Letter from the Director

What happened to spring? It’s summer already! We hope you find the updates in this issue of LabLines informative and useful. Look inside to find out the changes occurring at the Rocky Ford and Western Slope Branch Laboratories. We plan to keep these branch laboratories a vital part of our system and are partnering with the Colorado Department of Agriculture to increase our ability to serve you, especially in regards to regulatory diseases. We have filled our open pathologist position at the Fort Collins Laboratory with the return of Dr. E.J. Ehrhart who will arrive in August. Dr. Ehrhart completed his pathology residency and PhD at Colorado State University a few years ago and has been at the University of Illinois Diagnostic Laboratory since graduation from CSU.

In January, we had an excellent meeting with our External Advisory Committee; see inside for a listing of these people with a special note of thanks to them. This summer, we have our re-accreditation site visit by the American Association of Veterinary Laboratory Diagnosticians and look forward to their suggestions to improve our laboratory. Issues relating to Chronic Wasting Disease in deer and elk, and Biosecurity to protect our food animal industry are forefront at the laboratory. In addition to this, we continue to strive to provide quality diagnostic services for all animal species.

We remain active with the Colorado Veterinary Medical Association and look forward to an exciting, fun-filled, and informative Fall Annual Conference at Keystone, September 7-11th. Hope to see you there!

Barbara Powers, DVM/PhD/DACVP

Barbara Powers, DVM/PhD/DACVP

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Animal Diagnostic Laboratory
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DIAGNOSTIC LABORATORY ADVISORY COMMITTEE

Every January, we meet with our Diagnostic Laboratory Advisory Committee. These dedicated people spend over half a day with us and provide valuable input in assessing our service and pointing us in the correct direction for the future. This year, they have been called on repeatedly, in addition, to assist in obtaining support and funding for our planned new building. This summer, we will need their assistance as we have our five-year site visit for re-accreditation by the American Association of Veterinary Laboratory Diagnosticians. Please feel free to contact any of these people (or us directly) if you have comments about our laboratory.

DIAGNOSTIC LABORATORY ADVISORY COMMITTEE MEMBERS

Dr. Joan Bowen  5036 ECR 60  Wellington, CO  80549
Mr. Norm Brown   8167 NCR 11  Wellington, CO  80549
Dr. Wayne Cunningham  Colorado Dept of Ag  Denver, CO  80215
Dr. Robyn Elmslie  3550 S. Jason Street  Englewood, CO  80110
Dr. Jerome Geiger  5129 Kiowa Drive  Greeley, CO  80634
Dr. Greg Goodell  6875 CR 9  Loveland, CO  80538
Dr. Mike Gotchey  1878 Lincoln Avenue  Stbt Springs, CO  80487
Dr. Mary Gray  Coop Extension  Fort Collins, CO  80523
Dr. Marv Hamann  1124 - 20th Lane  Pueblo, CO 81006
Mr. Ed Hansen  4554 CR 74E  Livermore, CO  80636
Dr. Lenny Jonas  3695 Kipling Street  Wheat Ridge, CO  80033
Dr. Tony Knight  CSU VTH  Fort Collins, CO  80523
Dr. Larry Mackey  PO Box 336204  Greeley, CO  80632
Dr. Del Miles  5626 W. 19th Str, Suite A  Greeley, CO  80634
Dr. Wendell Nelson  CSU VTH  Fort Collins, CO  80523
Mr. Gene Schoonveld  317 W. Prospect  Fort Collins, CO  80526
Dr. Todd Towell  2601 Midpoint Drive  Fort Collins, CO  80525
Dr. T.P. Welsh  1336 W. Elizabeth  Fort Collins, CO  80521
Dr. Brian Wooming  16634 WCR 33  Platteville, CO  80651

AN UPDATE ON CHRONIC WASTING DISEASE
--Terry Spraker

Since the last issue of LabLines, a tremendous amount of activity has occurred in regards to captive elk with chronic wasting disease (CWD) in Colorado and surrounding states. We have worked closely with the Colorado Department of Agriculture, USDA, and other agencies in the surrounding states to address this problem. Within Colorado, positive cases have been found in nine herds. To date, over 3000 captive elk have been killed, including elk with trace-backs from positive cases and captive elk in the endemic area of northeastern Colorado. Of the first group of over 1500 elk, 46 were found positive in various herds. For the second group of nearly 1500 elk, four were found positive. The disease has been found in captive elk in South Dakota, Montana, Nebraska, Kansas, Oklahoma and Colorado. All of these herds have been depopulated except for the Oklahoma herd.

As has been highly publicized in the newspapers, CWD was found in wild deer on the Western Slope for the first time. This spring, we assisted the Colorado Division of Wildlife in determining the extent of the disease by performing the testing for them. We tested over 700 samples that arrived in two batches. We provided results in 2.5 days for the first batch and five days for the second batch. It appears from these groups of samples, that the incidence of CWD is less than 1% in these deer. We hope to be able to continue to work with the Division of Wildlife this coming fall to assist them with testing hunter-killed deer and elk. Last year, we provided CWD testing services for hunters who brought their deer or elk heads to us directly, delivering results in 5 to 10 working days. We will continue to offer this service to hunters this coming fall. The Colorado Veterinary Medical Association (CVMA) and member veterinarians have offered their assistance in collecting samples for hunters.

The immunohistochemical test is the “gold standard” to diagnose CWD in elk and deer. We developed the protocol for this test in the high volume automated form by working with Dr. Katherine O’Rourke, ARS/USDA/Pullman,
Washington. Dr. O'Rourke developed and produced a monoclonal antibody (F99/97.6.1) that has been validated for CWD in deer and is presently being validated in elk. This stain has been used on approximately 10,000 elk with extremely good results. The stain also has been used on approximately 3,000 deer and results have been very accurate in the diagnosis of CWD, even with very early cases.

Further work that will be accomplished in the next few months by collaborating with Dr. Mo Salman of the Animal Health Population Institute includes validation of several more rapid diagnostic tests for CWD. These rapid CWD tests are also being validated at federal laboratories. If these prove to be valid tests for CWD, a much larger volume of tests can be completed in a shorter period of time. We hope to have these tests in place by August if they are shown to be accurate in the diagnosis of CWD. In addition, in conjunction with USDA, two projects dealing with mapping of abnormal prion protein in whitetail deer and in elk should be completed. This mapping study will help us to understand how the prion spreads through the brain.

The origin of CWD is unknown. There are many theories and rumors about the origin of CWD in Colorado but there is no scientific proof to confirm these theories. We may never be able to answer this question. Some have stated that the disease may have started in wildlife research facilities, either in Colorado or Sybille, Wyoming since the disease was first found in these facilities. The literature states that CWD was first recognized by game biologists in Colorado Division of Wildlife (CDOW) pens in 1967. However, the CDOW pens were not built until 1968. There were wildlife facilities in the location of the present-day CDOW research facility deer pens, but these pens were originally built by personnel from Colorado State University who were working with deer captured at Rocky Flats. Deer pens were also built in the early 1960s at the CSU Foothills Campus. The experiments done at these pens were terminated by post-mortem examination of the deer and no deer with signs of CWD were observed. CWD was first recognized in the CDOW deer pens in the early 1970s, but was not confirmed as a spongiform encephalopathy until 1979 by Dr. Beth Williams. Shortly after that, deer with CWD were discovered in the Sybille Wildlife Research facilities in southeastern Wyoming. Some people believe that the disease may have originated in one of these research facilities because the deer in these facilities may have been exposed to domestic sheep with Scrapie, however, Scrapie was never observed in any of the sheep in these facilities.

One theory is that deer spontaneously developed CWD in the wild and some of these infected deer were trapped and brought into captivity. Because of the close contact in the pens, the disease spread and became established. Another theory is that close contact with deer and domestic sheep in areas either in southeastern Wyoming or northeastern Colorado allowed Scrapie to cross the species barrier and infect deer. None of these theories have been proven and the search for the origin of this disease will probably be fruitless. CWD has spread to various states and the most likely method of spread is through movement of animals, both captive and wild. It will be extremely difficult to control this disease in the wild because of the potential of environmental contamination. In regard to captive elk herds, at the present time, all of the known infected herds have been depopulated. Also, all of the herds in the endemic area have been killed, except one herd in Craig, CO. Depopulation of these captive herds is hoped to reduce the spread of CWD in the captive elk industry in Colorado and throughout the United States.

A new disease syndrome in captive North American elk (Cervus elaphus) has been recognized in the past year. The first set of cases included the occurrence of infertility in elk cows and weak calves with enteritis and respiratory disease. Sick calves were unresponsive to treatment with antibiotics and intravenous fluids. Sections of the lungs were positive for bovine respiratory syncytial virus (BRSV) by fluorescent antibody (FA). The lungs and spleens were positive for bovine viral diarrhea virus (BVDV) by FA, immunohistochemistry (IHC) and polymerase chain reaction (PCR). BVDVs were isolated from whole blood samples of the calves and determined to be type 1aBVDV by PCR. Tissues from the calves were negative for IBR and PI-3. Of all elk on the premises, 12.5% were seropositive for type 1aBVDV. However, an accurate vaccination history was not available to aid in interpretation of these titers.

In the second case, a male elk calf was presented for necropsy. Gross findings included petechial hemorrhages on the serosal surface of the lung and mucosal surface of the urinary bladder, multiple ulcers in the abomasum, and fluid-filled intestines. PCR tests for epizootic hemorrhagic disease virus (EHDV) and bluetongue virus were negative, as well as FA tests for...
IBR, BVDV, BRSV, and PI-3. Hemolytic E. coli was isolated from the intestine and no clostridial organisms were cultured. A non-cytopathic type 1b BVDV was isolated from the lung. Further details of the herd’s health were not available.

Problems in the third set of cases included infertility in first-calf heifers, abortions, stillbirths, and deaths in calves less than 2 weeks of age to scours. Of the calves examined, two had pneumonia and one also had a perforated ulcer and hepatocellular necrosis. Sections of the lungs were negative for IBR, BVD, BRSV, and PI-3 by FA. Pseudomonas and E. coli were cultured from the lungs. Fecal samples were negative for Salmonella, Cryptosporidia, and enteric viruses. Tissues from a 7-month-old calf with a two-week history of weight loss were submitted for histopathology. No significant histopathological lesions were noted. The tissues were positive for BVD by IHC but negative for BVDV by PCR and negative for EHDV. Fresh tissues were not available for VI. Ten-month-old calves from the same cohort were seronegative for BVDV. Factors under consideration for a role in this syndrome include BVDV, nutrition (copper deficiency), exposure to toxins, bacteria, parasitism, and management practices.

This note is to alert veterinarians that free-ranging and farmed wild cervids are susceptible to infection with BVDV and other pestiviruses. Surveys of free-ranging elk have demonstrated sero-prevalences of 30% to 87% in Colorado. Experimentally, elk have been shown to be susceptible to infection with BVDV, but the infection did not result in clinical disease. Here, we report the first isolations of BVDV from captive elk, and the first report of BVDV in association with disease in this species. Further characterization of these viruses is being performed by Dr. Julia Ridpath, NADC. The impact of BVDV on the health of captive or free-ranging elk is unknown at this time.

IONOPHORES
—Dwayne Hamar, Hana Van Campen, and Cathy Bedwell

Ionophores are routinely added to diets of ruminant animals to increase feed conversion and to decrease acidosis of feedlot cattle not adapted to high carbohydrate rations. Many different ionophores are used, but monensin (Rumensin®) and lasalocid (Bovatec®) are the most common in the Fort Collins/Loveland/Greeley area. Ruminant animals fed monensin and lasalocid have shown a decrease in feed intake but an increase in weight gain. Ionophores fed to cattle cause an increase in propionate with a corresponding decrease in acetate and butyrate with a concurrent decrease in methane production. Ionophore’s primary mechanism of action is related to their ability to bind and transport ions across membranes. Ionophores also are noted to control coccidia in poultry.

Like all xenobiotics, ionophores may be toxic, especially if consumed by non-target species or in excess quantity as occurs in mixing errors. A majority of ionophore toxicosis in ruminants and poultry are a result of feed mixing errors. Clinical signs include weakness, ataxia/incoordination, depression, and feed refusal. Feed refusal usually occurs after sufficient ionophores have been consumed to cause toxicity. Ionophore toxicosis affects the heart, muscular system, nervous system, and digestive tract, accompanied by elevated CK, LDH, and AST levels. Grossly, skeletal and cardiac muscles usually exhibit pale streaks with increased friability. There also may be pulmonary congestion and edema. The effects on the respiratory system can be confused with pneumonia. Histologic lesions are primarily muscle fiber swelling, loss of striation, and some fiber necrosis.

The maximum approved concentration of monensin is approximately 0.7mg/kg/day for slaughter cattle and 0.4mg/kg/day for growing or mature cattle. The LD50 and LD10 for cattle are approximately 26mg/kg and 11mg/kg, respectively, which indicates a very steep dose response curve. A common ionophore available for use in the poultry industry is maduramicin (Cygro®). This ionophore is not absorbed and is excreted in high concentrations. The feeding of poultry litter to cattle and sheep is a common practice in some parts of the country. Toxicoses have resulted from feeding maduramicin-contaminated poultry litter to cattle.

Horses that are fed or accidentally consume feed intended for the cattle in a feedlot, also can develop toxicosis. Ionophore toxicoses in dogs and cats usually are a result of contamination in food, or consuming premixes or feed designed for other species.

Confirming ionophore toxicosis usually involves detecting ionophores in feed or stomach contents. Ionophores cannot be detected in tissues or serum.

FROM THE WESTERN SLOPE
—Darrel Schweitzer

During the past few months, the Western Slope has been very busy with Trichomonas cultures. We have found several positive herds in three counties in western Colorado. This disease continues to plague producers, as many bulls in this area run together during the summer months in cooperative grazing associations. Increased awareness of the widespread nature of the problem has been instrumental in finding new positive herds, and we urge all veterinarians to speak with their producers about this disease.

At the same time, we have experienced a marked decline in the number of herd rams positive for Brucella ovis. During the winter months, this disease often takes a backburner in our minds, but a decrease in incidence is not an absence. The next few months are the time to be doing ram testing. Unlike in the fall, when rams are more active sexually, testing rams in the spring stands a better chance of finding all positive rams. A few will be infected recently and thus not yet seropositive.
Brucella ovis, as well as trichomoniasis, are diseases regulated by the state. Forms are available from the State Veterinarian’s office for use in submitting samples for both these diseases. It should be filled out completely, especially with owner’s name and address, and animal identification.

For those submitting Brucella ovis testing, please be sure and review the changes in the testing site. This is explained in the shaded box below as a “special note.” By taking a moment to read this note, you will ensure smooth testing and reporting of your Brucella ovis results during the upcoming testing season.

Trichomonas culture—Submit a preputial wash/vaginal wash in a lactated ringer solution. Fee=$8 (1-50), $7 (51+).

B. ovis serology—Submit 0.5-1.0ml serum. Fee=$7 (minimum lab fee) (1 sample); $4 (2-50), $3 (50+).

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**SPECIAL NOTE FROM THE WESTERN SLOPE**

The next few months is the time to be doing ram testing for *Brucella ovis*. Starting immediately, the Western Slope Laboratory will be performing ALL of the *B. ovis* ELISA tests. All samples should be sent directly to the Western Slope Laboratory and not the Fort Collins Laboratory. To avoid delays in receiving your results, please send *B. ovis* serum samples to:

**Western Slope Animal Diagnostic Laboratory**

425 29th Road

Grand Junction, CO 81501

(970-243-0673)

Please send 0.5-1.0ml of serum. Do not send unseparated whole blood, as either winter freezing or summer heat will likely result in severely hemolized, unusable samples. Our intent with centralizing this testing is that as the accessions increase, additional testing days will be added (currently, the test is performed only on Friday). This will improve turnaround time for results. If you send the samples to the Fort Collins Laboratory, the samples will be forwarded to the Western Slope Laboratory, but this will create a shipping delay which will cause a delay in receiving your results. The Laboratory is closed on the noon hour so please note on your courier form (Fed Ex/UPS/Express Mail) that the package should, however, be dropped off.

Thank you for adjusting to this change so that we can serve you better!

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**WHAT’S UP WITH NEOSPORA SEROLOGY RESULTS?**—The test kit used by the Parasitology Laboratory to test *Neospora caninum* antibody in bovine serum is currently unavailable. The IDEXX Laboratories are experiencing difficulty in the manufacture of the kits. Unfortunately, we do not have a projected date to begin testing again. We have a backlog of samples dating from March 20, 2002, and will test all stored samples as soon as the test kits are available. We apologize for the delay.

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**SPLLENS!**

---Barb Powers

Examination of enlarged spleens or splenic masses removed from dogs at surgery can be a challenge for the veterinary clinician as well as the veterinary pathologist. The problem begins in getting an enlarged spleen or a large splenic mass to the laboratory with proper fixation. Typically, these samples are quite large and so it is difficult to submit the entire spleen. An alternative approach is to submit multiple sections of the spleen. It is very difficult to tell which parts of the splenic mass are going to be the most diagnostic and this can potentially lead to sampling error. The best strategy is to submit multiple samples throughout the spleen consisting of at least five separate areas, preferably with different consistencies. These should be fixed in the appropriate volume of fixative and mailed to the laboratory. It is always wise to retain a portion of the splenic mass in the clinic in case the first sections fail to be diagnostic.

At the laboratory, we will make multiple sections through the spleen. We try to make at least three to five sections. If the first sections of the splenic samples reveal only blood or hematoma formation, we may go back and make further sections. If these still fail to reveal any neoplastic process and neoplasia is expected, then further submission of the tissue saved back in the clinic may be advisable. The difficulty is clearly that most splenic tumors are associated with large areas of hemorrhage and necrosis, so it can be very difficult to distinguish between tumor and hematoma formation on gross inspection.

In three separate reports, representing 2,000 total enlarged spleens with splenic masses, one-third of the samples were benign or hematomas or areas of nodular lymphoid hyperplasia. Two-thirds of the splenic masses were malignant and, of these tumors, two-thirds were hemangiosarcomas. In one study, it was found that at least five sections of a splenic mass were necessary to feel confident for a diagnosis of hemangiosarcoma versus hematoma.

Hematomas are the result of a benign process that occurs within the spleen. We are not certain as to why they develop. They frequently are associated with areas of extramedullary hematopoeisis. They can cause clinical signs similar to hemangiosarcoma, such as acute collapse and blood in the abdomen. These are, however, benign conditions. Nodular lymphoid hyperplasia is another benign condition that can lead to hematoma formation. Typically, with nodular lymphoid hyperplasia, the splenic mass is white whereas with hematomas, the splenic mass is darkly colored and bloody. Another benign tumor of the spleen is the myelolipoma. This is a nodular proliferation of extramedullary hematopoeisis and fat, resembling normal bone marrow in the spleen. These are more common in cats than dogs.

Vascular tumors of the spleen include hemangiomomas. These are relatively infrequent but can be associated with
hematomas. The most common malignant vascular tumor is hemangiosarcoma. Hemangiosarcomas of the spleen typically carry a guarded prognosis with a median survival of 1 to 3 months with surgery alone. In one study, these tumors were graded for degree of differentiation, number of mitotic figures, nuclear pleomorphism, and necrosis. Survival was prolonged a few months using doxorubicin chemotherapy in patients with tumors that were well-differentiated (versus those that were anaplastic), where there was no gross evidence of metastatic disease, and where the spleen had been removed. In various reports, use of chemotherapy after surgery results in median survival times of 5 to 11 months.

Another type of malignant tumor that can occur in the spleen is malignant lymphoma. This can be difficult at times to distinguish histologically and grossly from nodular lymphoid hyperplasia. Usually, malignant lymphoma is more diffuse and involves a larger portion of the spleen. Also, the population of cells in malignant lymphoma is monotonous versus nodular lymphoid hyperplasia which, by definition, is more nodular with a mixed population of cells. Another type of discrete cell tumor that can invade the spleen is mast cell tumor. These can occur in either dogs or cats, although more frequently in cats. Usually, this causes diffuse enlargement of the spleen and is generally readily distinguishable histologically from lymphoid tumors.

Other tumors that can occur in the spleen arise from the stromal elements. These can include leiomyosarcoma, fibrosarcoma, liposarcoma, extraskeletal osteosarcoma, undifferentiated sarcoma, malignant fibrous histiocytoma, and malignant histiocytosis. In one study of these non-lymphoid and non-vascular tumors of the spleen, mitotic rate was found to be of importance in estimating survival. Those tumors that had greater than 9 mitoses per 10 high power fields had a median survival of only one-month, while those tumors with a lower mitotic rate had a 40% chance of survival at one-year. In subsequent studies of these tumors, there was found to be a continuum of tumors termed fibrohistiocytic tumors, ranging from malignant fibrous histiocytomas to malignant histiocytosis. Sometimes, these had a substantial degree of lymphoid proliferation as well. The amount of lymphoid proliferation relative to the histiocytic proliferation was indicative of a different prognosis with less lymphoid cells and more histiocytic cells indicating a poorer prognosis. Tumors that were very histiocytic typical for malignant histiocytosis tended to have the worst prognosis.

Because of the above-mentioned challenges associated with sampling and interpretation of splenic masses, please feel free to call the pathologists any time you feel the histopathologic diagnosis does not concur with the clinical impression. It may be necessary to cut in more sections or to re-evaluate the samples in these cases.

Histopathology—Submit sample as described above in formalin. Fee=$23.50 for 3 slides, $7.00 for each additional slide over 3.

USER’S OF BIOPSY SERVICES

Please do NOT do a “SOLS” (small opening, large sample) by putting a large unfixed sample in a formalin-filled container with a small opening. Once it’s fixed, we can’t get it out!

QUESTIONS ABOUT WEST NILE VIRUS?

See Special Interest--“The West Nile Virus from the Ohio State University” under our homepage at http://www.cvmbs.colostate.edu/dlab or call Dr. Hana Van Campen at 970-491-1281, the State Veterinarian’s Office at 303-239-4161, or the Colorado Department of Public Health at 303-692-3090.

A YEAR’S WORTH OF MYCOLOGY  —Doreene Hyatt

We have had 396 requests for fungal culture between January 1, 2001 and March 1, 2002. In that time, 44% of the requests have been to culture canine samples, 27% have been for equine samples, and 20% for feline samples. A mixture of animal species makes up the rest of the test requests. The tables that follow give percent of the total of the organisms isolated from each animal species. The number in parenthesis indicates the number of samples from each animal species.
<table>
<thead>
<tr>
<th>Fungus</th>
<th>Canine % (n=175)</th>
<th>Equine % (n=106)</th>
<th>Feline % (n=78)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspergillus sp.</td>
<td>10</td>
<td>7.5</td>
<td>6.4</td>
</tr>
<tr>
<td>Penicillium sp.</td>
<td>5</td>
<td>3.8</td>
<td>3.8</td>
</tr>
<tr>
<td>Trichophyton</td>
<td>1</td>
<td>0.9</td>
<td>1.3</td>
</tr>
<tr>
<td>Zygomycete</td>
<td>1.7</td>
<td>1.9</td>
<td>1.3</td>
</tr>
<tr>
<td>Cladosporium</td>
<td>0.6</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>Fusarium</td>
<td>0.6</td>
<td>1.9</td>
<td></td>
</tr>
<tr>
<td>Cryptococcus</td>
<td>1</td>
<td>4.7</td>
<td></td>
</tr>
<tr>
<td>Microsporum canis</td>
<td>0.6</td>
<td></td>
<td>10.3</td>
</tr>
<tr>
<td>Sporothrix schenckii</td>
<td>0.6</td>
<td></td>
<td>1.3</td>
</tr>
<tr>
<td>Alternaria</td>
<td>0.9</td>
<td></td>
<td>10.3</td>
</tr>
<tr>
<td>Chaetomium sp.</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epicoccum</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coccioides immitis</td>
<td>0.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ulocladium</td>
<td>0.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bipolaris sp.</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Verticillium sp.</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curvularia sp.</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Torulopsis candida</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scopulariopsis sp.</td>
<td>0.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curvularia sp.</td>
<td></td>
<td>1.3</td>
<td></td>
</tr>
<tr>
<td>Trichosporon beigeli</td>
<td>1.7</td>
<td></td>
<td>1.3</td>
</tr>
<tr>
<td>Sporotrichum sp.</td>
<td>1.7</td>
<td></td>
<td>1.3</td>
</tr>
<tr>
<td>Multiple isolates</td>
<td>14</td>
<td>27</td>
<td>14</td>
</tr>
<tr>
<td>No fungus isolated</td>
<td>49.7</td>
<td>28</td>
<td>37</td>
</tr>
<tr>
<td>Mixed contaminants</td>
<td>9</td>
<td>14</td>
<td>10</td>
</tr>
</tbody>
</table>

For those samples in the table above that had more than one fungus found, the table below gives the fungal isolates that were found in the samples. The numbers are given as a percentage of the total isolates from that animal species. The number in parenthesis indicates the number of samples that had multiple isolates recovered.

<table>
<thead>
<tr>
<th>Fungus</th>
<th>Canine % (n=58)</th>
<th>Equine % (n=78)</th>
<th>Feline % (n=24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspergillus sp.</td>
<td>17</td>
<td>9.6</td>
<td>16.7</td>
</tr>
<tr>
<td>Epicoccum</td>
<td>1.7</td>
<td>2.7</td>
<td>12.5</td>
</tr>
<tr>
<td>Cladosporium</td>
<td>15.5</td>
<td>6.9</td>
<td>16.7</td>
</tr>
<tr>
<td>Alternaria</td>
<td>5</td>
<td>13.7</td>
<td>8.3</td>
</tr>
<tr>
<td>Penicillium sp.</td>
<td>10</td>
<td>8.2</td>
<td>16.7</td>
</tr>
<tr>
<td>Fusarium</td>
<td>1.7</td>
<td>6.8</td>
<td></td>
</tr>
<tr>
<td>Trichophyton</td>
<td>3.5</td>
<td>5.1</td>
<td></td>
</tr>
<tr>
<td>Cryptococcus sp.</td>
<td>12</td>
<td>2.7</td>
<td></td>
</tr>
<tr>
<td>Chrysosporium</td>
<td>1.7</td>
<td>5.5</td>
<td></td>
</tr>
<tr>
<td>Trichosporon beigeli</td>
<td>1.7</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td>Zygomycete sp.</td>
<td>1.7</td>
<td>9.6</td>
<td>16.7</td>
</tr>
<tr>
<td>Sporotrichum sp.</td>
<td>2.7</td>
<td>2.7</td>
<td>4.2</td>
</tr>
<tr>
<td>Candida sp.</td>
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<tr>
<td>Monilia sitophila</td>
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<tr>
<td>Exophiala</td>
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<tr>
<td>Sporothrix schenckii</td>
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<tr>
<td>Chaetomium sp.</td>
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<tr>
<td>Rhodotorula glutinis</td>
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<tr>
<td>Sporobolomyces salmonicolor</td>
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<td></td>
</tr>
<tr>
<td>Apophysomyces elegans</td>
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<td>1.4</td>
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</tr>
<tr>
<td>Acremonium sp.</td>
<td>1.4</td>
<td></td>
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<tr>
<td>Scopulariopsis sp.</td>
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<tr>
<td>Trichothecium roseum</td>
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</tr>
<tr>
<td>Verticillium sp.</td>
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</tr>
<tr>
<td>Microsporum sp.</td>
<td></td>
<td>4.2</td>
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<tr>
<td>Mixed contaminants</td>
<td>3</td>
<td>12.3</td>
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GET TO KNOW YOUR LABORATORY
Histopathology Laboratory

The histopathology laboratory staff keeps our pathologists working by providing them with high-quality slides so they can complete their diagnoses on your cases. Preparation of quality slides is a critical step in proper pathologic diagnoses and the pathologists depend heavily on the histotechnicians skill. We are pleased to have highly skilled histotechnicians that do an amazing amount of work.

Robert Zink supervises the histopathology laboratory. He received his BS degree in Biological Sciences in 1969. He received his histotechnician certificate in 1972. Robert has been with us for 25 years. Her experience enables her to be very efficient in addition to her high-quality work. Maryanne Tryjan received her histotechnician certification in 1973 and has been with us for 23 years. In addition to her excellent daily service work, she assists with many of the research projects. Currently, we have a position open for a histotechnician after the retirement of Frank Aquino after 23 years of service. The histotechnicians rotate through the various stations in the slide-making process (processing/embedding, sectioning and staining), although every day they all do some sectioning. On an average day, this group prepares 280 slides, and some days do as many as 400! In addition, Bruce Cummings is a research associate who has been with us for 20 years. His responsibility is performing the daily Immunohistochemistry staining procedures.

ADMISSIONS TO VETERINARY SCHOOL
—Pat Schultheiss and Sherry McConnell

Colorado State University has just completed another cycle of admissions, and we look forward to the August arrival of our newest professional colleagues, the Class of 2006. Diagnostic Laboratory clients and faculty play an important role in the admissions process. Many of our clients have mentored candidates for admission and this is an important contribution to the future of our profession. Five of the nine college faculty members on the admissions committee are associated with the Diagnostic Laboratory. An additional member of the committee is a practicing veterinarian representing the Colorado Veterinary Medical Association (CVMA).

Colorado State University looks at three separate pools of candidates—those that will be supported by the State of Colorado, those supported by other states through the Western Interstate Commission for Higher Education (WICHE), and a group of at-large students not supported by a State government (self-supporting). Admissions criteria are the same for all three groups, with the exception that interviews are conducted with some of the applicants in the Colorado pool but not with WICHE and non-sponsored students.

The CSU admissions process evaluates the quality of the applicant as a whole and does not use a rigid point system to rank the various aspects of an application. Each application is read by two committee members. The first step of reading an application is determining whether the applicant has the required academic preparation and an academic record that indicates the applicant will probably succeed in a rigorous science curriculum. Applicants considered academically unqualified will not be admitted even if they have strengths in other areas. Knowledge of the veterinary profession and motivation to enter the profession are assessed. This knowledge should be evident in experiences such as working in veterinary clinical practice and having experience with animals. Experience in biomedical research also is valued. Participation in community service and diverse background and experiences are desirable qualities. Character, work ethic, and communication skills are evaluated on the basis of the applicant’s personal essay and letters of recommendation. Committee members try to weigh these factors as to whether the applicant will be successful in the academic program and be a credit to the profession.

There are more qualified applicants than positions in the class, so not every qualified applicant will be admitted. When an applicant is denied a position in the incoming class, the Office of Admissions can arrange for an appointment with a committee member or the pre-veterinary advisor to review the application and get advice on how to strengthen it. Sometimes, veterinary practitioners are disappointed when a valued employee or an individual they are mentoring is not admitted. A veterinarian working with a handful of applicants cannot see how those applicants rank within the entire pool of 1300 people. In many cases, the applicant does indeed appear to be a fine person and valuable member of a clinic staff but has a weak academic record, which the veterinarian may not even know about. With the applicant’s permission, a mentoring veterinarian can attend the application review meeting and learn more ways to help the applicant.

Many of the applicants’ personal essays include praise for a particular veterinarian who was the inspiration to aspire to a

(L to R) Bruce Cummings, Robert Zink, Maryanne Tryjan, Yang Sun Shin
career in veterinary medicine. The help and encouragement of mentors is appreciated by the applicants, the College, and the profession as a whole.

Ms. Marti Stokes in the Dean’s Office can provide information about the admissions process. She can be contacted at 970-491-7052.

**EFFECT OF OPHTHAMOLOGY REAGENTS ON FELINE HERPESVIRUS-1 PCR PROCEDURE**
—Noelle C. LaCroix, Anita Schiebel, and Hana Van Campen

Feline herpesvirus-1 (FHV-1) is a common infectious cause of upper respiratory and ocular disease in cats. We offer a polymerase chain reaction (PCR) procedure to detect FHV-1 in conjunctival scrapings and other tissues. Frequently, conjunctiva from affected cats are exposed to ophthalmic reagents such as Rose Bengal, fluorescein and proparacaine solution prior to collection of the conjunctival samples.

To determine if these reagents inhibit the PCR test, feline kidney cells were inoculated with FHV-1, or treated with media alone (uninfected controls), and incubated for 72 hours to allow the virus to grow. FHV-1 infected and uninfected cells were treated with 0.5% proparacaine solution, fluorescein solution, fluorescein or Rose Bengal strips, then placed into tubes containing Hank’s balanced salt solution (PCR transport media).

Each tube was refrigerated at 4°C for 24 hours to simulate shipment conditions, and then stored at -70°C. DNA was extracted and then assayed for FHV-1 DNA by PCR. FHV-1 DNA was amplified from samples of FHV-1 infected cells subjected to all treatments. All uninfected control wells yielded negative FHV-1 PCR results. Therefore, these ophthalmic reagents did not inhibit the detection of FHV-1 by PCR.

To determine if ophthalmic reagents used in vivo interfere with the FHV-1 PCR, we are currently comparing the FHV-1 PCR results of conjunctival swabs taken from cats prior to and after treatment.

**PCR for FHV**—Submit sample in PCR transport media. Fee=$24.

**CHANGES AT ROCKY FORD**

After 39.5 years of distinguished service to the producers and veterinarians of southern Colorado, Dr. Charles Dickie is retiring, effective July 1, 2002. We will miss his excellent service to the area. We have a plan for a new, progressive model to meet the needs of people in the area based on building the collaboration between Colorado State University and the Colorado Department of Agriculture. The new laboratory director will have a joint appointment between these two entities with the primary appointment being with the Colorado Department of Agriculture. The needs of the laboratory work will be met, in addition to increased outreach education and regulatory work, which goes hand-in-hand with the laboratory work. The person will have special training in the recognition of foreign animal diseases. The current laboratory staff will remain, but we will add an additional microbiologist, Jane Carman, who wishes to transfer from Fort Collins to her original home in the Rocky Ford area. She brings with her additional expertise in molecular diagnostics currently not available at Rocky Ford. With the additional technical support and additional equipment already in place (or soon to be transferred), we hope to expand the range of tests provided. In the future, we hope to be able to bring 4th year veterinary students to the laboratory to provide them with a quality food animal and laboratory experience. We are currently advertising for the position of director, and hope to have the position filled soon.

If the director is not a board-certified pathologist, the histopathology support for necropsy, field necropsy, and biopsy cases will be provided by Fort Collins board-certified pathologists. We are dedicated to ensuring no delay in case results if this occurs. Special thanks to the Rocky Ford transition planning committee, chaired by Joel Plath, and consisting of members E. Blackburn, Bill Gray, Marv Hamann, Bill Hancock, Kevin Karney, Dave Kitch, Don Klinkerman, Rick Leone, Dave Mendenhall, Ken Newens, Joe Petramala, Beryl Scherler, John Stulp, and Bob Wiley. This group provided us with recommendations to ensure we meet all the needs of the users of the laboratory. Please take a moment to attend Dr. Dickie’s retirement celebration on Friday, June 28th from 2:00-5:00 at the Rocky Ford Laboratory.
NEW STUDY ANNOUNCEMENT—Melanoma Vaccine Trial

Study Description—This study is designed to determine whether either of two new tumor vaccines can prevent the development of tumor metastases in dogs with melanoma of the oral cavity. Dogs with Stage II or III melanoma of the mouth or lips are eligible for entry into the study, provided there is tumor tissue available for biopsy. Dogs that enter the study will receive a series of 8 vaccinations administered over a 19-week period. This new vaccination protocol has not been found to induce toxicity or unacceptable side-effects in the dogs that have been vaccinated to-date. The study will pay for a portion of the cost of tumor surgery to remove the tumor, as well as follow-up examinations and radiographs for one year. Animals can be enrolled and treated at either of two treatment centers: Animal Cancer Center/CSU (970-221-4535) or Veterinary Cancer Specialists/Denver (303-874-2054).

WHAT’S IN THIS ISSUE

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- Disease Syndrome in Elk with BVD
- Ionophores
- Western Slope Update
- Spleens!
- A Year’s Worth of Mycology
- Ophthalmology Reagents on Feline Herpesvirus-1
- Changes at Rocky Ford
- Admissions to Veterinary School