Message from the Director

Happy New Year!

A common question we are asked is why animals submitted for necropsy cannot be returned to their owners. Per State Agriculture regulation, carcasses of animals cannot be released in order to prevent disease dissemination. There are also public health and safety concerns. If prior arrangements have been made by clients, carcasses can be released to personnel of commercial crematory services with the following provisions: the laboratory has determined that no zoonotic disease health risk exists and, if a rabies test has been ordered, the animal cannot be released until a negative rabies test result is received from the State Laboratory of Public Health. Any requests for individual cremation should be written clearly on the laboratory submission form.

2012 was a bad year for Eastern Equine Encephalitis (EEE) virus infections in our state. Eight horses were diagnosed at NCVDLS with the disease. For post-mortem diagnosis, a polymerase chain reaction assay, histopathology or immunohistochemistry is performed on fresh brain tissue. For ante-mortem diagnosis, samples have been forwarded for testing to the National Veterinary Services laboratory in Ames, Iowa where the IgM-antigen capture enzyme linked immunosorbent assay was performed. We will soon be validating this assay in order to provide this valuable service in-house.

There are plans to conduct another client survey this year so we can better assess your needs. If you receive a survey, please take the time to complete it and return it to us. We are continually striving to make improvements to better serve you and the survey will greatly assist us to do so.

Respectfully,

Karen W. Post DVM, MS
2012 Review of Submissions from NCVDLS

By Dr. Jennifer Haugland

In the year of 2012, 33 samples were submitted to the Pennsylvania Animal Diagnostic Laboratory System for an organic chemical screen. This screen using gas chromatography and mass spectrometry is capable of detecting a large number of compounds including pesticides (avitrol, organophosphates, carbamates, and organochlorines), metaldehyde, a number of therapeutic and illicit drugs, euthanasia agents, and environmental contaminants.

From the 33 submitted liver and stomach contents samples there were 7 different compounds detected.

Carbamate – Aldicarb (2 animals) and Carbofuran (1 animal)
Organophosphate – Terbufos (2 animals), Phorate (1 animal), and Disulfoton (1 animal)
Petroleum metabolite – cattle
Conhydrin, a component of poison hemlock – cattle

In the year of 2012, 28 dogs were diagnosed with Canine Distemper Virus.

In the year of 2012, 7 herds of cattle were diagnosed with Anaplasmosis from October 10 to November 8. A diagnosis of Anaplasmosis was not made during any other time last year. One case came from the Northwestern Laboratory, one case came from the Griffin Laboratory and the remaining 5 cases came from the Rollins Laboratory. September to October is the period of time that Anaplasmosis is typically diagnosed in North Carolina.

Feature Article

Chronic Ulcerative Paradental Stomatitis in a German Shepherd Dog

Dr. Peter Moisan, Rollins Laboratory,
Dr. Eric Dorsch, The Animal Hospital of Carrboro
Dr. Don Hoover, Westside Animal Hospital

Chronic ulcerative paradental stomatitis (CUPS) is a relatively common panstomatitis disease of middle aged to older dogs. As the name implies, it is a “paradental” syndrome that occurs in the soft tissues adjacent to the teeth. It is important to the surgical pathologist inasmuch as the disease is a mimic for the more common, histologically similar lesions of hyperplastic gingivitis and bacterial periodontal diseases of dogs. Chronic ulcerative paradental stomatitis is most likely caused by the antibody response to antigenic stimulation (or overstimulation) to proteins in periodontal disease organisms. The bacteria in these plaques are capable of inciting antigen-antibody reactions under most conditions. If present for a prolonged period of time, it is speculated that they induce a sizeable antibody response.
A large number of bacteria reside normally on the canine tooth surface. Most are resident bacteria that are either non-pathogenic or at best opportunistic pathogens that invade through traumatic insult to the lip or gingiva. In combination, these bacteria form complex colonies. With the production of various adhesion molecules and metabolic by-products, these bacteria are synergistic with each other and the canid (or other toothed animal). In most conditions, these bacteria thrive and are readily replenished in natural fashion after eating, cleaning, or brushing. The balance is changed with disease conditions. Antibodies produced against the bacteria or extrinsic bacterial products are presumed, with CUPS, to be simultaneously developed against the tissue of the individual animal in a sort of aberrant autoimmune reaction. In some individual dogs and perhaps with some breed predisposition (Cocker spaniels, Maltese terriers, racing Greyhounds, for example) the condition of over exuberant antibody production is seen and the antigen-antibody response causes the bystander lesion of gingivostomatitis of a paradental orientation. In manifestations of the disease (Figure 1), there is a mirror image or “kissing lesion” of the tooth surface on the facing lip or gingival mucosa. This is presumably the mirror image of the bacterial plaque of the tooth surface.

In this presented case, a 10-year-old neutered male German Shepherd Dog was presented to the practitioner with severe stomatitis that was most prominent in the lips at contact sites with teeth and gingiva. Antibiotic therapy had been rather unsuccessful, as had repeated teeth cleaning. The classic “kissing lesions” are shown in Figure 1. Biopsy material from the lip lesions was submitted. The ulcers of the affected lip were covered by scant serocellular debris. The superficial submucosa contained lymphoid follicles that were suspended among additional mononuclear cells and smaller numbers of neutrophils. Mild hyperplasia of the gingival epithelium was present in the flanking mucosa (Figures 2&3).

Treatment via extraction of the teeth was elected. Teeth of the right arcades were extracted first (Figure 4), followed up by extraction of the upper and lower left arcades in the following weeks. Recovery following surgery was uneventful. Lesions of the oral mucosa resolved and granulation tissue and normal mucosa covered the extraction sites (Figure 5).

According to the literature provided by dental textbooks, the recovery from the radical tooth extraction procedure is uneventful (as with this patient) and dogs retain the ability to eat a nearly normal diet. In Figure 5, several weeks post-operatively, the patient in this report exhibited clinically normal mucosa of the oral cavity.

The importance of this case is illustrated by the cooperative efforts of clinician, surgeon, and pathologist. Without detailed description and photographs of the gross lesions and surgical repair of the condition, the correct diagnosis would have been impossible. Lesions of hyperplastic gingivitis and periodontal disease in dogs are close mimics for the CUPS condition.

References:
Figures:

Figure 1: Chronic ulcerative paradental stomatitis. The gross lesions of the lips are mirror images of the tooth surfaces or classic “kissing lesions”.

Figure 2: Photomicrograph of the CUPS lesion showing lymphoid follicles.

Figure 3: Higher magnification of the CUPS lesion showing intense inflammatory reaction in the ulcerated submucosa.

Figure 4: The surgical sites of the right upper and lower tooth arcades showing immediate post-surgical appearance.
Canine

Lingual Blastomycosis in a young dog

A section of a mass from the left lateral and dorsal surface of the tongue from a 2 year old male Rottweiler was submitted. History provided stated similar masses have been present on all four paws along the digital and inter-digital areas for months and recently the dog had difficulty eating and chewing found when the tongue mass was identified. No history was provided on other systemic changes associated with *Blastomyces*.

Figure 1: Digital lesion from *Blastomyces*

Photo and some text courtesy of https://www.addl.purdue.edu/newsletters/2006/spring/blastomyces_dermatitidis.htm
Histopathology revealed diffuse mucosal ulceration and effacement of the submucosa and underlying skeletal muscle by multifocal and coalescing pyogranulomas characterized by large aggregates of epithelioid macrophages with central areas of degenerate neutrophils that were themselves surrounded by small infiltrates of lymphocytes and plasma cells. Within the center of the granulomas are rare, 7 to 15 µm, round yeast that have a round to oval 5-10 µm basophilic nucleus separated from a 1-2 µm cell wall by a clear halo. There is rare broad based budding identified. Abundant granulation tissue is composed of fibroblasts and small, well differentiated blood vessels. Infiltrates extended to all margins.

Figure 2: Blastomyces yeast within pyogranulomatous inflammation

Fungal infections including Blastomyces dermatitidis, Histoplasma capsulatum, Cryptococcus neoformans and Coccidioides immitis are significant causes of systemic disease because they can gain entry through a single portal and then disseminate to multiple organ systems. Blastomyces is commonly seen in the Mississippi, Missouri, and Ohio River valleys, however, sporadic cases have been seen along the east coast. Blastomycosis is often spread from inhalation of spores and systemic spread via respiratory tract. Following inhalation, fungal spores are transformed from the mycelial to the yeast phase at normal body temperatures. Dogs and cats can both be affected, but blastomycosis is much more common in dogs. Dogs of any breed or age could become infected if they are in an area with the right environmental conditions for the fungal spores to develop (generally moist, acidic soil rich in decaying vegetation). Most infected dogs are young adults that are often in sporting breeds. Clinical signs of systemic spread include weight loss, anorexia, dyspnea, cough, and draining cutaneous lesions.

Diagnosis of blastomycosis is by identifying yeasts organisms via impression smears and aspiration for cytology as well as histopathology on biopsy sections. Characteristic features of intrallesional Blastomyces yeast forms include a thick basophilic cell wall, broad-based budding, are 5-20µm in size, and extracellular location. Cytologic analysis will also provide the type of inflammatory response present, which ranges from pyogranulomatous to granulomatous and can have multinucleated giant cells present. Aspirates of infected lymph nodes, or even exudates or aspirates from the dermal lesions, can contain the fungal organisms. Other common diagnostic procedures performed in dogs with severe respiratory signs include lung aspirate, transtracheal wash, or bronchoalveolar lavage to help find organisms. Blastomycosis can also be diagnosed through histopathology of tissue samples. The digital regions of the dog are fairly common in the cutaneous form and involvement of the oral cavity is quite rare.
“Barrell rolling” Dachshund

A 4 year old spayed female Dachshund was suddenly ataxic with head tilt. The next day the ataxia was so severe that when she would try to move she would turn her head to the side, fall, and then barrel roll across the room. Continuous nystagmus also developed. The dog died after 4 days of clinical signs and was submitted for necropsy.

Necropsy examination revealed a dura mater and meninges (figure 1) that were diffusely dark red. Cut sections of the brain revealed an 8x8 mm gray focus in the mid and right cerebellum (figure 2). There was another smaller but similar gray focus in the left caudodorsal cerebrum.

No bacteria were isolated on routine aerobic culture of the brain. A fungus identified as *Cladophialophora bantiana* was isolated from the brain lesion. Histopathology confirmed multifocal to coalescing granulomatous and necrotizing encephalomyelitis with intrallesional pigmented fungal hyphae, which were consistent with the isolated fungus.

**Diagnosis:** *Cerebral phaeohyphomycosis*

*C. bantiana* is considered as a neurotropic species which causes cerebral phaeohyphomycosis. The fungus is found in the soil and has a worldwide distribution. It is probably introduced by inhalation. This disease has been reported in dogs, cats, and humans and is usually fatal even with antifungal treatments.
Feline

A 5-year-old male neutered DSH feline was submitted for necropsy with no history provided. Exploration into the nasal cavity revealed bilateral hemorrhage of the turbinate bones. The thoracic cavity contained 50 ml of serosanguinous to hemorrhagic fluid admixed with large chunks of fibrin. Abundant fibrin was adhered to the pulmonary, costal and diaphragmatic pleura. The left caudal lung lobe was approximately 95% effaced with purulent material. The remaining lung lobes were dark red and rubbery. Histopathologic examination revealed (1) chronic active fibrinosuppurative vegetative valvular endocarditis with focal suppurative myocarditis, (2) suppurative bronchopneumonia with bacteria, interstitial pneumonia, and pleuritis, and (3) chronic diffuse lymphoplasmacytic rhinitis. Histologic lesions supported a bacterial etiology as confirmed by isolation of *Streptococcus canis* and *Pasteurella* sp. in the lung and nasal turbinates. Herpesvirus was not detected in the lung via fluorescent antibody testing.

Dr. Mahogany Caesar

A 12 year old female domestic medium haired cat was presented to the laboratory for post mortem examination. The cat was initially taken to the veterinarian for frequent urination and diarrhea. A urinary tract infection was diagnosed and treated. A couple weeks later, the cat presented back to the veterinarian with a broken canine tooth. Pre-anesthetic blood work was normal. The cat went into cardiac arrest during anesthetic induction and failed to respond to cardiopulmonary resuscitation.

On post mortem examination the cat weighed 7.5 kg, had a BCS of 4+/5 and was of normal hydration. There was minimal post mortem change to the body. The heart weighed 22.59 grams. The lungs were dark red in color, spongy and wet. There were multifocal to coalescing 1-3 mm white foci present in all liver lobes. The urine tested positive for > 100 mg/dl glucose using dip stick testing. The urine was clear and yellow in color. There was segmental placation of the small intestinal wall. The large intestinal wall was 2-3 times the usual thickness with a diffuse distribution of raised “cobblestone” mucosal lesions. (Figure 1) The colon contained liquid content with gross hemorrhage present. There was a 1 cm by 3 cm cystic mass present in the mesenteric tissue adjacent to the colon. The thyroid glands were prominent (~ 50% enlargement) with small cysts in the right gland. The left lower canine tooth was very loose. There were no additional lesions on examination of the body.

![Figure 1: Gross photograph of colonic mucosa](image_url)
On histopathologic examination of various tissues of the body, the following lesions were identified:

Heart: Multifocal myofiber degeneration with interstitial fibrosis and multifocal acute epicardial petechiation
Lung: Marked multifocal pulmonary artery thrombosis and multifocal metastatic adenocarcinoma
Liver: Acute centrilobular hepatocellular degeneration and moderate diffuse fatty change
Small intestine: Lymphoma and multifocal lymphoplasmacytic and eosinophilic enteritis
Colon: Adenocarcinoma with multifocal vascular invasion
Mesentery: Multifocal metastatic adenocarcinoma
Thyroid gland: Follicular cell hyperplasia (adenomatous hyperplasia)
Pancreas and peripancreatic vessels: Metastatic adenocarcinoma

The following disease conditions were diagnosed in this cat:

**Hypertrophic cardiomyopathy**
**Pulmonary thrombosis**
**Hepatic hypoxia**
**Intestinal lymphoma**
**Colonic adenocarcinoma with wide spread metastasis**
**Thyroid hyperplasia**

I have included this case as an example of how well cats can be at compensating subclinical disease. It also shows how much underlying disease can be found in some of the elderly pets we examine.

Dr. David Drum

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**Exotics**

Torticollis and rolling were identified in an adult rat. The animal failed to respond to antibiotic therapy and died. The entire rat was submitted in formalin solution. Histologic examination revealed diffuse severe **suppurative meningitis** with cocci. Since fresh tissues were not submitted by the practitioner, bacterial cultures were not performed. A differential for the bacterial meningitis is *Streptococcus pneumoniae*, which has been reported to cause pneumonia, pleuritis, pericarditis, peritonitis, otitis media and interna, metritis, orchitis and meningitis in rats.

Dr. Mahogany Caesar

On 11/20/2012 two < 12 year old Goldfish were presented to the laboratory for post mortem examination. The fish had tumor like growths on their bodies. The owner reported that they had an initial fish develop similar growths and it died three months ago.

On post mortem examination the first fish had a tumor on the left side where the dorsal fin starts (figure 1). The second fish had a tumor on the upper surface of the eye/cornea (figure 2), another at the base of the tail and another on the right side where the dorsal fin starts (figure 3). Both fish seemed for be of normal weight with internal fat evident.
The following lesions were identified upon histopathologic examination:

**Skin:** Dermal peripheral nerve sheath tumor,

**Eye:** Dermal peripheral nerve sheath tumor with periocular infiltration,

**Liver:** Diffuse hepatocellular fatty change

The tumors were identified as being **Peripheral Nerve Sheath Tumors.**

Peripheral nerve sheath tumors (PNSTs) are neoplasms that originate from neural crest cells and generally develop along the subcutaneous nerves. In fish and other vertebrates, benign PNSTs have been classified as schwannomas or neurofibromas, which can be difficult to distinguish from one another in standard histologic sections. Both tumor types usually appear as masses on the head, skin, or fins. The chief cellular component of neurofibromas and schwannomas is the Schwann cell, which is a variety of glial cell that provides myelin insulation to axons in the peripheral nervous system of jawed vertebrates. Other differential diagnoses for these type of tumors include: pigment cell tumors (eg, erythrophoromas, xanthophoromas, melanomas, and guanophoromas) that also originate from neural crest cells, and neoplasms derived from collagen-producing cells (ie, fibromas and fibrosarcomas). Schwannomas have been detected in many fish species, including goldfish, several species of snapper, coho salmon, the bicolor damselfish, and rainbow smelt. Schwannomas are usually considered benign, but they can also be locally invasive and progressive.

Based upon the fact that multiple fish have been reportedly affected in some populations, a viral cause of fish PNSTs has been proposed. However, the bicolor damselfish is the only species for which a virus-like agent has been definitively associated with a type of PNST (neurofibroma).

Peripheral nerve sheath tumors can be removed, either by excision or cryosurgery. However, hemorrhage is more likely with excision than with cryosurgery. Recurrence is more likely after excision than after cryosurgery because freezing allows for more complete removal of tumor cells.

Reference:


Dr. David Drum
**LIVESTOCK**

**Bovine**

A 1-day-old male Angus calf was found dead. A 2 cm **ventricular septal defect (VSD)** was identified in the perimembranous portion of the septum immediately below the aortic and tricuspid valves on gross examination. Ventricular septal defect is a congenital defect; the etiology is not completely known in cattle. Search of the veterinary literature (Can Vet J. 2006 March; 47(3): 246–252. Ventricular septal defects in cattle: a retrospective study of 25 cases) revealed that the defect is suspected to be due to hereditary transmission in Hereford and Limousin cattle. Genetic predisposition is reported in Jersey cattle. In other dairy breeds, information is not available on the possible genetic implications; although, VSD is reported in twin Holstein heifers.

Dr. Mahogany Caesar

**Equine**

An unvaccinated 6-year-old gelding mule developed neurologic signs, characterized by periodic seizures, mild hyperesthesia and hyperreactivity, and ataxia with progression to recumbency within an 18 hour period. Treatments included nonsteroidal anti-inflammatory drugs, Gentamicin and Penicillin. West Nile virus and Eastern equine encephalitis (EEE) were suspected. The brain tested negative for Rabies virus and EEE. The cerebrum, cerebellum and brainstem were positive for **West Nile virus** via PCR.

Dr. Mahogany Caesar

**Fetal Loss associated with Placental/Umbilical Cord Abnormality**

During December 2012 an American Saddlebred mare with a mid April 2013 due date aborted. She had been given an EHV 1 (Pneumabort-K) booster 7 days prior. This mare had 5 live foals in succession until March 29, 2012 when she passed a 10 month gestational age stillborn foal. (In this case the laboratory findings determined a premature, stillborn foal with some indications of birth trauma and not of infectious agent etiology). In both instances she was bred to the same stallion, and had not been bred to this stallion in the preceding pregnancies. There had been no recent management – including dietary – changes on this farm which had 15 other brood mares on the premises with none showing an indication of illness at the time of laboratory submission.

The submitted specimen consisted of fetal membranes and an equine fetus with a combined weight of 13.2 kg. The fetus was a 12.2 kg., 71.5 cm crown-rump length male equine fetus (consistent with ~ 220 days gestational age) having moderate autolytic change. The amniotic portion of the umbilical cord was twisted tightly and swollen as a result of being compressed by the entwined peduncle of a 10 X 9 X 6 cm round, red, fluid filled mass causing vascular stasis and urachal compression. (see Figures 1 and 2).
Histological evaluation:
Sections of equine fetal brain, heart, lung, spleen, liver, adrenal gland, kidney, thymus, thyroid gland, umbilicus, yolk sac, and placenta were examined.

Morphological diagnosis: 1. Umbilicus; vascular congestion, marked, diffuse, subacute. 2. Lung; alveolar squames and meconium, multifocal, moderate.

The fetal loss was attributed to umbilical cord compression (via pedunculated yolk sac) with fetal hypoxia / asphyxiation.

Comment: The yolk sac is typically displaced, to a large degree, by the allantoic sac by day 36 of pregnancy. In rare cases a remnant, sometimes pedunculated, may persist.

Camelids

An 11 year old 68kg female alpaca was submitted with a history of lethargy and weakness. She was down on her side and was treated with IV fluids and Vitamin B. She had delivered a premature cria 10 days earlier. She did not respond to supportive treatment and was euthanized 3 days later. Vaccinations were current and she was recently dewormed.

On necropsy, approximately 500mls of pale straw pleural effusion was noted. Numerous small 1cm diameter tan nodules were adhered to the pleura and pericardium. The nodules contained thick tan material. The lungs were pink to red and congested with foamy fluid. An irregular shaped approximately tennis ball size mass that was filled with caseous exudate was noted along the left lung lobes at the base of the heart (figure 1). The tracheal lymph nodes were enlarged and numerous tan .5cm diameter nodules were noted along the vena cava and aorta. A .5cm diameter pale tan irregular shaped foci was noted in the left ventricle.
The abdominal organs and brain were unremarkable. No other abnormalities were identified on gross exam. Histopathologic findings included: Granulomatous encephalitis with intracellular yeasts, multifocal granulomas with intracellular yeasts in the lungs, granulomatous myocarditis with intracellular yeasts, multifocal granulomatous myositis and pleuritis with intracellular yeasts in the diaphragm and pleura, a focal hepatic granuloma and a focal cortical interstitial granuloma with intracellular yeasts in the kidney. *Blastomyces dermatitidis* was isolated in culture from the thoracic mass.

**Blastomycosis** is an uncommon fungal infection in alpacas and is a fungal infection that is a threat to public health. The fungus is associated with decomposing organic matter and moist soil. Pulmonary infection occurs after inhalation of fungal spores. Flu like symptoms occur in people 3-15 weeks after exposure and infection can disseminate throughout the body.

Additional information on this infection is available at: http://www.cdc.gov/fungal/blastomycosis/

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**Poultry**

Small poultry flocks, mostly chickens, also known as backyard birds, which I like to call pet and breakfast chickens, are increasingly seen in the diagnostic laboratories in our state. One of the more common disease diagnosis found in these small backyard flocks is a type of infectious bacterial disease caused by *Mycoplasma gallisepticum*, also known as MG and Chronic Respiratory Disease. Common complaints by the owners of small flocks infected with MG are that the birds stop eating well, have some discharge from the nostrils, maybe some swelling around the eyes with sneezing or coughing observed. Sometimes the primary complaint is the birds just do not eat well, get thin and eventually die, with little other indication or symptom of disease.
Note the pictures below of 2 recent cases of MG infection, the first bird has severe lesions, with sinus swelling and discharge and the next one is of a chicken that is thin and just does not seem to feel well. (see Figures 1 and 2).

Figure 1: Severe sinus swelling.  
Figure 2: Thin bird.

Another type of *Mycoplasma* infection is caused by *Mycoplasma synoviae*, also called MS, tends to cause milder respiratory infection and sometimes joint infection, and is also of concern and found in small flocks.

Other species of birds found in small flocks including turkeys are also susceptible to these infections.

The North Carolina Veterinary Laboratory System is an important resource for small flock owners in the state seeking to diagnose and improve the health of their birds. Other valuable resources for small flock owners are the Poultry Section in the North Carolina Animal Health Programs, the NC State University Cooperative Extension Service and the North Carolina College of Veterinary Medicine poultry disease diagnostic group.

MG and MS diseases are reportable to state authorities in North Carolina due to the potential for spread of the infection to other flocks, both commercial as well as to non-commercial small flocks. Recently the quarantine requirements for these infections have been changed generally making it easier for small flock owners to comply.

Knowledge of what diseases may be present is very beneficial to the growing numbers of farms and households with small flocks. The maximum standard necropsy charge for poultry including small flocks is $30.00 and includes examining up to 8 birds. Necropsies may provide information about overall flock health in addition to awareness of infectious diseases such as MG and MS. Healthy birds are the goal and the laboratory system and the poultry agencies are available to assist owners with information about how to select and maintain healthy birds.

Dr. Reg Ridenhour
**Rollins Laboratory**

**Rollins Lab New Hires**
John Dockery, Veterinary Laboratory Assistant I, November, 2012  
Diane Kuo, Medical Laboratory Technologist II, December, 2012  
Rebecca Boone, Medical Laboratory Assistant II, December, 2012

**Rollins Lab Resignations**
Philip Renshaw, Medical Laboratory Assistant II, August, 2012  
Joelle Fernandez, Medical Laboratory Technologist II, August, 2012  
Krystal Plemmons, Medical Laboratory Technician II, January, 2013

**Rollins Lab Retirements**
Kathy Schmidt, Safety Officer, August, 2012  
Bing Tang, Medical Laboratory Technologist II, December, 2012

**CE Attendance**

Drs Karen Post, Richard Mock, Alison Tucker, David Drum, Richard Oliver, Stacy Robinson, Steve Rushton, Jennifer Haugland, Mahogany Caesar, and Reg Ridenhour attended the 55th Annual Conference of the American Association of Veterinary Laboratory Diagnosticians held October 20-21, 2012 in Greensboro, NC.

Drs Kim Hagans, Mahogany Caesar, David Drum, Peter Moisan, Alison Tucker, and Jennifer Haugland completed the Iowa State University, College of Veterinary Medicine’s online “Emerging and Exotic Diseases of Animals” course, which was held in conjunction with the Center for Food Security and Public Health over summer of 2012.


Dr Richard Oliver attended the Merial Rabies Symposium in Raleigh, NC on September 29, 2012, and “Bugs vs Drugs: A One Medicine Approach to Antimicrobial Resistance” in Durham, NC on December 5-6, 2012.

Dr David Drum attended a meeting on Veterinary Biomedical and Diagnostic Sciences at the University of Tennessee, College of Veterinary Medicine in Knoxville, TN on December 6, 2012.

Drs Jennifer Haugland and Stacy Robinson attended the North Carolina Veterinary Conference held November 2-4, 2012 in Raleigh, NC.
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Dr. Karen Post
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