A Note from Our Director...

The Rollins Animal Disease Diagnostic Laboratory will be moving to our new co-located agricultural facility, the Agricultural Science Center, this spring! As we move and transition into our new laboratory, we are hoping for only a minimal disruption on testing services and turn-around time. We are planning for an April move date but stay tuned for updates and the exact move timeline as final planning and logistics are completed.

Our new address will be:

Rollins Animal Disease Diagnostic Laboratory
4400 Reedy Creek Road
Raleigh, NC 27607

- Jim Trybus, DVM, DAVCP
Welcome our new Assistant Director Dr. Lalitha Peddireddi!

Dr. Lalitha Peddireddi joined us from Kansas State Veterinary Diagnostic Laboratory (KSVDL) and College of Veterinary Medicine at Kansas State University, Manhattan, KS. She started as the Section Head of Molecular Diagnostics section and Assistant professor at KSVDL and later promoted to an Associate Professor. As the Section Head for Molecular Diagnostic Services, she was actively involved with budgeting and fiscal management of her section, client consultation, collaborative research and managed several technical staff members and student employees.

Dr. Peddireddi is a veterinarian by profession. She obtained her professional degree in Veterinary Medicine from Acharya N.G. Ranga Agricultural University, Hyderabad, India. She obtained her Master's degree in Animal Sciences (with an emphasis on animal nutrition) from Purdue University, West Lafayette, IN and PhD in Pathobiology with a special emphasis on Molecular Microbiology from Kansas State University, Manhattan, KS. After completion of her PhD, she joined KSVDL as the Project Lead for KSU and Industry Research Collaborations. In this role, she led several molecular diagnostic test development and validation projects, which had resulted in >40 new molecular tests to her laboratory.

She has been actively involved in extramurally funded collaborative research focused on new pathogen discovery, understanding the pathogenicity and epidemiology of new and emerging viruses, and developing molecular-based diagnostic tools to detect veterinary pathogens. She has also collaborated with industry partners to develop and validate molecular assays and has led the validation of two USDA approved PCR-based tests to detect BVD and *Tritrichomonas foetus* offered by Thermofisher/Life Technologies. These tests are now routinely used in many accredited veterinary diagnostic laboratories. Her special interests include developing high throughput and cost-effective methods including multiplex panels, streamlining laboratory processes which help to reduce laboratory testing costs and improving turnaround time.

Dr. Peddireddi served in various important committees within the department such as the foreign animal disease committee, quality assurance committee, and subject matter expert for COVID-19 testing. She has been actively associated with various professional organizations such as American Association of Veterinary Laboratory Diagnosticians, CRWAD and ACVM. She has authored and co-authored several peer-reviewed publications in reputed journals and served as journal editor and ad-hoc reviewer for several peer-reviewed journals. Dr. Peddireddi has won numerous awards including Women of Distinction award from the Kansas State University and Distinguished Service Award from Kansas Veterinary Medical Association.

Dr. Peddireddi’s husband, Dr. Charan Ganta, is a veterinary pathologist with research interests in investigative pathology, regenerative medicine and stem cell biology. They have three kids, 8-yr. old twin boys Sam and Nik and a 14-yr. old daughter, Leena. They love to travel to different places and experience the beauty of diversity, new cultures, people and try new foods. They also love gardening and spending time together outdoors hiking and walking.
Please welcome Dr. Ashley Talley to our diagnostic laboratory system. Dr. Talley joins us as a veterinary anatomic pathologist. She completed a B.A. degree in biology from the University of Virginia and went on to earn her veterinary degree from Kansas State University. Following veterinary school, Dr. Talley completed a one year internship in small animal medicine and surgery from the University of Pennsylvania. She completed her 3 year anatomic pathology residency at North Carolina State University and became board certified in 2017. Her professional interest/focus is in diagnostic pathology.

Dr. Kristine Blankenship has joined the Griffin Laboratory team in Monroe, NC. Dr. Blankenship earned her veterinary degree from Ohio State University. For the past 6 years, Dr. Blankenship has been the Administrator of Shelter Operations at Gaston County Animal Care and Enforcement (GCACE). Prior to her position at GCACE, Dr. Blankenship was the Department Chairwoman of Gaston College’s Veterinary Medical Technology Program for 12 years where she was the primary instructor of 10 core courses including anatomy and physiology, parasitology, pharmacology, large animal practices, and laboratory techniques. She also practiced as an equine veterinarian in Ohio. Dr. Blankenship is the recipient of the Gaston County Police Chief’s Commendation and was the Gaston County Employee of the year in 2016. She is a member of the AVMA, the NCVMA, and the State Animal Response Corp.

Dr. Blankenship currently lives on a farm in Lincoln County where she keeps horses, donkeys, dogs, and cats.
Fibrinosuppurative Meningitis in a Cat

By: David Drum, DVM

The body of a 1 year, 4 month old, male domestic long haired cat was presented for postmortem examination. The provided history stated the cat had died at home. The owner reported no signs of illness until 2 days prior to death, which was the day the exterminator came to the house for routine in-home pesticide application. The owner felt the cat started "acting funny" afterward- wobbly, lethargic, and the cat wouldn't come when called. Also, they reported the cat didn't want to eat, drink, or use the bathroom. They reported the other animals in the household seemed fine, but they were not where the pesticide had been applied. They reported the cat was up to date on vaccinations.

On postmortem examination the cat weighed 4.08 kg, had a BCS of 2.5/5, was ~ 7% dehydrated and there was a moderate degree of postmortem change to the body. The lungs overall were dark red in color and slightly rubbery on palpation. The heart weighed 18.8 grams. The cat’s stomach contained a scant volume of brown colored mucoid content. There was a moderate amount of thick brown-yellow colored exudate in the right ear canal. The right tympanic membrane was red colored and swollen. On examination of the brain, there was ~ 2 ml of thick, green-yellow colored exudate overlaying the meninges of the right dorsolateral cerebrum. A small amount of exudate appeared to be subdural also. No signs of bodily trauma, no abnormalities on oral examination or on examination of the rest of the body. Upon completion of the gross examination of the body, an epidural abscess and otitis were diagnosed.

Tissue sections were submitted for histopathologic examination. The pathologist reported severe, subacute, fibrinosuppurative meningitis, along with congestion in the submitted sections of lung tissue. The pathologist commented there were coalescing colonies of bacteria in the epidural exudate, with histopathologic findings confirming an infectious/inflammatory process involving the meninges and epidural space.

The owner was still concerned about the possibility of a toxicosis from the applied in house pesticides, so liver tissue was referred to a veterinary toxicology laboratory for routine screening tests which cover a large number of compounds including pesticides, a number of therapeutic and illicit drugs and environmental contaminants. The laboratory reported they did not detect any toxins in the liver tissue.

I found this case to be of interest in emphasizing the point that it is important to keep an open mind as to possible causes of illness and to not let your focus turn toward a single fact or event in the presented history, otherwise differentials and antemortem diagnostic testing could be lead astray.
Johne's Disease

By: David Ackerman, DVM

Johne's Disease, also known as Paratuberculosis, is an infectious condition that often occurs in cattle, sheep, goats, and many other nontraditional ruminants. The etiological cause is the organism *Mycobacterium avium* spp. *paratuberculosis* (MAP). Most of us probably remember learning about Johne's Disease from veterinary school and board exams but let us do a little trip down memory lane.

Johne's Disease typically presents as a chronic debilitating enteritis. The infected symptomatic animal, typically an adult over two years old, often appears as a “poor doer”, will not gain weight, not producing to the level of its herd cohorts, has diarrhea, but eats well and looks alert. If the animal is alert and active enough, Johne’s Disease may not even make the differential list. Severe advanced cases will often look like other conditions which result in hypoproteinemia. When present in small ruminants the condition can be difficult to diagnose antemortem because of the sporadic nature of the condition in a group of animals. The organism MAP typically is introduced to a naive healthy, often young, animal by ingestion of contaminated feces in feedstuffs or milk from an often asymptomatic infected dam. Many times, an asymptomatic newcomer to the herd or group of animals will introduce the organism to the farm. Young ruminants may become infected in utero from a diseased dam.

A case recently presented to Northwestern Animal Disease Diagnostic Lab, part of North Carolina Veterinary Diagnostic Laboratory System. A dead six-year-old bull was presented for necropsy after being treated for chronic weight loss, diarrhea, and pneumonia by the referring veterinarian. While alive, the bull had been bright, alert and maintained an appetite, so feeding was increased and multiple deworming medications had been given. Recently the diarrhea had worsened, and the bull developed symptoms consistent with pneumonia. Prior to death the bull was treated for dehydration and with antibiotics.

**Gross necropsy revealed:** 1) diffuse mild interlobar emphysema with multifocal areas of acute, moderate pulmonary parenchymal congestion, 2) segmental serosal congestion with moderate thickening of the mucosa of the distal small intestine, (example figure 1) 3) cecal and colon distension with chronic moderate thickening of the bowel wall.

**Figure 1.** upper-normal mucosa; lower-thickened mucosa courtesy: johnes.org
**Histopathology noted:** 1) subacute mild to moderate neutrophilic bronchopneumonia, 2) expansion of small and large intestinal lamina propria and submucosa by sheets of macrophages (figure 2) and intralesional acid fast organisms (figure 3).

**Diagnosis:**

1) Granulomatous Enterocolitis with presence of acid-fast organisms consistent with the presence of *Mycobacterium avium* spp. *paratuberculosis* (MAP), the etiologic agent for Johne’s Disease.

2) Bacterial Bronchopneumonia

The clinical symptoms of weight loss and diarrhea are caused by the expansion and replacement of the lamina propria and submucosa by inflammatory cells and the organism MAP. To compare the tissue damage done by the presence of MAP and associated inflammation, note the difference in the photomicrographs from an infected (figure 2) and non-infected (figure 3) animal. The intestinal mucosa and submucosa are greatly thickened by macrophages/granulomatous inflammation (figure 2). In the postmortem animal, histopathology/acid-fast staining of the diseased sections of intestine to identify the organism MAP is diagnostic for the disease. The abundant red intracellular organisms present in photomicrograph figure 4 represent the acid-fast positive MAP bacteria.
Fecal cultures from dead and live animals are also diagnostic. However, the organism is difficult to culture, taking 5-16 weeks to grow the bacteria. Literature and studies suggest that only 40% of positive animals in a herd are discovered by bacterial culture as a diagnostic herd management tool, primarily as not all infected animals will be shedding the organism, depending on their stage of infection.

Since the discovery of Johne’s disease in 1895, there has been no successful treatment developed to eliminate MAP in infected ruminants. However, literature has noted the use and success of the ionophore Monensin to treat Johne’s disease in highly valued genetic cattle.

Prevention, identification of infected animals and removal from the herd are the primary options. Herd side identification of infected animals can be performed by serological testing such as ELISA and AGID to detect antibodies in serum. These tests are inexpensive, rapid and any accredited veterinarian can submit samples to a USDA approved laboratory. Polymerase Chain Reaction (PCR) testing on feces is the most accurate diagnostic test to identify MAP in live animals. PCR testing is a more expensive testing platform; however, pricing will come down as this format is more commonly used by diagnostic laboratories.

APHIS Veterinary Services has developed a Johne’s Disease Control Program to help livestock agricultural producers and veterinarians reduce the spread and maintain Johne’s disease free herds.

APHIS Veterinary Services can provide the list of program standards and additional information about Johne’s disease, which can be accessed at their website https://www.aphis.usda.gov

References:

Johne’s Disease https://www.aphis.usda.gov
Johne’s Information Center https://www.johnes.org
Photomicrographs: courtesy of James Trybus, DVM, DACVP
A Neurologic Presentation of Acorn Toxicity

By: Jennifer Haugland, DVM

In November, a beef farm had 5 to 7 calves, aged 7 months and younger, 2 heifers, aged 18 months, and 1 cow die in a 2-week period prior to a calf being submitted for necropsy. The clinical signs seen in the cattle were aggression (charging owner), straining to defecate, dropping feed from mouths, and excessive salivation. The calves were still nursing. After a couple of calves had died, the herd was moved to a pasture with fewer oak trees. Owner reported there were no sources of lead nor arsenic in either pasture. The 7-month calf submitted for necropsy had been found in sternal recumbency and when the owner approached, the calf jumped up and charged him. The calf was found dead the next morning. These aggressive clinical signs are suggestive of rabies, lead toxicity, grass tetany, salt toxicity, nervous coccidiosis, and hypoxia.

Necropsy examination found a calf in thin body condition (BCS 3/9) and moderately dehydrated. Rumen contents were watery, and the pH of the fluid was between 7.0-8.0. A single whole acorn and a few broken pieces of acorns were found in the rumen. There were a moderate number of black granulomas in the wall of the small intestine. Feces were green and loose but there was no manure on tail or on back legs. A scraping of colonic mucosa revealed only very few coccidia oocysts and very few trichostrongyle eggs. Ocular fluid was negative (0 ppm) for nitrate using water quality test strips. Rabies fluorescent antibody test was negative.

Histopathology of the major organs revealed marked degeneration and necrosis of the kidney tubules with interstitial nephritis. There were no indications of an infectious or inflammatory disease in the brain. Kidney failure was likely, given the extent of the kidney lesion. Renal tubular necrosis can be seen with ingestion of nephrotoxin containing weeds (pigweed, oak/acorn), heavy metal toxicity, and with some medications.

The cause of death was diagnosed as kidney failure due to acorn ingestion. Based on the herd history and the presence of acorns in the rumen, this is the most likely nephrotoxin to affect this herd.

The lack of any infectious or inflammatory lesion in the brain suggests the neurologic signs of charging, dysphagia and ptyalism are likely due to uremia. Neurologic clinical signs are not described in cattle with acorn toxicity. Only two case reports of uremic encephalopathy in cattle were found and one reported the cause as tannin intoxication.

Antemortem diagnostics would include physical exam and blood chemistry alterations consistent with kidney damage. Oral ulceration, abdominal pain, anorexia, diarrhea (sometime with blood) are early clinical signs that might be misinterpreted as parasitism. Then weight loss, rough hair coat, straining to defecate, and black firm feces are more subacute to chronic clinical signs. Abdominal fluid (ascites) and edema of legs can also be seen. Cattle usually die from kidney failure within 7 days of the onset of clinical signs. Nursing and weaned calves in the herd are more likely than mature cows to be affected in a herd. Tannic acid, a toxic metabolite, can concentrate in the milk.
Postmortem diagnostics require a necropsy and histopathology. Necropsy lesion of firm black feces, abomasal irritation and acorns in the rumen can make a presumptive diagnosis. Histopathology is needed to confirm kidney damage. After several days of anorexia, there may only be a few acorns left in the rumen and thus difficult to find. Therefore, the lack of abundant acorns does not rule out acorn toxicity until histopathology rules out tubular necrosis in the kidneys. For the eastern half of North Carolina, October and November are the months that acorn toxicity is diagnosed. We rarely diagnose oak bud toxicity in the spring. The early green acorns are considered more toxic and more palatable. Restricting access to acorns during this time, especially as pasture is often limited, is the best method to prevent acorn toxicity. Once clinical signs appear, there is nothing that can be done to reverse the kidney damage. Some cattle, usually the cows, may not die but will have diminished kidney function and perform poorly. By late November, the squirrels and deer will have removed many acorns and the remaining weathered ones are not as palatable. There are year to year variations in the number of acorns produced and in the level of tannin in the acorns as well as the amount of remaining pasture, so often farms comment that in prior years cattle did not seem to die from acorn toxicity. Acorn toxicity is not often a herd wide problem and so in some years the few deaths may be attributed to other problems. All varieties of Oak trees do produce tannins in the acorn, young leaves, and stems.

There is another consequence of acorn toxicity known as “Acorn Calf Syndrome” which occurs when cows consume a large amount of acorns during the second trimester (3-7 months of gestation). These calves will be deformed with short legs, abnormal hooves, misshapen heads and often look like dwarf calves. Poor nutrition for cow also contributes to this problem.

Sheep can also suffer from acorn toxicity. Goats and horse are less affected. Deer can eat large amounts of acorns without ill effect because their saliva produces proline-rich salivary proteins (PRPs) that bind the tannins in the mouth and prevent the GI and kidney damage. Cattle and sheep produce less PRPs than goats.

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