathologic lesions and use the PCR test as a further diagnostic tool.

---

MCF Diagnostics: Submit formalin-fixed tissues and/or blood.



Fee--\$21 (histopathology) plus \$21 for OHV-2 PCR.

---

## **BLUE-GREEN ALGAE POISONING**

**Charles Dickie/Rocky Ford Laboratory**

A practitioner recently submitted samples of pond water with a thick layer of slightly bluish, dull green algae floating on top. Six yearling cattle had died and seven more were sick. These Hereford/Angus crosses were from a herd consisting of 175 steers and heifers, weighing 700-750 lbs each. All of the dead and sick animals had algae on their legs and sides. Blue-green algae poisoning was strongly suspected.

Of the seven sick animals, three showed extreme nervousness. They would become alarmed at the approach of the truck and run away, while most of the herd remained calm. Another three animals were down and semi-comatose. The seventh animal was severely dehydrated, and manifested photosensitivity on the nose. Three days later, another sample of similar water was submitted, along with fresh and formalized tissues from a freshly dead animal. Necropsy by the practitioner had shown blue-green algae in the rumen, enteritis, and swollen liver and kidneys. Routine aerobic and anaerobic cultures were run on liver and intestine. The water was tested for nitrate and sulfate, and a sample was sent to Dr. Frank Galey, who has done research on blue-green algae poisoning at the California Veterinary Diagnostic Laboratory.

The water chemistries done at Rocky Ford showed nothing unusual, and no significant bacteria were grown from the tissues submitted. However, histopathology showed massive, almost complete destruction of hepatic parenchyma. Severe renal tubular necrosis and catarrhal enteritis was present. All of these findings contributed to a diagnosis of blue-green algae poisoning, one of the most striking manifestations of which is extreme liver destruction. Dr. Galey confirmed the presence of *Microcystis*, a potentially

hepatotoxic blue-green alga. He was able to produce death in mice with an extract from this alga, which is a standard method of testing the toxicity of an algal species.

Although the herd of 175 animals had been denied access to the stock pond following the practitioner's recommendations, a total of 24 head had died. All animals showing hyperexcitability or a comatose state had died. There is no specific antidote for algal poisoning. Often, animals succumb before treatment can be attempted. Problem algal growth may be controlled by a variety of organic herbicides, as well as copper sulfate. However, not all algal control substances are cleared for use in food-producing animals. When all else fails, read the directions!

---

## **SWINE CORNER--Some Frequent Questions and Answers**

**Bob Glock**

**Sunburn**--The majority of modern pigs have white skin and little hair. At our high altitude, the result is SUNBURN. The usual presentation is young "feeder" pigs placed in outdoor pens a few days earlier. They are usually suspected of central nervous system (CNS) disease because they seem to act normal and then periodically suddenly dip their backs, squeal and go down on their knees as if someone had hit them in the back with a hot poker. They continue to eat and recover but some shade helps. Gilts and sows also get sunburned and there is suspicion that resultant prostaglandin release may disrupt pregnancy. Animals recovering from sunburn may get dry scaly skin and be suspected of having mange. Diagnosis of sunburn is based on observation.

**Agalactia**--Modern gilts and sows are capable of prolific production of pigs and milk but they also are very sensitive to management errors which result in agalactia. Errors usually are related to feeding or housing practices, but mastitis involving only one or two glands can cause complete agalactia with a febrile response. The history as received from inexperienced owners often includes piglet diarrhea which can result from hypoglycemia. Diagnosis is based on the sow's body temperature, udder palpation and history. Milk cultures often identify causative organisms which are frequently gram negative bacteria such as *E. coli* or *Klebsiella* sp. Milk replacers and dry feeds formulated for week-old piglets are commercially available if sows develop agalactia.

**Water Deprivation/Sodium Ion Toxicity**--So-called "salt poisoning" is usually a misnomer. It is very difficult to poison pigs with salt if they have adequate water. However, the salt normally present in most rations can contribute to elevated sodium levels if water is inaccessible or unavailable, as in feeding overly concentrated whey as a sole source of water. Frozen pipes in winter also can be a cause. Diagnosis depends on typical convulsions, histologic evidence of eosinophilic encephalitis, and elevated sodium in serum or cerebrospinal fluid (CSF).

---

If you have any swine questions you would like to see addressed, give us a call!

**WHY NEMATODIRUS INFECTIONS IN CALVES OCCUR IN THE WINTER**

**John Cheney**

This fall, many cow-calf producers will wean their 1997 calf crop. In processing these calves, some producers will include an anthelmintic treatment. Most broad spectrum anthelmintics used today should eliminate 95-100% of the common adult and larval gastrointestinal parasites from these animals. Fecal samples taken 10-14 days following anthelmintic treatment should be negative for worm eggs.

In the Rocky Mountain region, however, it's not uncommon for a producer to worm calves in the fall at weaning, only to check fecal samples on these calves later in the winter and find significant *Nematodirus* eggs.

The data below is from one ranch and is typical of what we see.

Date	Procedure	# of Calves with <i>Nematodirus</i> Eggs			
		Neg	10-20	30-60	70-120
Nov 12	Wormed & Weaned	10	2	0	0
Dec 21	Re-Test	6	1	4	1

During the time the *Nematodirus* egg counts increased, the strongyle egg counts decreased.

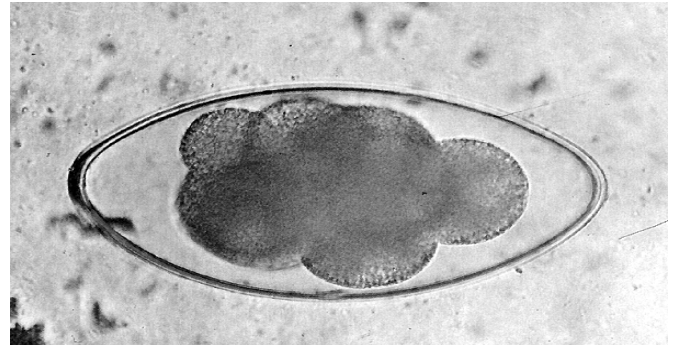
Understanding the difference in the developmental cycle of *Nematodirus*, as compared to other gastrointestinal nematodes in cattle, explains why these infections occur. In the developmental cycle of most of the common nematodes in cattle (*Haemonchus*, *Ostertagia*, *Cooperia*, *Trichostrongyleus*, etc.), the egg passed in the feces hatch to a first-stage, free-living larvae. This larvae then molts to a second-stage, free-living larvae and then molts again to an infective third-stage larval. Under optimum conditions (moderate temperature and moisture), development from egg to third-stage larvae occurs within two weeks. The pre-parasite development of *Nematodirus* is unique in that development to the third-stage larvae occurs within the egg shell. This development is generally very slow and, in temperate climates, takes at least two months. Once the infective larvae is present in the egg, there is often a lag period prior to hatching. Hatching usually occurs during colder weather and in many cases, after a frost. *Nematodirus* larvae have an inherent resistance to climatic extremes far beyond that of other gastrointestinal nematodes and

*Nematodirus* egg

their survival capacity in freezing conditions is especially great. That is why we see calves with *Nematodirus* infections during the winter.

Fecal exam: Submit feces. Fee--\$8.

**ASSESSING VITAMIN A AND VITAMIN E STATUS OF ANIMALS**



**Dwayne Hamar and Cathy Bedwell**

Determining the Vitamin A and E status of animals can be a valuable diagnostic procedure to aid in ensuring good herd health and productivity. Signs of Vitamin A deficiency vary, but the most common is blindness. More subtle effects of Vitamin A deficiency, such as decreased reproductive performance, are important in the livestock industry. The most severe form of Vitamin E deficiency is myodegeneration, but reduced fertility, decreased immune response, and poor growth are major practical concerns. Vitamin E and selenium both have antioxidant properties. Some of the dietary requirement for Vitamin E can be replaced by selenium and vice versa. Vitamin A and E toxicoses can occur but are not a practical problem.

Serum (plasma) and liver are the best samples for assessing the Vitamin A or Vitamin E status of an animal. Serum and plasma Vitamin A and Vitamin E levels are comparable. RBCs destroy Vitamin E, so avoid hemolysis and/or extended contact of the serum or plasma from the RBCs. Separate the serum/plasma from the RBCs as soon as possible and ship the sample cool or frozen. Vitamin A is destroyed by ultraviolet light, so avoid extensive sample exposure to light. We can also analyze forage or feed samples for Vitamin E to determine dietary intake. Our method of Vitamin A analysis does not detect the carotenoids, the natural occurring Vitamin A precursor, but does detect supplemented Vitamin A.

Vitamin A/Vitamin E levels: Submit serum (plasma) or liver. Fee--\$12 for Vitamin A or Vitamin E; \$16 for Vitamin A and Vitamin E.

**SULFATE-ASSOCIATED POLIOENCEPHALOMALACIA (PEM): Part II. Dietary analysis for estimation of total sulfur intake**

**Dan Gould and Dwayne Hamar**

As indicated in Part 1 of this article (see Volume 1, Number 2), PEM in our region is associated with high sulfur intake. As part of the work-up for an outbreak of PEM, we recommend that when sulfate-associated PEM is suspected, the total sulfur intake be estimated to see if it exceeds the NRC recommended maximal tolerated level of .4% of dry matter intake (DMI). Accounting for the sulfur content of all dietary components, as well as water, is required.

**Estimation of the TOTAL daily per head intake of sulfur (S) as %S DMI should be done as follows:**

1. Determine the contribution as sulfur in water to the total %S DMI.

--The water analysis is usually reported in ppm sulfate (= mg  $\text{SO}_4/\text{L}$ ). Sulfate is 1/3 sulfur; therefore, ppm sulfate divided by 3 and divided by 1000 = g S/L.

--With appropriate consideration for temperature and type of animal (Nutrient Requirements of Beef Cattle, NRC, 1996), estimate the daily water intake (about 7% body weight in kg (BW) @ 40°F; 10% BW @ 70°F; 18% BW @ 90°F).

--Liters  $\text{H}_2\text{O}$  consumed / d X g S/L = g S consumed/head/d.

--To evaluate in terms DMI maximum, estimate DMI/head/d (about 3% BW in kg).

--Then, % S from water as DMI = g S/head/d divided by kg DMI divided by 10.

2. Determine the contribution of sulfur in the diet to the total %S.

--Sulfur in feed ingredients or mixed diets is usually reported in analyses as % S DM. Therefore, the % S can be used directly.

--If individual feed components are analyzed, multiply the % S DM of each ingredient by the fraction of the diet that the ingredient represents (for example, if alfalfa hay is .4% S DM and is fed at 75% of the diet, multiply .4% by .75 and you get .3% as the contribution to the total S intake from alfalfa). To determine total intake of % S contributed by the feed, add all individual % S DM values.

3. To estimate the TOTAL daily per head intake of sulfur as if it was all DMI -- Add the % S DMI from water to % S DMI of all feed. This value represents all sulfur consumed equated to DMI, and can therefore, be used to evaluate in terms of the NRC maximal tolerated level of S, .4% DM.

It is surprising how drastically the amount of water consumed, especially in hot weather, can affect sulfur intake, the risk of excessive ruminal  $\text{H}_2\text{S}$  production and the occurrence of PEM. The following example illustrates this:

Assume cattle grazing on forage that is .2% S and using a water source with 2000ppm sulfate. At an ambient temperature of about 40°F, the estimated total S intake as DM is about .36%; below the maximum tolerated level of .4%. However, at 90°F, the water consumption is increased sufficient to elevate the total S intake to .6%, which increases the risk of PEM.

---

**MYSTERIOUS NONSUPPURATIVE ENCEPHALITIS IN CAPTIVE ELK**

---

**Gary Mason**

A previously unrecognized form of nonsuppurative encephalitis in captive elk has emerged in Colorado. We had four case submissions in the summer of 1996 and nine cases this summer and fall. Most cases have been from captive herds on the plains, but a single case was from a captive herd on the Western Slope. There is no apparent sex or age predilection. Affected elk suffer from a progressive disease characterized clinically by exophthalmia, inappetence and posterior weakness, followed by recumbency, progressive dementia, blindness, seizures and death. Supportive therapy has proven ineffective and the disease appears uniformly fatal. There are no consistent gross post-mortem lesions. Histologically, all affected elk have a nonsuppurative encephalitis characterized by microvascular injury with perivascular edema and cuffing of cerebral vessels by mononuclear cells. These microscopic lesions suggest a viral etiology. Diagnostic efforts aimed at identifying an etiologic agent are ongoing and include serology, virus isolation, immunohistochemical demonstration of viral antigen in fixed tissue, and identification of viral nucleic acid by polymerase chain reaction (PCR). Some testing is being performed at the National Veterinary Services Laboratory in Ames, Iowa. Diagnostic samples requested from field cases include serum or clotted blood in red top tubes, whole blood in both purple and green top tubes, fresh and formalin fixed brain tissue, and formalin-fixed tissue from all other parenchymal organs. This neurologic disease has distinctly different lesions than the spongiform encephalopathies reported in cervids. Please contact Dr. Gary Mason if you have a case of this mysterious disease.

## What's Inside This Issue of LabLines

*Clostridium perfringens* Genotyping  
Changes in Antimicrobial Susceptibility Testing  
Soft Tissue Sarcomas  
New Toxicology/Chemistry Testing  
Malignant Catarrhal Fever  
Mysterious Encephalitis in Elk

Blue-Green Algae Poisoning  
Swine Corner  
*Nematodirus* in Calves  
Assessing Vitamin A & E Status  
Polioencephalomalacia

