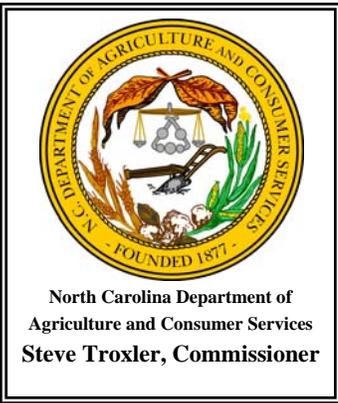


# The NCVDLS REPORT



## Veterinary News and Information From North Carolina's Diagnostic Laboratories



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### Holiday Closings...

- January 17, 2011
- April 22, 2011
- May 30, 2011

Our laboratories will be closed on the above listed days.

Please e-mail [NCVDL@ncmail.net](mailto:NCVDL@ncmail.net) with any comments and/or suggestions concerning The NCVDLS Report

Editor - [Dr. Richard Oliver](mailto:Dr. Richard Oliver)

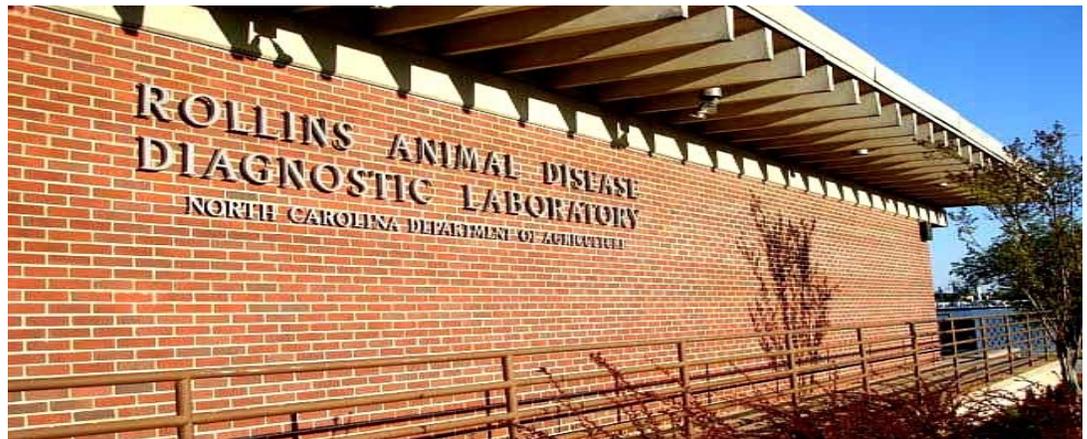
## Message from the Director

On August 19, 2010 the Rollins Animal Disease Diagnostic Laboratory hosted a daylong tabletop exercise for Foot and Mouth Disease (FMD) which was coordinated by the USDA's National Animal Health Laboratory Network (NAHLN). Participants included Rollins Laboratory personnel, State Animal Health Officials and Field Forces staff, North Carolina Department of Agriculture & Consumer Services Emergency Programs Division staff, USDA officials, and representatives from NAHLN, State Emergency Management, the NC Wildlife Resources Commission, livestock industry stakeholders and the Virginia Veterinary Diagnostic Laboratory System. The exercise was designed to move the attendees through the many challenges encountered during a simulated field outbreak of FMD in North Carolina and was specifically focused on laboratory response. All participants gained tremendous insight as to the laboratory challenges associated with responding to a foreign animal disease outbreak. The exercise afforded the attendees an opportunity to assess the completeness of their emergency response plans. As a positive outcome, multiple strengths were identified as well as opportunities for improvement related to specimen transport, sample accessioning and prioritization, high volume testing and result reporting.

The exercise ensured the readiness and cooperation between livestock industries, laboratory personnel, and State and Federal regulatory partners.



In September, the NCVDLS Hoyle C. Griffin facility was officially recognized as a STAR participant site. The Carolina Star Programs are administered by the NC Department of Labor and are designed to recognize and promote effective safety and health management. In STAR, management, labor, and OSH establish cooperative relationships at a workplace that has implemented a strong safety



## Message from the Director, *continued*

program. Goals of the program are to provide a safer work environment, improve employee motivation to work safely, and reduce worker's compensation costs.

As a convenience to our clients, the Rollins Laboratory now offers the Global Vet Link service for online submittals of Equine Infectious Anemia (EIA) tests. This web-based certification system utilizes digital photos and eSignatures and integrates diagnostic laboratory data directly with private veterinarians and animal health officials. Global Vet Link is approved by USDA-APHIS-VS for international movement of horses (See Client Corner for more details)

As a result of the state budget that was approved by the General Assembly in late June, funding was eliminated for the Rose Hill Animal Disease Diagnostic Laboratory in Duplin County with closure of the facility on Friday, July 16. The lab conducted necropsies and poultry serological testing. In a press release from the Department of Agriculture, State Veterinarian Dr. David Marshall said, "We regret that the Rose Hill lab is closing, but continued budget reductions forced agencies and legislators to make some very difficult decisions. Trying to keep five labs properly maintained and equipped became too costly." In closing the Rose Hill Lab, seven positions were eliminated resulting in approximately \$432,000 of annual savings to the State. The seven displaced employees have priority status in applying for other state jobs for which they are qualified. Diagnostic tests that were performed at Rose Hill were absorbed by the Rollins Laboratory in Raleigh. NCVDLs worked closely with poultry companies during the transitional phase to ensure continuity of serological services. To date, lab closure has not affected the State's ability to conduct mandated regulatory disease testing.

*Karen W. Post DVM, MS*

## Client Corner

GlobalVetLink service available at Rollins Laboratory

As a service to our clients, the Rollins Laboratory is now connected to GlobalVetLink for Equine Infectious Anemia Virus (EIA) (Coggins test: AGID and ELISA) test requesting and reporting. The GlobalVetLink system provides for electronic reporting of EIA test results immediately to the submitting Veterinarian when reported by the laboratory. The reported results is recognized by all 50 states, and accepted by USDA for export as official documentation of a negative EIA test result. GlobalVetLink subscribing practices may allow their clients secure read-only access to their EIA forms as soon as tests are reported.

For more information on the use of the GlobalVetLink system for EIA testing and reporting call 515-296-0861 (new accounts) or email [info@globalvetlink.com](mailto:info@globalvetlink.com). The website is [www.globalvetlink.com](http://www.globalvetlink.com).

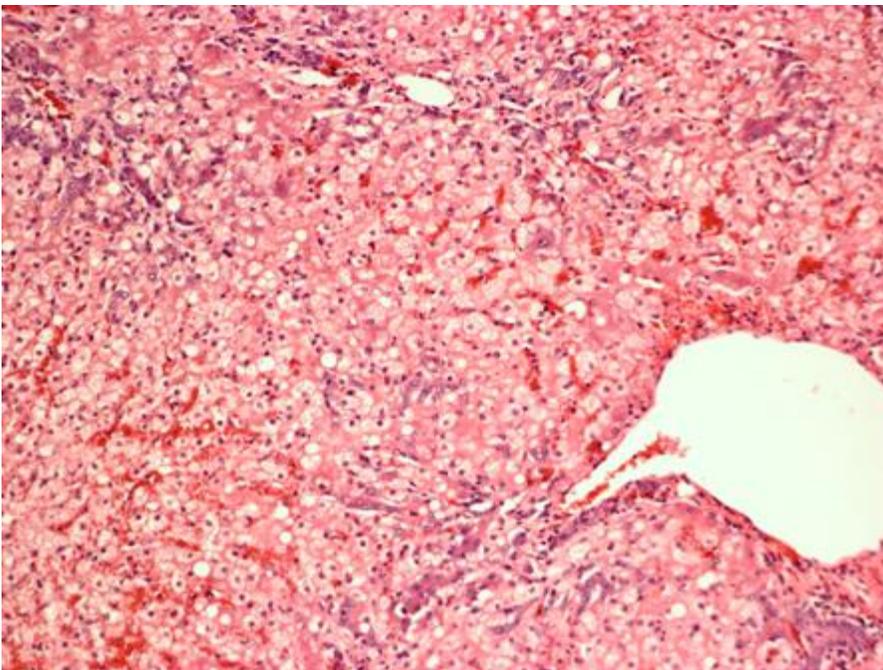
For additional information on EIA testing or fees at Rollins ADDL please call the laboratory at 919-733-3986 or visit the website at [www.ncagr.gov/vet/ncvdl/](http://www.ncagr.gov/vet/ncvdl/).

## NEWS RELEASE

**Liver failure and Aflatoxicosis associated with dog food**

A two year old Cocker Spaniel from a breeding operation was submitted to the Rollins Animal Disease Diagnostic Laboratory in Raleigh for necropsy examination in November 2010. Clinical signs included anorexia and lethargy with clinical icterus. The dog is the fifth dog to die in a similar manner in the last month.

Necropsy examination of the dog revealed generalized icterus with hepatomegaly and swollen edges to the liver lobes. Hemorrhage was identified in the intestinal and uterine walls. Histopathology of numerous organs was performed. The liver showed marked hepatocellular degeneration and loss within the centrilobular and midzonal areas. There was also abundant bile accumulations within the bile canaliculi.



The histological changes identified in the liver of the dog are consistent with intoxication of Aflatoxin, which is a toxin produced by certain fungi that grow mainly on corn.

A sample of "Old Yeller" brand dry dog food was presented along with the first dog. The sample of dog food along with sections of liver and kidney were submitted to the Pennsylvania Animal Disease Diagnostic Laboratory toxicology section for Aflatoxin analysis. The toxicology laboratory found 47.9 ppb of Aflatoxin in the feed which is considered in the toxic range.

This result is likely the cause of the recent recall of dog and cat food from a major grocery store chain over a dozen states.

## Feature Article

### Equine Protozoal Myeloencephalitis

Alison Tucker, M.A., V.M.D., Diplomate American College of Veterinary Pathologists  
Rollins Diagnostic Laboratory  
Raleigh, North Carolina

A seventeen year old Standardbred gelding was presented for necropsy at the Griffin Laboratory in July 2010 following a 5 month history of progressive weakness and difficulty walking. The horse had been boarded at a large facility where one other horse had been reported to have similar symptoms. Vaccinations were current and appetite was not affected. The rear legs were affected first, with weakness, occasional cross stepping, and difficulty rising. Ultimately the front legs were affected and the horse also began to walk in circles. At this point, given the lack of response to treatment, the horse was humanely euthanized and was presented for necropsy.

The horse was in good body condition and significant gross lesions correlating to the symptoms were not noted. Cerebrospinal fluid was clear. As part of the necropsy, histologic examination of sections of brain and spinal cord was performed.

Changes were identified throughout the spinal cord but were most marked in sections of lumbar cord. Here, there was lymphohistiocytic, neutrophilic and eosinophilic meningomyelitis with necrosis, marked Wallerian degeneration, and intralosomal protozoal schizonts. The diagnosis was Equine Protozoal Myeloencephalitis (EPM).

EPM is caused by protozoa invading, and gradually damaging, the central nervous system of the horse. *Sarcocystis neurona* has been identified as the primary causative organism. Another, very rare, protozoal organism that causes similar disease in the horse is *Neospora hughesi*, but little is known about the life cycle of this organism. Opossums are the definitive host for *Sarcocystis neurona*, shedding infectious sporocysts into the environment. Intermediate hosts ingest the sporocysts which ultimately form cysts within muscle (sarcocysts) and in other tissues. The sarcocyst is the form that is infectious for opossums. Aberrant intermediate hosts, such as the horse, do not form sarcocysts, thus, infected horses do not serve as a source of infection for opossums and are not infectious to other horses.

Serum samples can be tested for antibodies to *Sarcocystis neurona*. Exposure and seroconversion are moderately common in North Carolina, but only a small percentage of horses exposed to the organism develop EPM. Cases are most frequently seen in spring and summer. Clinical signs include weakness, ataxia, lameness and occasionally seizures. Western blot testing on serum and cerebrospinal fluid for *Sarcocystis neurona*-specific IgG can determine if the organism has crossed the blood-brain barrier but sampling cerebrospinal fluid is not without risk and is not commonly performed. Other tests are in development but at this point treatment of horses with both clinical signs and prior exposure is widely recommended. Please contact your veterinarian for further information on treatment options for EPM.

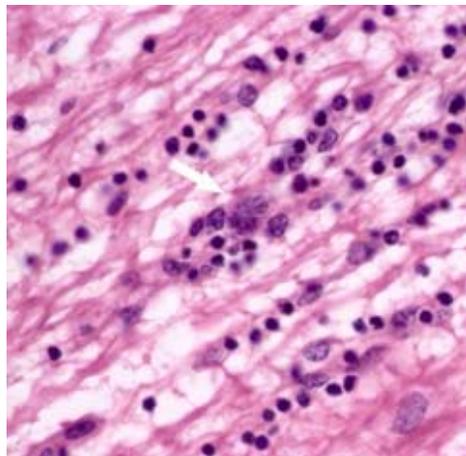


Figure: Photomicrograph of the lumbar spinal cord of a horse demonstrating lymphohistiocytic myelitis, Wallerian degeneration, and a protozoal schizont (arrow). H & E 400 X.

Thanks extended to Dr. Tahseen Aziz for photomicrograph expertise.

## Short Cuts

## COMPANION ANIMAL

## Canine

A 9 year old male neutered Labrador Retriever dog was found dead one morning with no prior clinical signs. The dog was presented for necropsy at the Rollins Laboratory. On **gross examination** the dog was in slightly obese body condition and normal hydration status. The mucous membranes were white to pale pink. The abdomen contained approximately 350 to 400 ml of blood admixed with large blood clots. The liver was diffusely pale mahogany to slightly yellow. The gall bladder contained approximately 5 ml of slightly inspissated bile. Cranial to the left kidney there were two approximately 3 cm diameter dark red soft masses. The more ventral of the two masses was irregular and reddened on the surface as it had ruptured. A small portion of the pancreas and omentum were adhered to that region of the mass. Attached to the soft masses but cranial and dorsal to them was a firm, elongated, multilobulated mass that was 5 cm x 2 cm. Extending from the masses that were cranial to the kidney into the caudal vena cava was a soft pink tubular mass that was approximately 4 cm long x 2 cm in diameter. The tubular mass extended to the point that the caudal vena cava exits the liver. Multifocal white nodules were adhered to the skeletal muscle and fascia in the left paralumbar area cranial to the left kidney. **Histopathology findings** included: Left adrenal gland; Pheochromocytoma with necrosis and hemorrhage. Vena Cava: Intraluminal pheochromocytoma. **Diagnosis:** Pheochromocytoma with tumor necrosis, subsequent tumor rupture and exsanguination. Pheochromocytoma is a tumor of the medulla of the adrenal gland. This type of neoplasia occurs most often in dogs and cattle. Dogs with a pheochromocytoma may be asymptomatic or may have nonspecific, intermittent and vague clinical signs. Usually dogs are 6 years or older when they are diagnosed with this type of tumor. We periodically see dogs with this tumor on the necropsy floor. They often invade the caudal vena cava as in this dog. These tumors can be functional, producing catecholamines which can result in cardiac arrhythmias, and paroxysmal hypertension. This case was a little unusual from what we typically see in dogs with this tumor as this dog died secondary to the tumor necrosis, rupture of the tumor and subsequent exsanguination. Most of the dogs seen here with this tumor have died secondary to the hypertension and its affects on the heart and thrombosis of blood vessels.

Capen, CC Tumors of the Endocrine Glands. In: Tumors in Domestic Animals 4<sup>th</sup> edition, Meuten, DJ, pp 632-636.

Dr. Stacy Robinson

Intestinal Schistosomiasis in a Dog (case of *Heterobilharzia americana*)

This case is from an 8 yr old mix breed female dog presented to the veterinarian for ovariohysterectomy. During the procedure numerous small, tan nodules located within the small intestinal serosa. Multiple sections were submitted for histopathology.

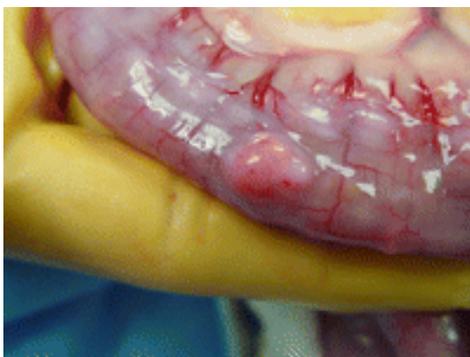


Figure 1 : Nodules located on intestinal serosa of dog

## COMPANION ANIMAL, CONTINUED

Expanding the intestinal serosa are coalescing nodules (eosinophilic granulomas) composed of extracellular trematode eggs surrounded by concentric layers of numerous multinucleated giant cells and epithelioid macrophages. These infiltrates are further surrounded by numerous eosinophils, fewer lymphocytes and plasma cells, and occasional layers of plump fibroblasts. Eggs have a yellow-brown, 2 um thick refractile shell, prominent lateral spines, and a central 30-40 um-wide, irregular eosinophilic structure with numerous 3 um-diameter basophilic round structures (schistosome eggs with miracidia). Multifocally, giant cells contain small amounts of granular basophilic material (mineralized debris).

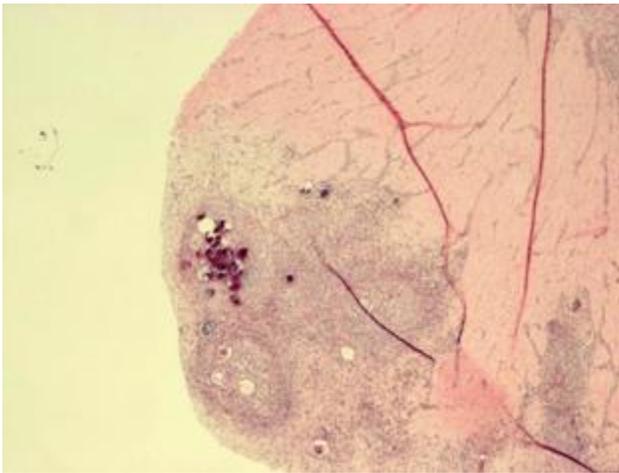


Photo 1: Nodule of inflammatory cells and organisms within the muscular wall of intestine

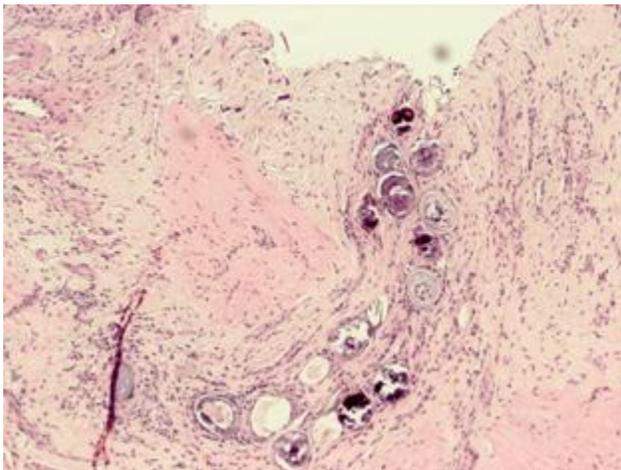


Photo 2: Aggregate of trematode eggs and inflammatory cells in intestine

## COMPANION ANIMAL, CONTINUED

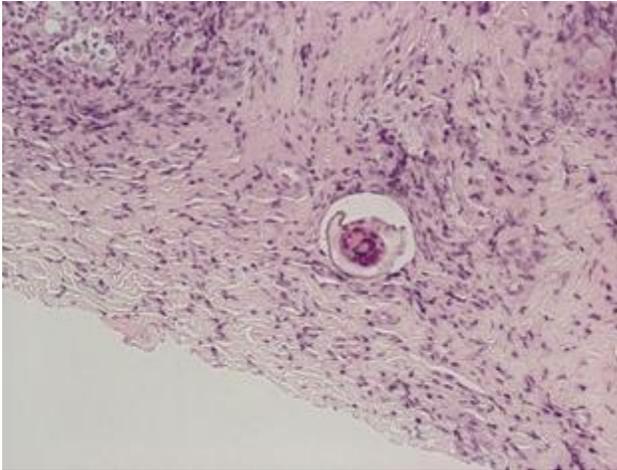


Photo 3: Single developing egg (miracidium) in intestine wall

Dr. Steve Rushton

## Feline

### Rhinosporidiosis in a Cat

A 2 mm x 4 mm polypoid mass was removed from the nasal mucosa of an otherwise healthy, 3 year old, neutered female domestic shorthair cat and was submitted to the NCVDLs biopsy service. In the tissue examined, the epithelium was mildly and irregularly hyperplastic and there were infiltrates of lymphocytes, plasma cells, macrophages with few neutrophils throughout the submucosa. Within the inflamed submucosa were numerous spherical organisms measuring up to 0.4 mm diameter. The smaller organisms had hyaline borders surrounding sparse and faintly granular cytoplasm and a single small nucleus. Larger mature sporangia were thin-walled and contained myriad endospores measuring up to 5  $\mu$ m diameter.

The morphologic diagnosis was hyperplastic and granulomatous rhinitis with intralesional organisms most consistent with *Rhinosporidium seeberi*.

*Rhinosporidium seeberi* is a protist organism that is a member of the class Mesomycetozoa which primarily infects fish and amphibians. Of this class, *Rhinosporidium* causes disease in humans, dogs, horses, cattle and, rarely, in cats. Although long known as a cause of disease, little is known about the organism because in vitro culture has not been possible. The findings in this case are consistent with those typically seen, with a polypoid proliferation of nasal mucosa containing organisms. Unilateral or bilateral presentation has been described. Rarely, the nasopharynx may be involved. The mode of transmission is not completely understood but the organism is thought to be acquired from an aquatic environment and spread between animals, or between humans and animals, has not been documented.

Surgical excision is the treatment of choice. Complete excision of the nasal polyp is generally curative with a risk of recurrence associated with incomplete excision or autoinoculation due to release of endospores at surgery.

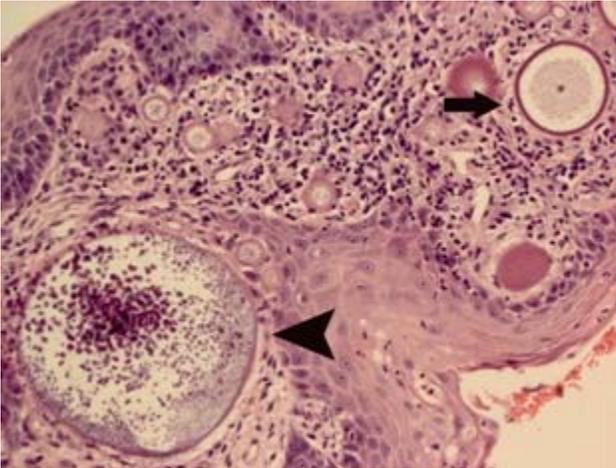


Figure: Photomicrograph of nasal mucosal polyp containing mature (arrowhead) and juvenile (arrow) sporangia, H & E 200 X

References:

Gail Leeming, Ken C. Smith, Mark E. Bestbier, Annalisa Barrelet, and Anja Kipar, Equine Rhinosporidiosis in United Kingdom, *Emerging Infectious Diseases* • [www.cdc.gov/eid](http://www.cdc.gov/eid) • Vol. 13, No. 9, September 2007.

Peter Moisan and Stephanie Baker, Rhinosporidiosis in a cat, *JVDI*, Vol. 13, pp. 352-354, 2001.

Thanks extended to Dr. Tahseen Aziz for photomicrograph expertise.

Dr. Alison Tucker

An intact female DSH feline that displayed neurologic signs (comatose and stupor) failed to respond to medical therapy and was euthanized. Necropsy examination revealed a body condition score of 1.5/5, moderate dehydration, few ticks and moderate flea dirt along the caudal thighs, tail base and caudal dorsum. A 0.5 mm petechial hemorrhage was located in the left brainstem. Histopathology indicated a focally extensive suppurative encephalitis and polioencephalomalacia of the brainstem with hemorrhage, necrosis and axonal (Wallerian) degeneration. The brain tested negative for rabies. The histologic lesions are consistent with feline ischemic encephalopathy, which sporadically occurs in cats. The cause of this condition is unknown but is speculated to be a result of aberrant *Cuterebra* sp. migration. Clinical findings depend on the degree and location of infarction; clinical signs may include depression with mild ataxia, behavioral changes, seizures, blindness, or propulsive pacing and circling.

Dr. Mahogany Wade-Cesar

An adult female spayed DSH barn cat became ill, developed seizures and was presented to the veterinarian in a semi-comatose state. The pet was hypothermic and bloodwork revealed severe azotemia and hyperphosphatemia. The pet died before treatment could be initiated. On necropsy, the feline was in thin body condition (1.5/5) and severe dehydration. Bilaterally, the renal cortices were expanded by large multifocal to coalescing cysts (up to 1 cm) filled with transparent yellow fluid. Severe bilateral renomegaly was identified and the cranial to caudal pole of each kidney measured 6 cm. Gross and histologic examination was consistent with **polycystic kidney disease**, which resulted in chronic renal failure. Polycystic Kidney Disease (PKD) is a progressive, inherited condition which causes multiple fluid filled cysts on the kidneys, most commonly in Persians/Exotic cats & breeds with Persians/Exotics in their lines. The cysts are present from birth and slowly increase in size over the years. Clinical signs include anorexia, vomiting, lethargy, depression, polyuria/polydipsia, and/or weight loss. Enlarged cystic filled kidneys are identified via abdominal ultrasound. While there is no cure for PKD, standard chronic renal failure therapy may be administered.

Dr. Mahogany Wade-Caesar

## LIVESTOCK

### Cattle

#### Calf Diphtheria (necrobacillosis of cattle)

Calf diphtheria is a disease of the aerodigestive tract of calves. It is caused by *Fusobacterium necrophorum*, possibly in concert with other anaerobes and opportunistic pathogens of cattle, and is most common in calves up to 3-4 months of age. Occasional cases occur in older, weaned calves on feed. The disease should be considered of moderate contagiousness between affected animals, though the bacterial agent is ubiquitous in cattle populations and is usually associated with the mucous membranes and gastrointestinal tract.

In North Carolina (laboratories), our experience with necrobacillosis is usually with individual fatal cases. The disease is one that is usually associated with unsanitary conditions and dampness, though any calves can be affected. *Fusobacterium necrophorum* usually gains access through trauma to the mucosa of the oral cavity or larynx to cause illness. In (usually) the younger calves, an oral infection occurs. Stomatitis, glossitis, and cheilitis occur to varying degrees in the calf. Often there is feed impacted in the cheeks and there is invariably a foul odor associated with the growth of the anaerobic bacteria. This oral form is usually associated with consumption of poor quality, abrasive feed by young calves. These calves often have limited milk intake due to mastitis or poor udder conformation in the dam, and thus are forced to scrounge for other available feed. Oral necrobacillosis is less often fatal than the laryngeal form.

Laryngeal necrobacillosis, or the traditional form of calf diphtheria, involves the laryngeal mucosa and deeper tissues of the submucosa and cartilaginous tissues of the larynx. As such, the laryngeal form is a more severe disease, usually causing death in untreated calves. Asphyxiation follows a period of fever, hoarseness, inappetance, and possibly secondary aspiration pneumonia. The gross lesion, involving the larynx and underlying tissues, is fibrinonecrotizing, with ulcerative laryngitis and pharyngitis, and extension to the adjacent oral cavity and trachea (Figures 1 and 2). Microscopic lesions include similar fibrinonecrotizing changes associated with colonies of often mixed bacterial species (Figure 3).

Other diseases caused by *F. necrophorum* include liver abscesses, rumenitis, interdigital dermatitis, and metritis of cattle and other ruminants. A newly emergent disease of captive deer, septicemic necrobacillosis caused by *F. necrophorum*, has been seen with increasing frequency in the North Carolina Laboratory System, as well as other locations throughout the country. The organism is often or usually present in mixed infections with other organisms such as *Dichelobacter nodosus* and *Arcanobacterium pyogenes*. Virulence factors present in *F. necrophorum* that make it of such concern to health of cattle and other ruminants consist of leukotoxin, endotoxin (it is a gram negative organism), adhesion factors, hemolysins, and multiple proteases, among other factors. It is generally concluded that the leukotoxin is the most significant of these factors. Leukotoxin of *F. necrophorum* is a large protein that has specificity for ruminant neutrophils. Damage to neutrophils and release of intracytoplasmic enzymes into the surrounding environment is responsible for much of the injury in the pathogenesis of *F. necrophorum*. Other organisms, such as *Mannheimia haemolytica*, also produce leukotoxins that are structurally different but functionally similar.

References: 1. Nagaraja TG, Narayanan SK, Stewart GC, Chengappa, MM. *Fusobacterium necrophorum* infections in animals: pathogenesis and pathogenic mechanisms. *Anaerobe*. 2005; 11:239-246.

2. Zhang F, Nagaraja TG, George D, Stewart GC. The two major subspecies of *Fusobacterium necrophorum* have distinct leukotoxin operon promoter regions. *Vet Microbiol*. 2006; 112:73-78.



Figure 1: Calf diphtheria: Necrotizing laryngitis. Exudate over the mucosal surface of the larynx has nearly occluded the airway. Photograph courtesy of Dr. Steve Rushton.



Figure 2: Calf diphtheria: Ulcerated larynx. The fibrinonecrotic pseudomembrane over the mucosal surface is evident. Photograph courtesy of Dr. Steve Rushton.

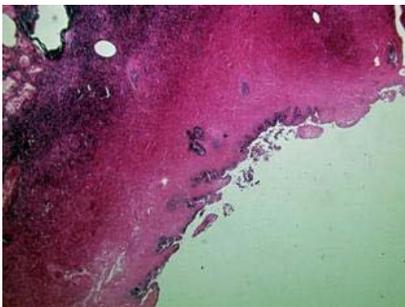


Figure 3: Calf diphtheria: Photomicrograph of ulcerated larynx. Note the surface bacterial colonies.

Dr. Pete Moisan

## Equine

A 8 year old female mixed breed horse was presented to the laboratory system for post mortem examination. The provided history stated the horse presented to the referring veterinarian for colic. The horse remained painful despite medical treatment. Abdominal tap showed a protein level of 4.2, white blood cell count of 45,000 with a few plant material particles. The horse was euthanized. On post mortem examination the animal weighed 450 kg, had a body condition score of 3.5 / 5 and was of normal hydration. Irregularly shaped white colored firm masses were present all along the intestinal mesentery and on the serosal surface of the intestinal tract. There were multifocal well demarcated areas of intestinal ischemia no longer than 5 cm in length in the large colon and distal colon. The intestinal mucosa corresponding to the areas of ischemia were purple to brown in color and frequently contained areas of deep ulceration.

Sections of equine small intestine and colon were examined.

Histopathologic examination of small intestine and colon revealed enterocolitis, eosinophilic and lymphoplasmacytic, diffuse, severe, chronic, with fibrin vascular thrombi and multifocal ulcers.

The histologic lesions were diagnostic for Multisystemic eosinophilic epitheliotropic disease.

Multisystemic eosinophilic epitheliotropic is not a frequently diagnosed disease among horses. It is typically a disease of young horses, with the normal age range being 3 to 13 years of age. Horses usually present with severe weight loss and pitting edema. Diarrhea may or may not be present. The majority of horses will also have cutaneous lesions. Interestingly a peripheral eosinophilia is often absent. As a rule, the horses respond poorly to supportive therapy and are eventually euthanized. The disease is characterized by eosinophilic and lymphoplasmacytic infiltrates with formation of eosinophilic granulomas. These lesions are usually found in multiple tissues including pancreas, salivary glands, gastrointestinal tract, biliary epithelium, bronchial epithelium and skin. The disease has also been reported in cats, dogs, ferrets and humans. The etiology of the disease is unknown, although parasitic, allergic, toxic and viral causes have all been suggested.

In this case the signs of colic were attributed to discomfort secondary to vascular thrombosis of the vessels supplying the intestinal tract and from the mucosa ulcers. The areas of ulceration were worse in the colon, and were likely the source of the peritonitis detected on abdominocentesis.

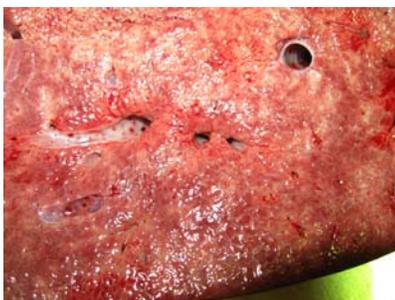
K. M. D. La Perle, et al. **Multisystemic, Eosinophilic, Epitheliotropic Disease with Intestinal Lymphosarcoma in a Horse**, *Vet Pathol March 1998 35: 144-146*

Dr. David Drum

According to the history provided three wild donkeys were found dead on a farm. One was observed to lie down and die with no sign of a struggle. A zebra was also down and unable to stand. The zebra was observed to be chewing on his tongue and paddling prior to euthanasia. His temperature was 97 and he had hay in his mouth. The farms remaining zebra mares were lethargic, stuporous and ataxic. They were unable to swallow and had grain falling out of their mouths. Other hoof stock were in this 100 acre enclosure but were not affected. Botulism was a concern as partially buried carcasses were found on the property with standing water. A chemistry panel on the submitted Zebra revealed elevations in Alk Phos 583, ALT 71, AST 636, CK 959, GGT 72, Amylase 25, Lipase 92, Albumin 3.5, Total Protein 7.8, Glucose 158, Phosphorus 6.7 and decreased Calcium 9.1. Histopathological findings included a hepatopathy that was characterized as microvacuolar and macrovacuolar, moderate to severe, diffuse, acute which could have certainly accounted for neurologic signs (hepatic encephalopathy); however, Botulism could not be eliminated as a primary differential. On the farm, two more zebras exhibited clinical signs consistent with Botulism. The owner elected to treat the remaining zebras with antitoxin. The zebras that were treated with anti toxin, Banamine and other supportive therapies slowly improved. Botulism is caused by a neurotoxin produced by the anaerobic bacterium *Clostridium botulinum* and horses are particularly sensitive to botulinum toxin. Type B toxin is often implicated in botulism in horses and foals in the eastern USA. Although sporadic cases of botulism often are suspected because of the characteristic motor paralysis, it is sometimes difficult to establish the diagnosis by demonstrating the toxin in animal tissues or sera or in the suspect feed.

Liver photo showing hepatic Lipidosis below

Dr. Kim Hagans



History provided for a 14-year-old Percheron mare included recent recumbency, inappetence, dehydration and anuria. Bloodwork revealed liver and kidney abnormalities. The horse was current on vaccinations (rabies, eastern equine encephalitis, West Nile virus, western equine encephalitis, rhinopneumonitis and tetanus). On necropsy, this 809 kg mare was in good body condition and moderate dehydration. The mucous membranes were severely pale pink to white. Severe right renomegaly was identified. The right and left kidney measured 30 x 24 x 18 cm and 25 x 15 x 8 cm, respectively. Expanding approximately 95% of the right kidney was a firm well-circumscribed multinodular tan to white mass containing multifocal to coalescing regions of hemorrhage and necrotic debris. The renal pelvis was markedly ectatic and there was severe hydronephrosis of the ureter. Diffusely lining the mucosa of the right and left renal pelvis and ureters were multifocal to coalescing cystically dilated nodules ranging from 0.5 to 3 mm. Histologic examination revealed renal carcinoma with interstitial fibrosis, lymphoplasmacytic interstitial nephritis and proteinuria, severe hydroureter with diffuse lymphoplasmacytic ureteritis with glandular ectasia and lymphoplasmacytic cystitis. Renal carcinoma is a rare tumor in horses and can affect horses of any sex and age. The tumor is usually unilateral but bilateral kidney involvement has been reported. The most common clinical signs are weight loss, colic and hematuria. Although the neoplasm is locally invasive and metastatic; metastatic lesions are not identified in this case.

Dr. Mahogany Wade-Caesar

## Caprine

According to the history provided this goat was one of 30 that were vaccinated with a CDT injection, given a 1ml injection of a prescription mixed mineral supplement that contained Selenium, Manganese and Zinc (product label 1ml per 100lb) and dewormed with Ivomec. Three goats were found dead two days later in this same group. The goats were fed twice a day a dairy goat pellet mixed with sweet feed in a bunk type feeder and coastal hay was available free choice. The goats that died were all vaccinated from the same bottle of CDT vaccine. Forty two goats were on the farm and no recent losses had occurred. Three 5-7 month old 9-16kg Nigerian Dwarf does were submitted. Autolysis was mild to moderate in each doe. Necropsy findings were essentially identical in each doe and were as follows: external exam revealed a BCS of 3.0/5, hyperemic mucous membranes and bloated abdomens. Abundant adipose tissue was identified in the abdomens. The rumen pH was 4.5 and microbes were nonmotile. Shell corn and ground feed mixed with forage were noted throughout the rumen compartments. The livers were pale tan in color. The abomasums contained a small amount of forage. The small intestines were thin walled and gas distended with a small amount of digesta remaining. Formed feces filled the colons. Mild renal hyperemia was noted and no urine remained in the urinary bladders. The lungs were diffusely hyperemic and congested with a rubbery texture on palpation. Petechia were noted along the epicardial surfaces. No other abnormalities were identified on gross exam. Histopathology abnormalities were confined to the liver in each doe and were characterized as severe acute periacinar hepatocellular necrosis. Liver samples from each doe were referred to PADLS for Selenium testing with the resulting Selenium levels ranging from 5.2 ppm -10.6ppm (normal range .25-1.20ppm). Selenium Toxicosis was diagnosed. Selenium has a narrow margin of safety. Young animals are most susceptible to acute parenteral selenium toxicosis with dosages of 0.2-0.5 mg/kg. Clinical signs are characterized by abnormal behavior, respiratory difficulty, gastrointestinal upset, and sudden death. Abnormal posture and depression, anorexia, unsteady gait, diarrhea, colic, increased pulse and respiration rates, frothy nasal discharge, moist rales, and cyanosis may be noted. The major lesions are lung edema and congestion, and necrosis of multiple organs, including lung, liver, and kidney.

Dr. Kim Hagans

## POULTRY

Mycotic or fungal pneumonia in avian species is a disease usually caused by fungi belonging to the genus *Aspergillus*. Aspergillosis and brooder pneumonia are other names often used to describe this condition. *Aspergillus fumigatus* is the primary agent causing Aspergillosis although other species of fungus are sometimes encountered. The fungal spores are usually inhaled from the environment and mostly infect the trachea, lungs and air sacs. Sometimes the conjunctiva and brain are involved. Young turkey poults, chicks and game bird flocks may experience significant mortality from exposure to high levels of the fungal spores soon after hatching. A recent case of mycotic pneumonia in quail was diagnosed in the Griffin Laboratory. In a flock of 10,000 quail, 5 days of age, approximately 500 birds died over a 2 day period. Some of the birds had been observed coughing before death. Small white to pale tan foci were present in the lungs of all 6 birds presented for necropsies. It is common to find pale tan to yellow nodules in the lungs and caseous plaques in the trachea and air sacs with this disease. Histopathology findings indicated bronchopneumonia, caseous, subacute, multifocal and extensive, with many intralesional fungal hyphae that were morphologically suggestive of an *Aspergillus* sp. The source of infection for these quail was determined to likely have come from a recycled wood product made into shavings used for litter. Treatment for Aspergillosis is generally considered not practical except in birds of high value. Prevention by eliminating any source of exposure is the key component in controlling this disease.

Dr. Reginald Ridenhour

**DEPARTMENTAL NEWS****ROLLINS LABORATORY**

Cindy Nipper (Histology) fulfilled the requirement of the American Society for Clinical Pathology and national Society for Histotechnology and is now a certified Histotechnician.

Katheryn Schmidt was recently notified by the Board of Directors that she has completed the requirements and earned the Manager of Environmental, Safety and Health (MESH) certificate, which is a certificate series sponsored by NC State University, the Safety and Health Council of NC and the NC Department of Labor.

**ELKIN LABORATORY**

Dr. Bradley Barlow is now employed at the Elkin Laboratory this week as a veterinary diagnostician. Dr. Barlow, a native Oklahoman, received BS degrees from Texas A&M University in Animal Science and Biomedical Science, and a DVM degree from Oklahoma State University. His practice experience includes 6 years of Mixed Animal Private Practice in Texas & Virginia and 5 years of Small Animal Private Practice in NC. His professional interest is diagnostic pathology of food and companion animals. He is a member of both the American Veterinary Medical Association and the North Carolina Veterinary Medical Association.

**MONROE LABORATORY**

Griffin Animal Disease Diagnostic Lab received the Public Sector Star designation from the NCDOL in September. The North Carolina Public Sector Star program recognizes state agencies and local governments for their leadership and success in providing a safe and healthy work environment.

**ARDEN LABORATORY**

Dr. William R. "Bill" Rapp passed away at his home in Hendersonville, NC on December 8, 2010. Dr. Rapp served as pathologist and director of the Western Animal Disease Diagnostic Laboratory from September 1987 through August 2001.

**CE ATTENDANCE**

Drs. Pete Moisan, Alison R. Tucker, Richard Mock, Gene Erickson, Mahogany Wade-Caesar and Karen Post all attended the American Association of Veterinary Laboratory Diagnosticians (AAVLD) Annual meeting November 11-17, 2001 in Minneapolis, Minnesota.

Alison R. Tucker attended the 5<sup>th</sup> RTP Rodent Pathology (Hepatobiliary Pathology) conference in Cary NC during September 2010.

Jennifer Haugland attended the Veterinary Medical Forum – New Drugs for Veterinary Medicine, the Small Ruminant Medicine, and the Veterinary Medical Forum – Tick Diseases meetings at NCSU CVM.

Stacy K. Robinson, attended the Small Ruminant Medicine Seminar at the NCSU College of Veterinary Medicine on 08/28/2010.

Dr. Jennifer Haugland, Stacy K. Robinson, Reginald Ridenhour and Gene Erickson attended the 15th Annual North Carolina Veterinary Conference, November 5 - 7 held in Raleigh, NC.  
David Drum attended the Diagnostics in Veterinary Practice meeting on August 29, 2010 at the University of Georgia, and the South Carolina Association of Veterinarians Fall Food Animals and Large Animal Medicine Conference on October 21, 2010 in Greenville, SC.

**CE ATTENDANCE—CONTINUED**

Dr. Kim Hagans attended the American Association of Bovine Practitioners (AABP) conference in Albuquerque, NM on August 18-21, and she attended the North Carolina Star Safety Conference in Greensboro on September 15-17.

Dr. Reginald Ridenhour attended the North Carolina Poultry Health Meeting on November 5, 2010.

Dr. Richard Oliver attended the Pathobiology Seminar at the December Conference for Veterinarians held at the University Of Tennessee College Of Veterinary Medicine on December 1, 2010.

Dr. Richard Oliver spoke on NCDA & CS programs and services at the Small Flocks Poultry Seminar at the Mountain Horticultural Crops Research Station in Mills River on September 8, 2010.

Dr. David Drum attended the Diagnostics in Veterinary Practice meeting at the University of Georgia, college of Veterinary medicine during August 2010, and the South Carolina Association of Veterinarians, Food and Large Animal Medicine Conference in Greenville, SC during October, 2010

## Directory

**Rollins Laboratory - 919-733-3986**

**Director**

[Dr. Karen Post](#)

**Assistant Director**

[Dr. Richard Mock](#)

**Veterinary Pathologists**

[Dr. Tahseen Abdul-Aziz](#)

[Dr. Peter Moisan](#)

[Dr. Steven Rushton](#)

[Dr. Alison Tucker](#)

**Veterinary Diagnosticians**

[Dr. Jennifer Haugland](#)

[Dr. Stacy Robinson](#)

[Dr. Mahogany Wade](#)

**Veterinary Microbiologists**

[Dr. Gene Erickson](#)

[Dr. Karen Post](#)

**Laboratory Section Supervisors**

[Kim Bennett—Virology](#)

[Sandy Murphy—Bacteriology](#)

[Mary Horne—Histopathology](#)

[Jennifer Pruitt—Serology](#)

[Beverly Wood—Molecular Diagnostics](#)

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**Veterinary Diagnostician**

[Dr. Reg Ridenhour](#)

### Diagnostic Laboratory Advisory Committee

Mr. Larry Wooten	N.C. Farm Bureau
Dr. Richard Kirkman	Private Veterinary Practitioner
Dr. Gene Erickson	NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Rick Sharpton	Perdue, Inc
Dr. Shannon Jennings	Nash Johnson Farms
Dr. Leslie Wolf	DHHS- State Public Health Laboratory
Dr. Karen Post	NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Eric Gonder	Goldsboro Milling
Dr. David Marshall	NCDA&CS Veterinary Division
Dr. Randy Jones	Livestock Veterinary Services
Dr. Jennifer Haugland	NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Betsy Sigmon	Creature Comforts Animal Hospital