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VIRGINIA ANIMAL DIAGNOSTIC NEWSLETTER

A joint publication between Virginia Department of Agriculture and Consumer Services and the Virginia Tech Animal Laboratory Services

EDITORIAL

Testing for Chronic Wasting Disease (CWD)

The last thing a hunter wants to hear after a successful deer harvest, time and money spent processing, and awaiting the first meal it will provide, are the words, “Your deer has tested positive for chronic wasting disease.” Yet that is the unfortunate reality for more hunters each year in Virginia and nationwide.

Chronic wasting disease (CWD) is a neurological disease that affects deer species and eventually leads to death. For 1.5 to 2 years after becoming infected, deer will appear healthy, making it impossible for hunters to discern whether a deer has the disease or not. The only way to confirm CWD in a deer is through post-mortem testing. The infectious agent of CWD, a prion, or misfolded protein, makes it a particularly difficult disease to fight. Infected deer can easily spread prions to each other, and those prions can remain infectious for years in soil. There is currently no treatment for CWD and no cure. In addition, any potential risk to humans is not well understood, though there have not yet been any documented cases in people.

Wildlife management agencies are concerned about what CWD means for the future of wild deer herds. In certain western states, population-level declines have already been observed as a result of CWD. Although Virginia has not yet seen similar declines, the disease continues to spread. The Virginia Department of Wildlife Resources has developed a CWD Management Plan with goals of preventing the spread to new areas of Virginia and slowing the spread of CWD in areas where it already exists. In order to understand the spread and detect the disease in new areas, Virginia has an extensive surveillance system that relies on submissions from hunter-harvested deer. DWR collects samples from deer submitted by hunters through taxidermists, meat processors, voluntary testing stations, and through a mandatory testing day in certain counties.

If you would like to get a deer tested for CWD, you can find your nearest testing station through the DWR online map using this link: https://experience.arcgis.com/experience/6805eec0b6534f3cb6bc6c85ddcc2ee0 or by going to www.dwr.virginia.gov/cwd. In addition to the voluntary testing stations, VDACS laboratories in Wytheville,

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Lynchburg, and Warrenton are able to submit samples for CWD testing for a fee. In order to have a deer tested, the head and 3-4 inches of the neck will need to be brought to one of these sites. Results can take anywhere from 2-6 weeks to receive back and are available on the DWR website. More information about CWD in Virginia, current regulations, and CWD best practices can also be found on the website. We rely on you to help slow the spread of CWD!

Alexandra Lombard, MS.
Wildlife Health Coordinator
Virginia Department of Wildlife Resources
www.dwr.virginia.gov

HORSES

Rhodococcus equi (*Prescotella equi*) in a foal

A two-month-old American Saddlebred foal was submitted for necropsy, with history of progressive respiratory problems and diarrhea. At necropsy, multifocal to coalescing tan to white firm nodules ranging from 2 mm to 6 cm diameter were identified in the lungs. Nodules were encapsulated and contained white granular to caseous material. The mesocolonic lymph nodes were markedly enlarged, tan, and firm, and contained similar white caseous material. The mucosa of the right ventral and dorsal colon was red and had multifocal umbilicated ulcers (approximately 1cm diameter) that could be visualized from the serosal surface. The microbiology laboratory isolated *Rhodococcus equi* from a sample of lung collected during necropsy. *Rhodococcus equi* is a bacterium that lives in the soil and is frequently isolated in cases of foals with granulomas in the lung. Infection progresses slowly and foals display clinical signs late in the disease process. Half of the cases can present ulcerative colitis and mesocolonic lymphadenitis. This infection is rare in animals older than a year, and in such cases, it is associated with immunosuppression.

Francisco R Carvallo, DVM, DSc, DACVP. Virginia Tech.

Ruminants and South American Camelids

Goiter in a doeling

A premature (approximately 4-month gestation) doeling was submitted for necropsy in good condition with severely enlarged thyroids and a history of multiple other kids being born with similar conditions. The thyroids bilaterally were 2-3 inches in diameter. This condition is called thyroid hyperplasia or Goiter and is caused by an iodine deficiency. This is common in small ruminants in iodine-deficient areas. When the case is mild, treating with iodized salt can help to resolve the goiter and clinical signs associated with it, however many die before or soon after birth due to this condition. Causes of iodine deficiency include insufficient intake from the pasture, as well as goitrogens which are substances in feed that inhibit the ability the body to utilize dietary iodine. These have been detected in some forage crops and legumes but are often not considered to be a significant cause of goiters. The thyroid produces hormones which are essential for the development of fetuses and are essential to newborn lamb and kid survival. Iodine is lowest on pasture during the winter which is often when it is needed most for pregnant animals. It is recommended to have a

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balanced ration, provide ionized salt licks, mineral supplements or drench animals as needed.

Jamie Horstmann, DVM. Harrisonburg Regional Animal Health Laboratory

Polioencephalomalacia in an alpaca

A 19-year-old male alpaca suddenly developed a wobbly gate, became blind, went down, and eventually died despite medical therapy. No identifiable lesions were present in the brain or spinal cord on gross examination. Microscopically there was widespread laminar cortical necrosis of the cerebrum, which was diagnostic for polioencephalomalacia. Although South American camelids are considered to be more resistant to polioncephalomalacia compared to ruminants, polio in llamas and alpacas has been associated with carbohydrate rich rations, abrupt changes in diet, or heat stress which are thought to culminate in either increased forestomach thiaminase producing bacteria or decrease in thiamine producing bacteria (Aust Vet J 74(5) Nov 1996). Ingestion of thiaminase (usually from thiaminase containing plants) or inability to absorb thiamine through the gut wall due to gastrointestinal disease are also reported risk factors, though neither of these factors were identified in this case.

Thomas E. Cecere DVM, PhD, DACVP. Virginia Tech

Leukosis in a cow

A 4-year-old female angus cow was submitted for necropsy after being found dead on pasture. She was pregnant and had been losing weight over the past couple months. On necropsy she was a BCS 3/9, approximately 4 months pregnant with moderately enlarged cranial lymph nodes and a severely distended abomasum. When opening the abomasum all mucosal folds were significantly enlarged and thickened with minimal to no contents within. This confirmed a suspicion of lymphoma. Lymphoma in cattle occurs spontaneously or it can occur secondary to being infected with bovine leukemia virus (BLV). BLV is caused by a retrovirus and the prevalence of BLV varies but recent studies have estimated that 94.2% of dairy herds and 46.5% of beef cattle. BLV is transmitted horizontally and close contact between animals that are negative and positive is considered a risk factor. Common practices such as rectal palpation, injections (with the same needle), dehorning, tattooing and blood collection have been known to cause transmission. Vectors such as large biting flies can also transmit the disease and transmission can also occur vertically from an infected dam to fetus via the placenta or colostrum. There is no treatment for this disease and culling positive animals is the recommended procedure for reducing the prevalence within your herd.

Jamie Horstmann, DVM. Harrisonburg Regional Animal Health Laboratory

Mesenteric fat necrosis in an adult cow

A 6-year-old, female Ankole-Watusi cow was diagnosed on postmortem examination with abdominal fat necrosis associated with partial obstruction of the large colon that had led to chronic weight loss, muscle wasting and progressive weakening. The cow had birthed twins in June, 2023, and the debilitating signs started shortly after. The wasting continued until the cow was down in early October, 2023, when she was brought into the Veterinary Teaching Hospital at Virginia Tech. Unsuccessful attempts to assist lifting the patient were made. The cow showed no willingness to attempt to stand and humane euthanasia was performed. The postmortem examination showed a firm, yellow, fatty mass in the mesentery of the spiral colon. The mass was compressing the loops of spiral colon, which caused significant impairment to the passage of food down the intestinal tract, as evidenced by the very distended, food-filled, small intestine and cecum and relatively scant food past the spiral colon. PCR testing of Johne's disease, Anaplasmosis, and Theileriosis were negative. Necrosis of mesenteric fat occurs sporadically in many breeds of cattle. It usually affects individual cows and does not pose a problem to the rest of the herd. However, because of the chronic wasting, it is an important differential diagnosis of diseases that may cause similar clinical signs and are of herd health concern, such as Johne's disease, gastrointestinal parasitism, nutritional deficiencies, and others.

Santiago Diab DVM, DACVP. Virginia Tech.

Coxiella burnetti detected in the placenta of a cow

A placenta from a 9-year-old cow was submitted to our laboratory. The cow had aborted approximately 1 month before her due date, and the placenta showed small areas of necrosis. Our in-house Abortion Panel, which uses real-time Polymerase Chain Reaction to look for 8 different groups of abortion-causing pathogens, detected *Coxiella burnetii*.

C. burnetii is a bacterium that causes disease in many species worldwide. In veterinary medicine, it is most commonly recognized as a cause of abortions in sheep, goats, and, less commonly, cattle. It is also the cause of a zoonotic disease (a disease of animals that can infect humans) known as "Q fever". This case highlights the value of good diagnostics for both human and animal health. Though many abortions do not have an identified cause, ruling out infectious causes is an important part of an abortion workup. Although all abortion cases should be treated as potentially zoonotic, having a pathogen identified should lead to increased measures to prevent further spread and infection.

Taylor Young, DVM and Kara Harrison, MS.
Lynchburg Regional Animal Health Laboratory

Avian

Dilated cardiomyopathy in a chicken

A 6-month-old chicken presented to the referring veterinarian with a history of acute-onset respiratory distress. The bird had been treated by the owner with one dose of fenbendazole. Another bird in the flock also had respiratory signs and a third bird had recently died. The veterinarian recommended euthanasia due to the severity of the respiratory distress. The bird was euthanized and submitted for necropsy. External examination revealed urate and fecal staining of the feathers around the vent and severe coelomic distention. The ventral subcutaneous tissue was expanded by a small amount of yellow-tinged, gelatinous fluid (edema). The coelomic cavity contained 250 ml of yellow, slightly viscous fluid and the pericardial sac contained 5 ml of a similar fluid. The heart was severely enlarged with thin walls and the visceral pericardium was focally adhered to the parietal pericardium near the apex of the heart. The lungs were slightly wet and heavy. Histologic examination confirmed pulmonary edema and thinning of the heart walls with a focal area of epicardial fibrosis. The lung and liver were also congested. These findings are consistent with dilated cardiomyopathy with right sided heart failure and ascites. Dilated cardiomyopathy and ascites are often associated with meat-type chickens selected for rapid growth. Studies of chickens with dilated cardiomyopathy (Olkowski et al, 2020) suggest that both genetic factors (ability of cardiomyocytes to dispose of damaged proteins) and environmental factors (rapid growth, cold stress, hypoxia) contribute to dilation of the heart and subsequent heart failure. Strategies for preventing dilated cardiomyopathy in chickens include restricting food intake and avoiding nutrient-dense grower feed.

Reference:
A. Olkowski, C. Wojnarowicz & B. Laarveld (2020) Pa thophysiology and pathological remodeling associated with dilated cardiomyopathy in broiler chickens predisposed to heart pump failure, Avian Pathology, 49:5, 428-439

Teresa Southard DVM, PhD, DACVP. Virginia Tech.

Companion Animals

Sudden onset of anemia in a cat

A 5-year-old, castrated male, domestic short hair cat presented to the Virginia Tech Community Practice service of the Veterinary Teaching Hospital for a comprehensive oral health assessment and treatment. His physical examination was unremarkable (other than grade ¾ dental disease).

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His complete blood count and chemistry panel were also mostly unremarkable. Four days after the dental cleaning, the patient returned due to lethargy, depression, and for not eating. The physical examination was again unremarkable, but the repeated blood work revealed several abnormalities. His hematocrit dropped from 43 to 16% and platelet count from 286 to 19 x 106/µL. The anemia was mildly regenerative (59 x 106/µL, reference interval > 50 x 106/µL). Total protein, sodium, and chloride also decreased in the 4-day period (from 8.8 to 5.9 g/dL, 163 to 143 mmol/L, and 124 to 110 mmol/L, respectively) and ALT liver enzyme increased from 132 to 708 U/L (reference internal 9-130 U/L). All laboratory findings pointed out to a hemorrhage, possibly due to an acute intoxication, with immune-mediated hemolysis, and infectious agents as additional, less likely, differential diagnoses. However, clinically, there was no evidence of external or internal bleeding. Abdominal ultrasound and empirical treatment with antibiotics and corticosteroids were declined since the patient was an outdoor cat and not amenable to frequent administration of oral medications. Due to the severity of the disease process and difficultly administering medications, humane euthanasia was elected.

On necropsy, two abnormalities were readily identified: approximately 35 mL of blood was found in the abdominal cavity and the liver had a dark red to purple film, firmly attached to the surface of its right middle and caudate lobes. When transected, the organ was cavitated with many pinpoint dark red to purple spots. On histology, the liver had multifocal large dilations of the parenchyma and vascular spaces, and the diagnosis of peliosis hepatis was made.

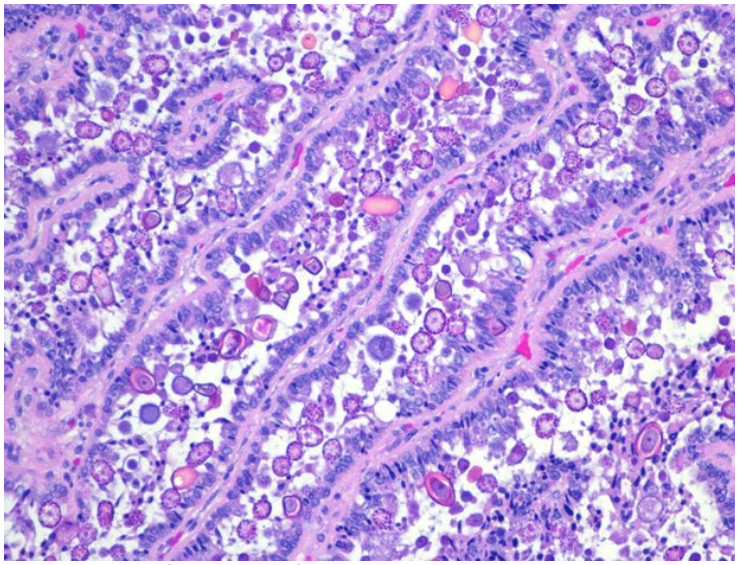
Peliosis hepatis is a rare vascular disease characterized by multiple blood-filled cysts within the hepatic parenchyma. These abnormal structures can rupture and cause death by acute hemorrhage. The cause is unknown. Symptoms can be nonspecific (pallor, jaundiced, weakness, weight loss, dyspnea, abdominal swelling) to absent. Abdominal ultrasound can detect liver enlargement, its abnormal appearance, and abdominal hemorrhage. The onset of signs right after the dental cleaning was likely just a coincidence and confounding factor in this case.

Priscila B. S. Serpa, DVM, MSc, DSc, DACVP (Clinical), Virginia Tech

Hepatic coccidiosis in rabbits

Six rabbits, primarily young postweaning animals, died suddenly within a 3-4-day period, either with no premonitory signs or a short period of decreased appetite and lethargy. These were in a group of approximately 20 rabbits. The owner reported acquiring multiple new juvenile rabbits at an animal swap several days prior to the outbreak. A young adult male rabbit that died spontaneously was submitted for necropsy. Postmortem examination revealed multiple 1-2mm diameter slightly raised white nodules throughout the liver. Based on microscopic examination, the diagnosis was hepatic coccidiosis. Hepatic coccidiosis in rabbits is due to infection with *Eimeria stiedae*, a protozoal parasite that lives within the biliary epithelium of rabbits. Not all rabbits show clinical signs or succumb to the disease; these carrier rabbits remain infected and serve as a reservoir. Stress, such as weaning, transport, or overcrowding can contribute to outbreaks of clinical coccidiosis and death, particularly in young rabbits.

Thomas E Cecere DVM, PhD, DACVP. Virginia Tech



Severe proliferation of biliary ducts, containing numerous apicomplexan parasites in the cytoplasm of epithelial cells and free within the lumen.

It is not a bug!

Pocket pets, including Guinea pigs, rats, mice, hamsters, Sugar Gliders, and hedgehogs, are small mammals that can serve as great pets. They do have unique needs in comparison with dogs and cats and also need regular visits to the vet to ensure they live healthy lives. In case they get sick, complete blood counts may be necessary. Some of these species may have unique features in their blood cells, and here are some observed in Guinea pigs.

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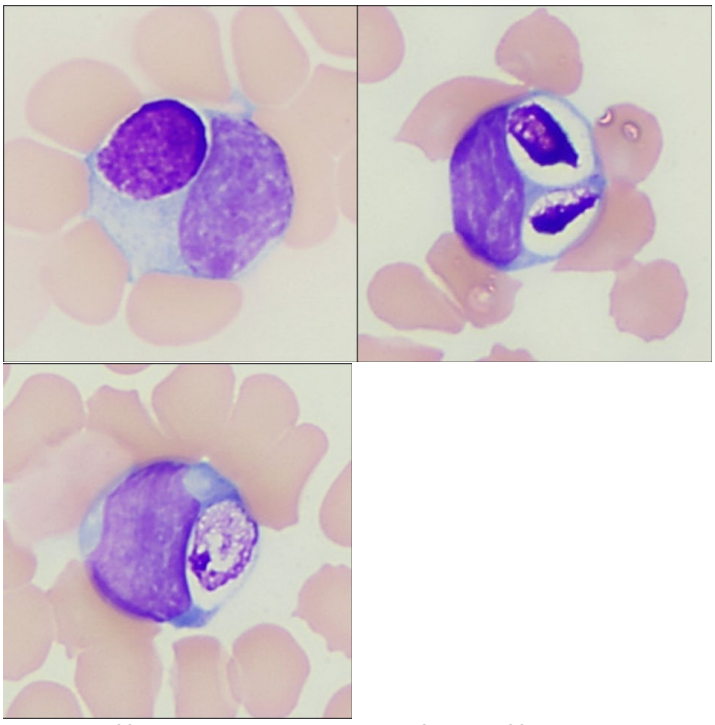
Firstly, instead of neutrophils, guinea pigs have heterophils. Those are the functional counterpart of neutrophils. However, they have scattered small pink granules in their cytoplasm and might look very similar to eosinophils. The latter are usually slightly bigger and have larger, brighter pink granules.

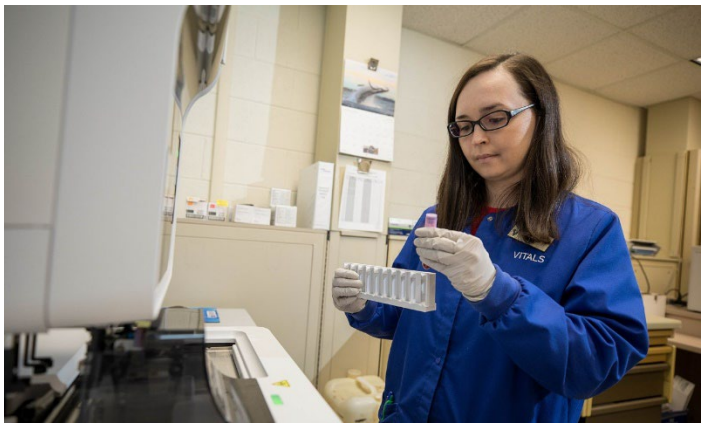
Secondly, Guinea pigs have unique cells called Foa-Kurloff cells or only Kurloff cells (picture). Kurloff cells are specialized mononuclear cells that contain a variably-sized intracytoplasmic inclusion. It is not a bug! It is not an infectious agent! The inclusions are pink to purple, smooth to mildly granular, might be within a colorless vacuole, and often dislocate the nucleus of the cell. They are made of mucopolysaccharides, are more abundant in females than in males, and can count as up to 5% of all white blood cells in the blood of healthy animals. Kurloff cells are believed to function as natural killer cells in Cavian species and might be increased in numbers during estrus, pregnancy, or when there is antigenic stimulation caused by infectious agents.



Three different presentations of Kurloff cells in the peripheral blood.

Priscila B. S. Serpa, DVM, MSc, DSc, DACVP (Clinical), Virginia Tech

LABORATORY NEWS



ViTALS

ViTALS was selected by USDA to represent Region 2 in a partnership between the National Animal Health Regional Network (NAHLN) and the National Bio and Agro- Defense Facility (NBAF). As part of this partnership, ViTALS will host a scientist to assess the risk of emerging diseases in the region and help the lab develop tests capable of detecting these diseases. We so appreciate the recognition that we have the expertise to lead this exciting work!

More big changes are coming to the lab - Carolyn Sink, the Assistant Director, will be retiring after almost 35 years of service to the College. Carolyn has been responsible for bringing ViTALS from an un-named hospital lab in 1989, to the nationally-recognized lab it is today. Her guidance and support have been critical to our success, and she has helped me develop as a faculty member and Lab Director. I will miss being able to knock on her office door, but I appreciate that she will still be available on a part-time basis. Diamond McClendon will be moving from her role as Section Head of bacteriology to Assistant Director, and I am so thankful to have her on board.

Tanya LeRoith DVM, PhD, DACVP. ViTALS Director

VDACS

The VDACS Laboratory System has been working diligently to improve outreach communications and promotions, and to expand and improve testing modalities. We have developed promotional fliers and will continue to work to promote services. Our staff have hosted booths at four fairs this year in Fauquier, Rockingham, Washington and Amherst counties. We are excited to be able to show off our system and what we do!

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Our new fee schedule will go into effect on January 1, 2024. Fees are analyzed through looking at cost of testing supplies and labor, equipment costs, national averages, and government subsidy.

Lab staff in both Lynchburg and Wytheville have been working to bring on NAHLN testing associated with their new Level 1 NAHLN Branch Lab designations. Warrenton successfully passed their NAHLN audit (verbal approval) in October and will be joining as a Level 1 Branch Lab next year!

We are continuing to move through the process of our Capital Expansion Projects affecting three of our facilities- Warrenton, Harrisonburg and Lynchburg. These projects will result in the expansion and minor remodeling of these facilities, and we are excited to see the outcome!

Our quality team is growing! We will soon have an enhanced quality team consisting of a Quality Manager and two regional quality coordinators- one based in Harrisonburg and overseeing quality at both Harrisonburg and Warrenton, and the second based in Lynchburg overseeing quality at both the Lynchburg and Wytheville labs. We are excited to be able to expand our team for a deeper quality focus in standardizing our system.

Necropsy services began again in the Lynchburg Laboratory on November 1st and we are grateful for the opportunity to serve client needs in central Virginia again!

Welcome to VDACS!

- A new laboratory assistant started with us in the Lynchburg Laboratory on October 10th. We are excited to have Caitlin Reichenbach on board to assist!
- A new bacteriologist will be starting at the Warrenton Laboratory on November 25th! We are excited to have Hayley Crossman join our team in that location.
- A new regional quality coordinator will be starting based out of Harrisonburg on December 10th. We are excited to have Sarah Mayer to assist in Quality Assurance!

Jessica Walters DVM, PhD, DACPV. Program Manager, Office of Laboratory Services

LABORATORY LOCATIONS

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