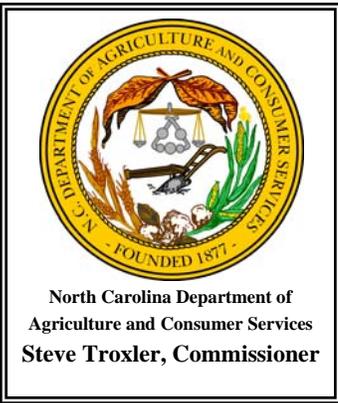


The NCVDLS REPORT



Veterinary News and Information From North Carolina's Diagnostic Laboratories



North Carolina Department of Agriculture and Consumer Services
Steve Troxler, Commissioner

Message from the Director

I am pleased to announce that we are in the final stages of our \$400,000 plus Rollins Laboratory renovation project which began in the Fall of 2008. It is slated for completion by the end of September. The renovation of our former chemistry-toxicology space will provide us with a larger histopathology laboratory and an additional 750 square feet of BSL3 laboratory space complete with robotics equipment to enable us with high-throughput capacity to respond to a high consequence disease outbreak.

Although we have not yet received our certified operating budget for 2009-2010, word has it that we will be operating with approximately 10% less funding than last year. Four vacant positions were also lost when the General Assembly's deliberations were completed. We are being asked to work towards another 5% reversion for this fiscal year. We are continuing to investigate ways to better conserve our resources while expanding diagnostic services. On September 29, a meeting of the External Laboratory Advisory Committee will convene to address these challenges.

In 2008, the Rollins Laboratory was designated as "protected critical infrastructure" for the State of North Carolina by the Department of Homeland Security. Critical infrastructure is defined as "a system or asset so vital to the United States that the incapacity or destruction of such may have a debilitating impact on the security, economy, public health or environment". As a result, a team from DHS conducted a comprehensive security risk site assessment on July 15 to identify and document vulnerabilities. With the final report will come increased awareness of our vulnerabilities and options for considera-

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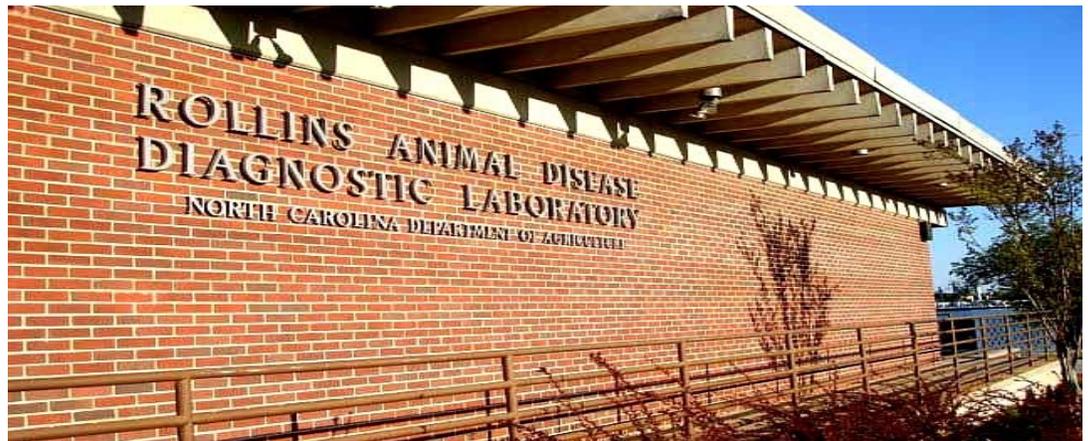
In This Issue...

| | |
|-------------------|----|
| Feature Article | 3 |
| Client Corner | 2 |
| Short Cuts | 4 |
| Departmental News | 10 |
| Directory | 11 |

Holiday Closings...

September 7, 2009
November 11, 2009
November 26-27, 2009
December 24-25, 2009
Our laboratories will be closed on the above listed days.

Please e-mail NCVDL@ncmail.net with any comments and/or suggestions concerning The NCVDLS Report



Message from the Director, *continued*

tion for enhancement of protective measures. The final report will be used as support for coordinating the development and ultimate submission of a Homeland Security Grant for FY 2010 HSGP Funding.

Sincerely,

Karen W. Post DVM, MS

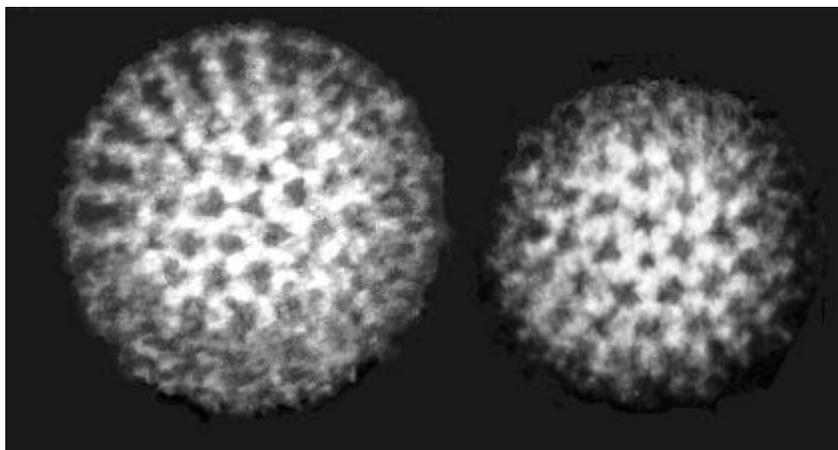
Director of Laboratories

Client Corner

In September, 2009, we will be changing the type of procedure used for detection of rotaviral infection in neonatal animals. For many years, we have used the Wampole Type A rotavirus latex agglutination test kit, which has been discontinued by the manufacturer. Accordingly, we will now change to direct detection by electron microscopy.

Electron microscopic examination has been shown to have a somewhat greater sensitivity, as it also detects type B and C rotavirus particles. The in-laboratory testing time is greater for electron microscopy but should not affect turn-around time.

Dr. Gene Erickson



Human Rotavirus taken from
<http://www.ncbi.nlm.nih.gov/ICTVdb/ICTVdB/00.060.0.03.htm>

Feature Article

By Dr. Tim McComb

As of August 25, 2009, 12 cases of Eastern Equine Encephalitis (EEE) in horses had been reported in North Carolina. The earliest case was reported on July 10 in Gates County, located along the Virginia border in western NC. Other counties with positive diagnoses are also along the Coastal Region, and include Beaufort, Bladen, Columbus, Craven, Currituck, Robeson, Washington, and Wilson Counties. Beaufort County has had the most activity with three positive EEE cases. In 2008 there were a total of 12 cases, reaching into the Piedmont Region with one case each for Johnston and Harnett Counties. While no cases were reported for 2007, only 5 were found in 2006, 15 in 2005, 5 in 2004 and 113 cases in 2003. For the 2009 season, sentinel sampling demonstrates the presence of the virus in Carteret, Chowan, New Hanover, and Pender Counties as well. Sentinels are typically chickens or other birds that are routinely sampled for monitoring purposes.

Eastern Equine Encephalitis is a viral disease closely related to Western Equine Encephalitis and Venezuelan Equine Encephalitis. It is transmitted by mosquitoes and produces disease primarily in horses and less often, humans. Wild birds can carry the disease, but are rarely affected by any illness. In horses, inflammation of the brain and spinal cord can occur in as little as four days post infection. The first signs include fever and generalized weakness, which progress to aberrant behavior and neurologic signs including aggression, head pressing, circling, and blindness. Convulsions and coma ultimately lead to death.

There is no available treatment for EEE, however there are many simple measures that can be taken to prevent the disease. These include routine vaccination for EEE prior to mosquito season, and environmental management to limit the presence of mosquitoes on the farm. This includes eliminating sources of standing or stagnant water, use of insect repellants, fans and screens, and keeping horses inside overnight.

More information can be found at the following websites.

<http://www.ncagr.gov/vet/FactSheets/EEE.htm>

<http://www.aphis.usda.gov/vs/nahss/equine/ee/index.htm>



Short Cuts

COMPANION ANIMAL

Canine

An 8 – 10 week old, unvaccinated, male **German Shepherd** cross puppy died after a 1 day history of anorexia and lethargy with diarrhea and vomiting. An unvaccinated young adult dog on the same premises had died 10 days previously but necropsy was not performed. At necropsy, the liver was diffusely red and friable and there was enteric serosal petechiation. An additional finding was intestinal cestodiasis. Histopathologic changes included marked periacinar hepatocellular necrosis with intranuclear inclusion bodies and lymphoplasmacytic cerebral encephalitis with endothelial cell intranuclear inclusion bodies. Lymph nodes were edematous with lymphoid necrosis and there was also lymphoid depletion with necrosis in the thymus, spleen, and gut-associated lymphoid tissue. A second case submitted to NCVDL a few weeks later was a 10 week old, unvaccinated, female mixed breed dog that died at an animal shelter. The diagnosis in both cases was infectious canine hepatitis (ICH). In this later case there was co-infection with canine parvovirus 2, however, in the first case parvovirus was not present.

Infectious canine hepatitis in dogs and other canids is due to infection with canine adenovirus 1. Pups and young adults are most at risk and immune suppression can exacerbate the progression of ICH. Canine adenovirus 1 infection can also be asymptomatic, or animals can be febrile with mild pharyngitis and tonsillitis. The virus has tropism for hepatocytes, mesothelium and endothelium. In addition to hepatic necrosis, the classic lesions seen at necropsy include lymph node and gall bladder edema, and enteric serosal petechiation, but these are not consistently seen in all fatal cases. Corneal edema can be seen during convalescence. ICH is no longer commonly seen in North Carolina due to the efficacy of vaccination programs; however, the virus does persist in unvaccinated canine populations.

Dr. Alison Tucker

A 13 year old female **Collie dog** was presented to the laboratory for post mortem examination. The history stated the dog lost control of its rear legs and was found dead the next morning. On post mortem examination there was gross distension of the abdomen with approximately 150 ml of blood tinged effusion. The gall bladder had ruptured with over 57 grams of black colored, firmly congealed bile expelled into the abdomen. The margins of the various liver lobes were irregular. Histopathology of the liver was consistent with **hepatic fibrosis and cholestasis**.

Dr. David Drum

A 9-year-old adult male **mixed breed canine** was nonambulatory for 3 weeks and developed labored breathing 3 days prior to death. Nonsteroidal anti-inflammatory drugs were administered during an unspecified period of time. Serum biochemical chemistry panel performed the day prior to death was

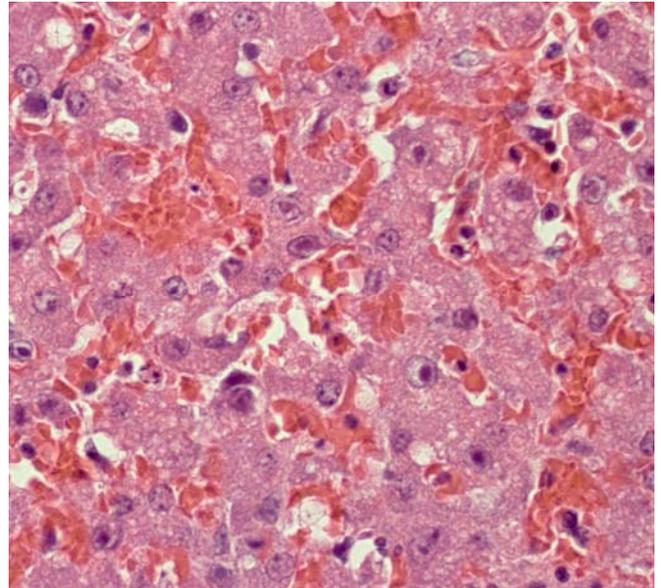


Figure 1 Liver: Hepatocytes contain dark eosinophilic intranuclear inclusions

COMPANION ANIMAL, CONTINUED

unremarkable. On necropsy, this dog was in good body condition with normal hydration. Approximately 600 ml of hemorrhagic fluid (hemothorax) was identified in the thoracic cavity. A 9 x 8 cm and 5 x 7 cm tumor infiltrated right ribs 4-6 and 8-10, respectively. The tumors contained extensive regions of cavitated mineralized trabeculae filled with abundant hemorrhage and necrosis. An identical tumor was located along the dorsal aspect of the L6 vertebra. The remainder of the examination was unremarkable. Histopathologic examination indicated a neoplasm suggestive of a **metastatic carcinoma** in the two masses and lung. The carcinoma was confirmed via pancytokeratin immunohistochemical analysis.

Dr. Mahogany Wade

The body of a 6.5 month old female **Chow Chow** breed dog was presented to the laboratory for post mortem examination. The presented history stated the dog died overnight. The dog was imported from Hungary four months prior. The dog had pneumonia when it arrived in the United States. The infection resolved within two weeks with antibiotics. A week prior to its death, the dog developed lethargy followed by syncope and seizures. The seizures continued to worsen and were non-responsive to medical treatment. Rabies and Canine Distemper were of concern. Lesions of interest on gross examination included multiple pleural adhesions between the chest wall and left lung lobes. Also there was marked dilatation of the lateral ventricles of the brain. The brain was negative for Rabies by FA. Histopathology revealed perivascular, lymphocytic and neutrophilic encephalitis with rare intranuclear inclusions. Also present were rare intraepithelial and intracytoplasmic inclusions in the kidneys. These histologic lesions were consistent with a diagnosis of **Canine Distemper Virus** infection. The hydrocephalus was attributed to tissue damage secondary to the viral infection.

Dr. David Drum

Exotics

According to the history provided the owner reported this 10 month old female **zebra** had been coughing for one week. The veterinarian examined the zebra and administered Banamine, Dexamethasone and Gentamicin for suspected pneumonia. The only clinical sign observed was a temperature of 106 F. Fans were placed in the stall to cool her but she was found dead later that evening. Gross exam revealed approximately 40% of the left lung lobes and 70% of the right lobes were consolidated and dark red in color. The dorsal portions of the lungs were dark pink and aerated. Submucosal tracheal edema and hemorrhage were noted. A hemorrhagic froth extended from the bronchi to the nares. Approximately 150mls of clear red tinged fluid distended the pericardial sac. The myocardium was pale tan. Histopathologic examination identified a bronchopneumonia that was characterized as suppurative, severe, multifocal and coalescing, with cocci bacteria. *Streptococcus equi subsp. zooepidemicus* was identified on cultures of the liver, lung and pericardial fluid. This bacterium is a common isolate from horse wounds. It also is commonly associated with infections of the upper respiratory tract and may be secondary to viral infections.

Dr. Kim Hagans

A 3 month old female **Sugar Glider** was presented to the laboratory for post mortem examination. The presented history stated the animal was acquired 2.5 weeks prior along with a "family" of sugar gliders. The breeding female died approximately a week later. The two baby Sugar Gliders initially seemed fine after their mother's death, but four days later the presented animal was found dead. There were no signs

COMPANION ANIMAL, CONTINUED

of prior illness observed, but it was noted the animals were in thin body condition when acquired. The adults were eating, but didn't seem to be eating well. The previous owner had two other Sugar Gliders die in the recent past. On post mortem examination the animal was in thin body condition and mildly dehydrated. There were multifocal white foci (< 1 mm wide) throughout the liver. The spleen contained similar white foci. Heavy growth of *Listeria monocytogenes* was isolated from the kidney, liver, lung and spleen. As a rule, *Listeria monocytogenes* infections are attributed to contaminated food. It is also possible that "carrier animals" were the source of infection.

Dr. David Drum

Acute pancreatic necrosis was diagnosed in a recent case of sudden death in a **Quaker parrot**. The seven year old female was observed to be healthy in the morning and was found deceased that afternoon. This bird had a history of feather plucking and other than feather loss over the breast area was in good general body condition. Significant findings included coagulative necrosis of the pancreas, hepatocellular necrosis and mesenteric fat necrosis. Acute pancreatic necrosis is occasionally seen in psittacine birds, particularly in Quaker parrots. A review of our laboratory accessions indicates this to be the fifth necropsy diagnosis of acute pancreatic necrosis in parrots diagnosed dating back to 1995. Four of these cases involved Quaker parrots and the other was an African Grey. A cause for this condition is unknown; however excess body fat has been suggested to be an associated factor. Quakers and other parrots can be prone to becoming overweight and developing fatty liver disease.

Dr. Reg Ridenhour

A 32 year old 160kg male **Black bear** was reported to be congested and the caretaker noted the respiratory distress was worse when the bear was down and improved upon standing. The bear died shortly before the veterinarian arrived. Gross exam revealed a football size lobulated mass that filled the thoracic inlet and extended up the ventral neck. The mass was tan in color and cystic with cavitations that were distended with yellow fluid that was gelatinous to liquid in consistency. Tracheal mucosal hyperemia with submucosal edema was noted. Five small .25cm diameter white calcified foci were noted along the tracheal submucosa in the distal trachea. Generalized cardiomegaly was evident with the left ventricular wall thickened. The lungs were fluid congested and were hyperemic. The liver was enlarged with a prominent nutmeg pattern to the parenchyma. Two small 1cm light pink nodules were identified on the splenic parenchyma. The stomach was distended with feed and fluid. The intestines were pale. Pitted depressions and dark focal lesions were noted on the kidneys. No other abnormalities were identified on gross exam. Histopathologic examinations of the mass revealed a **cystic thyroid adenoma** and metaplastic bone in the trachea. This tumor had enlarged to the point of restricting air flow in the trachea.

Dr. Kim Hagans

An 11 year old **pot bellied pig** was presented to the laboratory for post mortem examination. The presented history stated the pig had hard ears and purple skin. The pig had stopped eating about 5 days prior. Necropsy findings included two choleliths in the gall bladder (4 cm x 2 cm x 2 cm, and 2.5 cm x 2 cm x 2 cm). There was gross sludging of bile in the intralobular bile ducts (more prominent at the edges of the lobes). The distal one-third of the ears (tips) were dark in color, hard on palpation and there was a sharp line of demarcation between the effected skin and normal appearing skin. A neutrophilic and lymphocytic portal hepatitis with necrosis, fibrosis and severe cholestasis was identified on histopathology.

Additionally there was fibrinonecrotizing, vasculitis of the ear with necrotic cartilage. Bacterial culture of the liver was non-diagnostic. The pig's death was attributed to a **bacterial septicemia** likely secondary to the gall stones and bacterial infection of the liver. The ear lesions were due to vasculitis, which is a commonly seen in septic pigs.

Dr. David Drum

SURGICAL BIOPSY

Toxoplasma gondii is an Apicomplexan protozoan associated with disease in a wide range of species. Three cases of feline systemic toxoplasmosis have been diagnosed at the NCVDLs/Rollins Animal Diagnostic Laboratory recently.

Domestic and wild cats are definitive hosts and all warm blooded animals, including cats, can serve as paratenic hosts. *Toxoplasma* replicates by endodyogeny (two progeny form within a parent cell) in tissue of paratenic hosts and by merogony (asexual replication) and gametogony (sexual replication) in the enterocytes of the definitive host. Unsporulated oocysts are the product of gametogony and are shed in the feces of infected cats. Oocysts are not infectious until sporulation, which occurs 1 to 5 days later. Enteritis may be observed in kittens and shedding of oocysts lasts approximately 2 weeks. Paratenic hosts are infected by ingesting sporulated oocysts. Sporozoites ex-cyst in the intestine and rapidly multiply (tachyzoites) and spread through the body. Transplacental transmission can occur at this stage. In tissue, tachyzoites can continue to replicate or can develop into intracellular tissue cysts. These tissue cysts measure 10 to 100 μm diameter and contain numerous slowly replicating bradyzoites. Consumption of tissue containing cysts is also a source of infection, but only in the intestinal tract of cats will zoites enter into merogony and gametogony in enterocytes with fertilized oocysts shed in the feces for the cycle to begin again.

Paratenic host reaction to *Toxoplasma gondii* varies by species and ranges from asymptomatic to sudden death. Tachyzoites free in the tissue cause necrosis as they replicate. Inflammation is secondary

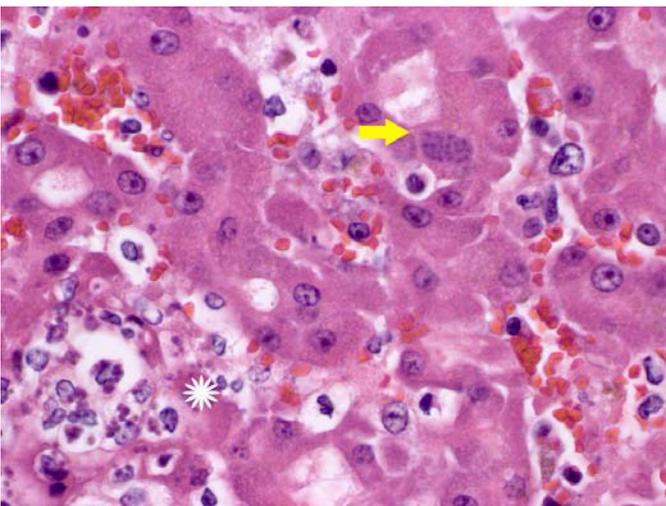


Figure 1: Photomicrograph of a liver from a *Toxoplasma gondii* infected cat demonstrating both tachyzoites (star) within tissue necrosis, and intracellular cysts (arrow) containing numerous bradyzoites. HE

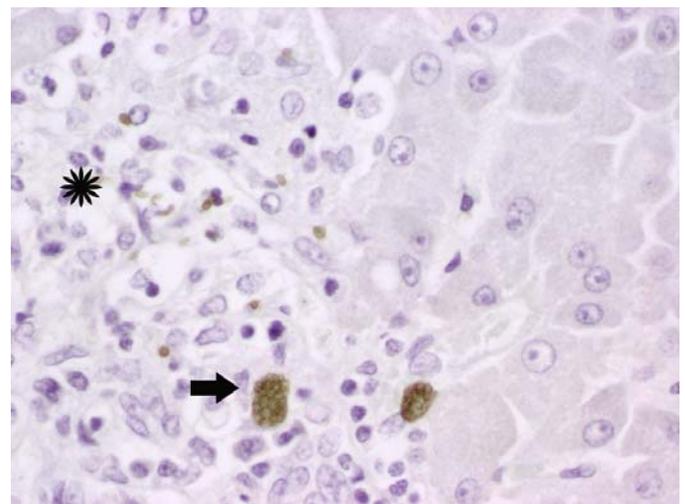


Figure 2: Photomicrograph of a liver from an infected cat with immunoreactivity to *Toxoplasma gondii* tachyzoites (star) and intracellular cysts (arrow) containing bradyzoites.

SURGICAL BIOPSY, CONTINUED

to the necrosis and when there is sufficient damage animals can become clinically ill. Clinical signs of course depend upon the organs infected. In cats as paratenic hosts disease is most frequently associated with immunosuppression and pneumonia, encephalitis, hepatitis, myocarditis and other signs of systemic disease associated with toxoplasmosis. Dogs can have similar signs and in small ruminants, necrotizing placentitis leads to abortion. Ante-mortem, serum antibody titers can determine exposure. Immunohistochemistry is a sensitive and specific tool used to identify *Toxoplasma gondii* as a cause of disease in paratenic hosts, including cats, when few tachyzoites and no tissue cysts are present or when tissue destruction obscures the organisms.

Toxoplasma gondii is a zoonotic disease. Humans most at risk are pregnant women, due to the risk of transplacental transmission, and immunosuppressed individuals. Immunity in previously exposed women can be protective but women who have not previously been exposed should avoid handling cat feces or eating undercooked meat, particularly lamb or pork, during pregnancy. Concerned individuals should consult with their physician with questions.

Dr. Alison Tucker

LIVESTOCK

Equine

A 20 year old black colored female **Tennessee Walking Horse** was presented to the laboratory for post mortem examination. The presented history stated the horse was euthanized. The horse had exhibited neurological signs for the past month. The horse had an attitude change, difficulty swallowing, bad breath, weight loss, and increased sensitivity to sounds. Rabies testing was requested. On post mortem examination the horse was in good body condition and adequately hydrated. The brain was very soft and did not fluoresce when viewed under ultraviolet light. There was mild edema of the retropharyngeal tissues. Multiple internal nodules were visualized on cut section of the thyroid glands. Testing for Rabies virus, West Nile virus and the Eastern Equine Encephalomyelitis virus were all negative. An **infiltrative carcinoma**, most suggestive of nasal carcinoma, was identified histologically in the cerebrum along with nodular adenomatous hyperplasia of the thyroid gland

Dr. David Drum

A 2-day-old **Hanoverian filly** developed colic post-foaling. Meconium impaction or intestinal intussusception was suspected; therefore a nasogastric tube was passed and the foal was treated with Banamine and enemas (every four hours). The animal eventually became depressed, inappetent, and failed to defecate. Fecal material was not identified on rectal examination. On gross examination, the abdomen was moderately distended and firm. There was segmental atresia of the colon, characterized by an absence of communication between the proximal and distal colon. The proximal colon was severely distended with ingesta and air. The distal segment of the colon had a blind end. **Atresia coli** is a rare condition in newborn foals and is characterized by an incomplete digestive tract, due to part of the colon being missing. This condition is most likely congenital; however some scientists suspect that a loss of blood supply induces atrophy and even sometimes atresia of the affected digestive tract segment. Foals affected by atresia coli usually begin to show clinical signs within the first 48 hours of life. At the beginning they seem to be generally in good condition but are unable to pass the meconium. Later the foals show signs

LIVESTOCK, CONTINUED

of abdominal pain and abdominal distension.

Reference: Turek, Bernard and Gregory Verhoeven. "Atresia coli in a foal - case report." *Medycyna Wet.* 2008, 64(3), pages 280-283.

Dr. Mahogany Wade

A 10 year old **Oldenburg mare** was found dead in her stable with no prior signs of illness. One day prior she had been exercised on a treadmill with no reported problems, and maintained a normal appetite. On necropsy examination, the abdomen contained approximately 2 liters of brown watery fluid with ingesta and manure present. A large transmural rent was discovered at the base of the colon and present within the colon was an **enterolith** measuring 19cm x 13cm x 12cm and weighing 3.5 kg. Enteroliths form in much the same way as uroliths, through a combination of pH and mineral balances. Other risk factors in horses include a diet consisting of greater than 50% alfalfa hay (due to Ca:P balance), lack of exercise and pasture access, and ingestion of foreign bodies. The rate at which enteroliths develop are poorly understood.

Dr. Tim McComb

A 1-year-old female **Quarter Horse** became febrile and neurologic (circling), was found recumbent with decreased mentation, and was euthanized. Bloodwork revealed mild anemia and lymphopenia. The vaccination history was unclear; however the horse was not vaccinated for rabies virus. Two other horses on the farm were also febrile with decreased mentation. Gross examination of the horse was unremarkable. Histologic examination revealed moderate to severe multifocal perivascular and lymphohistiocytic and neutrophilic encephalitis and myelitis with neuronal necrosis. These findings are consistent with **Equine Eastern Encephalomyelitis**, which was confirmed in the brain stem, cerebellum, and cerebrum by PCR analysis. The neural tissues were negative for West Nile Virus and Rabies virus.

Dr. Mahogany Wade

Camelid

An older adult male gelded **Llama** was presented to the laboratory for post mortem examination. The presented history stated the llama was found down, then stopped eating, and appeared cold and exhausted. The animal was euthanized two days later. On post mortem examination the llama was in lean body condition and mildly dehydrated. There was 20 ml of serosanguinous effusion in the pericardial sac. The cranial lung lobes were rubbery on palpation. The bone marrow was pale in color. A chronic moderate, regionally extensive **axonal degeneration** of the white matter of the brain was identified on histopathology, along with acute, diffuse bronchopneumonia. The etiology of the axonal degeneration in llamas is unknown. In other animal species, copper deficiency can cause "swayback" and this type of vacuolar change, particularly in spinal cord.

Dr. David Drum

A four year old female **alpaca** was found in the pasture with shallow respirations and an aborted near term fetus. The adult had been purchased and transported from New Mexico in the early spring and was confirmed pregnant on arrival. The owner noted thin body condition throughout the entire pregnancy, otherwise no significant health problems. The adult and fetus were both presented for necropsy

LIVESTOCK, CONTINUED

examination. There were no abnormalities noted in the fetus. In the adult, the abdomen contained approximately 1.5 liters of clear, straw colored fluid and a large amount of fibrin. A 1 cm diameter **perforating ulcer** was present in the pyloric region of C3. Just cranial to the perforation, the mucosa of C3 contained a large non-perforating ulcer. As with other species, ulcers in camelids can be stress induced. The stress of transportation and pregnancy may have triggered the ulcers in this case.

Dr. Tim McComb

DEPARTMENTAL NEWS

ROLLINS LABORATORY

Virology: Sharon Graham's son Kelvin accompanied his team from Partnership Elementary School to a world competition "Odyssey of the Mind" in Ames, Iowa. This competition presented problems to teams that challenged their creativity with problem solving. Kelvin's team earned a 25th place finish. Sharon is a Medical Laboratory Technologist I in Virology, and she attended the competition as a chaperone.

The Virology Laboratory recently passed the NVSL Proficiency tests for EIA, BLV, and BT testing with a 100% score on each one.

With the state hiring freeze finally lifted, we are welcoming new employees at Rollins.

In Bacteriology: Medical Laboratory Technologists Deborah Buffalo, Wendy Smith-Kerr, and Brenda Cummins

In Molecular Diagnostics: Angela Murphy.

Jeremi Clark has resigned her position in Quality Assurance and has accepted a position in the clinical trials section at Lab-Corp. We thank Jeremi for her 4 years of service and wish her much success in her new endeavor.

CE ATTENDANCE

Several pathologists and diagnosticians from the Rollins Laboratory attended the 37th Annual Southeast Veterinary Pathology Conference in Tifton, Georgia on May 16-17, 2009. Dr. Wade presented "Lymphocytic Thyroiditis and Atherosclerosis in a Dog"; Dr. Moisan presented "Hepatic Encephalopathy in a Calf"; Dr. Rushton presented "*Chromobacterium violaceum* Septicemia in a Dog"; Dr. Tucker presented "Cutaneous Dracunculosis in a Dog"; and Dr. Wendy Royce (in conjunction with Dr. Richard Oliver) presented "Atypical Pasture Myopathy in Two Groups of Horses". There were 81 presentations at the meeting.

Dr. Moisan also attended the 28th Annual Meeting of the Midwest Association of Veterinary Pathologists in Turkey Run State Park, Indiana on August 13-14, 2009. There were 52 presentations at the meeting. Dr. Moisan presented "Actinobacillosis in Swine".

Dr. David Drum attended the 5th Annual Foreign Animal Disease Training Course sponsored by the Colorado State Department of Agriculture, Animal Population Health Institute, and USDA APHIS Centers for Epidemiology and Animal Health (CEAH) at the USDA-APHIS facilities in Fort Collins, Colorado from July 20-24, 2009.

Directory

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Diagnostic Laboratory Advisory Committee

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