Chronic Wasting Disease Update:

Chronic Wasting Disease (CWD) in deer was first reported in North Carolina earlier this year (March) in northern Yadkin County. Recently, a second positive deer has been confirmed in the same area.

CWD is a fatal and transmissible disease affecting cervids, including white-tailed deer and elk. Abnormal proteins, called prions, spread through the animal’s nervous system slowly, eventually causing spongy holes in the brain that lead to death. The disease spreads among deer through direct and indirect contact from infected saliva, urine or feces of live deer, carcasses, and body parts.

“With deer season opening in less than a month, we wanted to get the news of this second positive out as quickly as possible,” said Brad Howard, chief of the Wildlife Commission’s Wildlife Management Division. “It’s imperative that hunters understand how important it is to submit samples to help determine how prevalent CWD is here in North Carolina. It’s also crucial that we enlist their help to not give the disease a ride to new areas.”

Officials say the biggest message to hunters this season is, “Don’t give it a ride.” CWD spreads via infected saliva, urine and feces of live deer, or the movement of deer carcasses and carcass parts. Even infected deer may appear healthy. Therefore, it is important that precautions are taken when transporting or disposing of deer carcasses.

“CWD is highly transmissible. It’s imperative that if you hunt and harvest deer that you responsibly dispose of deer remains,” said Howard.

Howard suggests hunters follow one of the following disposal methods:

1. Bury the deer remains where you harvest the animal when possible.
2. Double bag deer remains for disposal at the closest landfill.
3. Leave the deer remains on the ground where the animal was harvested.
**Staffing:**

Veterinary diagnostic laboratories are feeling the workforce shortage strain experienced by many fields nationwide. NCVDLS is no exception and we continue to be short-staffed in several laboratory sections. Your patience is appreciated as our dedicated, customer service oriented staff are working harder to complete your laboratory testing, accurately and timely, during this prolonged period of staffing vacancies.

**Annual Survey:**

We are seeking your feedback and comments on our annual Customer Satisfaction Survey. Your participation is very important to us as we work to continually improve our services to the agricultural community, veterinarians, and animal owners of North Carolina. Please take a few minutes to share with us your experiences with our laboratories.

Please click on the following link to participate in our survey:

https://www.surveymonkey.com/r/NVQH9NM

- Jim Trybus, DVM, DAVCP

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**New Toxicology Testing Now Available at Rollins!**

**Toxicology Evaluation (visual/microscopic), $35:** Visual and/or microscopic examination for toxic plants/seeds, insect fragments, blue-green algae (cyanobacteria), foreign bodies (plastic, lead, etc.), visually identifiable pesticides or baits. Suitable samples include rumen or reticulum content (500 g), stomach or cecal content (500 g), small animal ingesta or vomitus (all available), water (50 mL), forage or hay (1-2 flakes).

This test request can mean several different things depending on the sample sent – please specify what your concern is on the submittal form, because that determines how the sample is handled. If cantharidin or lead poisoning is suspected, any insect or battery plate fragments will be lost if the herbivore ingesta sample is processed through sieves as if to find toxic plant material.

Most often the Toxicology Evaluation test is used to determine whether toxic plants or seeds or some kind of visually identifiable pesticide bait or foreign object was eaten near the time an animal died (rumen content, stomach content). If lead poisoning in a ruminant is suspected, reticulum content can be examined to determine possible ingestion. If fatal blister beetle poisoning is suspected, stomach or cecal content can be searched for insect fragments. Hay or forage that looks ‘clean’ may be contaminated with potentially toxic plant material. Water samples can be checked microscopically for blue-green algae (cyanobacteria). (This is not an analytical chemistry test for the algal toxins themselves.) Recognition of visually identifiable materials is based on years of diagnostic Toxicology experience, lots of reference books, computer image searches and a large library of microscopic images. In some cases, the findings of a Toxicology Evaluation may indicate an appropriate course of action for further Toxicology testing (for example, cantharidin, lead, strychnine, microcystin, antifreeze ...) and avoid other expensive tests. This is a fit-for-purpose test – sometimes it may help find a diagnosis.
Calculi Identification (FTIR), $25: The percent composition of biologically generated stones (usually 1-3 components) is determined using Fourier-transform infrared spectroscopy (FTIR) by chemical matching against validated spectral libraries based on the sample’s characteristic infrared spectrum. The primary component will be reported along with any additional components and their percentages. This test can be applied to any biological concretion but is usually used to identify kidney and bladder stones so they can be avoided in the future. Please rinse the stones send them dry. Do not send them in formalin. Results are available in 3-5 days.

Toxic Plant Identification (visual/microscopic), $15: The more complete a plant is, the more likely it can be identified properly. Flowers and fruits are very helpful if present. Recognition of visually identifiable toxic plants is based on years of diagnostic Toxicology experience, botany training, a good working relationship with the NC Botanical Garden and Horticulture staff, lots of reference books, computer image searches and a large library of microscopic images. Plants that are not known to be toxic may not always be identified. Submissions without enough identifying characteristics may not be identified. Results available in 1-5 days.

For mushroom identification, please contact your regional mushroom club or mycological society (found at https://namyco.org/clubs.php; e.g., Asheville, Charlotte, Piedmont Triad). If toxic mushrooms are suspected in a veterinary emergency, the animal owner should photograph the mushrooms in their surroundings before collecting anything. Specimens at several growth stages can help with identification. Once imaged in place, harvest one mature specimen (including any underground parts) and photograph the underside. Cut the mushroom in half lengthwise through the cap, stem and foot and photograph the cross-section. If the animal vomited something up, that should also be photographed. Upload the images to the Poison Help; Emergency Identification for Mushrooms & Plants global facebook group at https://www.facebook.com/groups/144798092849300/ and include the geographic location, species affected, and any clinical signs. They will provide identification only. ASPCA uses this group to confirm identities as needed. From my own observations over the past 2 years, results are usually pretty quick. Once identified, if you need help with determining toxic potential and potential clinical signs, please email catherine.barr@ncagr.gov and I’ll get back to as soon as I’ve verified the information.

If you have questions about any of these tests, or about suspected poisonings or nutritional cases, please feel free to email Dr. Cat Barr in Toxicology at the Rollins lab (catherine.barr@ncagr.gov).
Ulcerative enteritis (UE) is an infectious condition of the intestine of many bird species including bobwhite quail and other game birds such as pheasants and grouse, along with chickens and turkeys. Ulcerative Enteritis of quail is a highly contagious disease caused by the bacterium *Clostridium colinum*. Although *Clostridium colinum* causes mild ulcerative enteritis in chickens and turkeys, when severe ulcerative enteritis or ulcerative enteritis-like conditions are diagnosed in birds other than quail, such as chickens or turkeys, it is typically associated with *Clostridium perfringens*. In chickens, it is more properly called necrotic enteritis and often also associated with the presence of coccidiosis in those birds. Our focus for this article is on Ulcerative enteritis of Quail or “Quail Enteritis”. This disease has high mortality, up to 100% in quail. In quail, clinical symptoms are acute and commonly associated with diarrhea, severe hemorrhage in the droppings or sudden death (see fig. 1). Quail can become more susceptible to UE if coccidiosis is present. Other factors that effect quail susceptibility to UE are environmental stressors or overcrowding. Cross contamination of feed, water and bedding can play a role in transmission. UE occurs world-wide in quail, but to put it into a more home-grown perspective, NCVDLS diagnosed 55 quail flocks with UE out 153 quail flocks evaluated over a recent five-year period or 1/3 of the quail examined, indicating an average of eleven quail flocks presented to NCVDLS with UE a year in North Carolina. As of the writing of this article, there are 431 licensed game bird propagators in North Carolina according to the NC Wildlife Resources Commission. So, UE has the potential for significant economic impact on the game bird industry in NC. UE is not just a threat to game birds in confinement, but also game birds in the wild.

Ulcerative enteritis can present acutely with symptoms of sudden death, especially in young birds or more chronically with signs of lethargy, muscle wasting, weight loss and diarrhea in more mature birds. UE is spread by fecal oral transmission, so birds become infected by ingesting contaminated feed, water, litter, or dirt. Birds which survive UE can become chronic carriers and spread disease. When birds become exposed to the bacteria *Clostridium colinum*, causing UE, the incubation time until clinical disease is 1-3 days. The disease process can run through an entire group within three weeks, depending on management style and the size of the flock.

Coccidiosis is the primary differential diagnosis in quail. In other species of birds, such as chickens and turkeys differentials include necrotic enteritis, coccidiosis and Histomoniasis. Diagnosis can initially be made based on gross necropsy lesions noted on necropsy (fig. 2). Hemorrhagic enteritis of duodenum and jejunum will be grossly evident in acute cases.
Figure 2 Erosive caseous lesions; courtesy of Dr. Aziz

More advanced cases will demonstrate classic multiple rounded coalescing ulcers with yellow caseous exudate coverings of the intestinal mucosa, which are visible through the serosa. Bloody contents are typically noted in the intestinal lumen. Lesions of the liver are sometimes noted grossly as miliary to mottled areas of necrosis. The lesions of the liver are not common in quail. Bacteriology is not consistently reliable to diagnosis UE in quail as *Clostridium colinum* is typically difficult to culture for identification.

PCR testing for *Clostridium colinum* where available is a reliable tool for diagnosis. Histopathology can often be used for confirmation of the diagnosis for UE and eliminate the other differentials (see fig. 3). The early lesions reveal congested mucosal erosions with loss of villi and heterophilic infiltration of the lamina propria to the submucosa. More advanced lesions will extend into the submucosa and contain caseous exudate and debris. These erosive lesions and ulcers often perforate the intestinal wall and result in severe peritonitis and death.
Prevention of the disease is important. Good management of game birds include limiting overcrowding in pens and overgrazing of fields and pastures. Strict biosecurity measures should be practiced consistently even in field setting where possible. Dead birds should be removed from the flock immediately. Birds surviving previous outbreaks should be worked out of farm or culled where possible and at minimum not mixed with new non-diseased birds. There are treatment plans which include various antibiotic protocols systematically used in feed or water, which is a topic for another article.

References:
1. Disease of Poultry 13th Edition [Book], AAAP, Singapore Markono Print Media Pte Ltd 2013 Pages 944 -949
2. Aziz and Barnes: Gross Pathology of Avian Diseases; Text and Atlas, AAAP, Jacksonville, FLA AAAP Inc. 2018
4. NC Wildlife Resource Commission

Figure 3 Mucosal ulceration of quail intestine; courtesy of Dr. Aziz
Listeria monocytogenes Metritis with Hepatic Coccidiosis

By: David Drum, DVM

The body of a 22 week old female rabbit was presented to the animal disease diagnostic laboratory for post mortem examination. The provided history stated owner reported they felt the rabbit was doing fine and then 24 hours later was found deceased.

On post mortem examination of the body, the rabbit weighed 1.84 kg, was of lean body condition and there was at least a moderate degree of post mortem change to the body. There were multifocal ~ 3mm wide pale foci throughout the lung and liver tissues. The uterus was markedly distended (figure 1) with firm, white colored, semi-desiccated exudate. No additional diagnostic lesions on examination of the body.

Figure 1 Distended uterus
Based upon gross post mortem lesions, the morphologic diagnosis was pyometritis with pulmonary and hepatic microabscessation. The lesions were indicative of a bacterial infection of the uterus that spread systematically.

On aerobic bacterial culture, \textit{Listeria monocytogenes} was isolated from the liver tissue and a culture swab of the uterine contents.

Histopathologic examination of submitted tissues was diagnostic for 1) Severe heterophilic placentitis and transmural metritis with luminal fetal tissue (fetal resorption), 2) Multifocal moderate subacute heterophilic embolic pneumonia, 3) Multifocal moderate chronic granulomatous cholangitis with numerous intraluminal oocysts and 4) Multifocal random subacute hepatocellular necrosis with mineralization.

The diagnosis for this case was \textit{Listeria monocytogenes} metritis with hepatic coccidiosis

Listeriosis in rabbits is characterized by abortions and sudden death, particularly in does during advanced pregnancy. In sporadic outbreaks of the disease, the source of the organism is frequently attributed to contaminated feed or water. Carrier and shedder animals can also occur. The organism has a particular predisposition for the gravid uterus in advanced pregnancy. Abortions, stillbirths and mortality of the dam is the usual outcome. In animals that survive; uterine infection may persist post kidding and serve as a source of infection of the following pregnancy.

As previously stated, mortally of the doe during late pregnancy is common. On post mortem examination, typical lesions include straw colored peritoneal effusion, disseminated pale military foci of necrosis in the liver, edema of regional lymph nodes and visceral congestion. The uterus may contain relatively intact, near term kits or fetuses in various stages of decomposition or mummification. Differential diagnosis for these type of lesions should include other disease that cause disseminated foci of hepatic necrosis such as Tyzzer's Disease, Tularemia or Salmonellosis. Perinatal deaths of does can also occur with acute Pasturellosis and metritis, but the liver lesions are normally absent with these types of infections. – Source \textit{Pathology of Laboratory Rodents and Rabbits} by Percy and Barthold.
Ammonia-induced Corneal Erosions

By: Heather Wyss, BVSc

Ten, 15 day old broilers were presented to the animal disease diagnostic laboratory for necropsy examinations for an acute onset of blindness. Of the ten birds, six of them had severe blepharospasm. The eyelids of two of the birds were crusted shut with dried serous discharge. Dried serous discharge was also present peri-orbitally in a few of the birds. The epithelium of the central corneas were roughened and irregular in appearance. No other gross lesions were present.

Histopathology showed corneal epithelia erosion and detachment with bluish thickening of the basement membrane due to calcium deposition and mild heterophilic conjunctivitis with swelling and hyperplasia of the conjunctival epithelial cells. Subconjunctival edema was also seen.

Ammonia in a poultry house comes from the birds themselves. An ammonia concentration above 25 ppm will have adverse effects on the health and performance of the birds.

Factors that can affect the ammonia concentration include the type of bedding substrate used, the frequency of manure removal, humidity levels, ventilation rate, the number of chickens in the house, feeding high protein diets, and the pH of the manure produced.

Strategies to prevent ammonia-induced injury includes feeding a balanced diet, keeping an appropriate stocking density, proper ventilation, and good litter management.
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