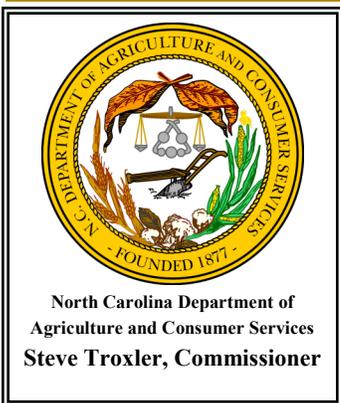


The NCVDLS REPORT



Accredited by the American Association of Veterinary Laboratory Diagnosticians

Veterinary News and Information From North Carolina's Diagnostic Laboratories



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

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In This Issue...

Feature Article	2
Short Cuts	3
Departmental News	10
Directory	11

Holiday Closings...

- April 3, 2015
- May 25, 2015
- July 3, 2015

Our laboratories will be closed on the above listed days.

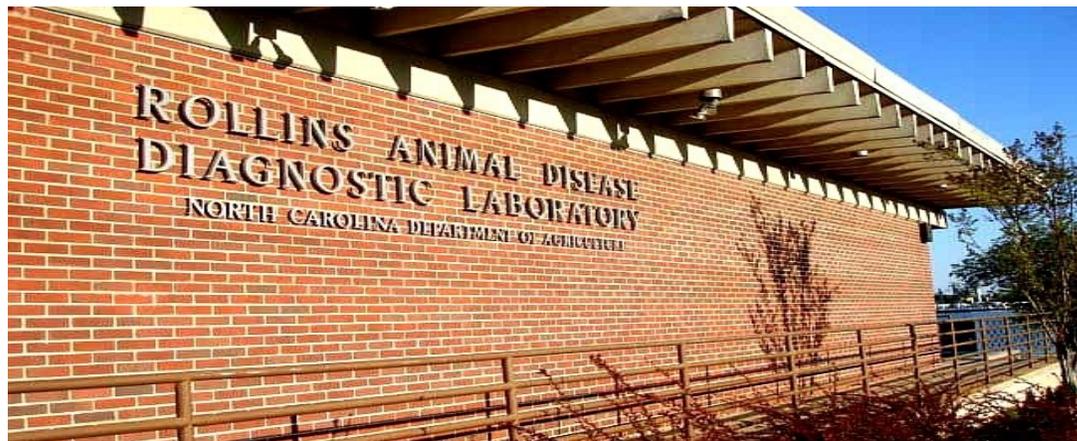
Please e-mail NCVDL@ncagr.gov with any comments and/or suggestions concerning The NCVDLS Report

Editor - Dr. David Drum

Message from the Director

On November 3, 2014, Commissioner Troxler named Dr. R. Douglas Meckes as the new State Veterinarian. In this capacity, Dr. Meckes serves as Head of the Veterinary Division which includes the Diagnostic Laboratory System. Dr. Meckes comes to the N.C. Department of Agriculture & Consumer Services from the U. S. Department of Homeland Security. Dr. Meckes received both undergraduate and Doctor of Veterinary Medicine degrees from Auburn University. He spent nearly 30 years in private mixed-animal practice in Apex before moving to Washington, D.C. to serve as a congressional fellow for Sen. Chuck Hagel. At the conclusion of his fellowship, he worked as the Assistant Director of Government Relations for the American Veterinary Medical Association. He then joined the Department of Homeland Security and worked in the Office of Health Affairs as Chief of the Food, Agriculture and Veterinary Defense Branch. Please join me in welcoming Dr. Meckes back "home" to North Carolina.

Karen W. Post DVM, MS



Feature Article

Epizootic Hemorrhagic Disease

Alison Tucker, MA, VMD, Dipl ACVP

A captive cervid herd had over 60% losses during a 5-week period in late August and September. According to the history provided, the deer would have an acute onset of clinical signs and would be found standing in a pond or dead near the pond. Tongues were swollen and blue. Deer treated with anti-inflammatory medication and antibiotics had limited response to therapy. Animals submitted for necropsy were adults in good physical condition. On necropsy there were petechiae and ecchymosis in the subcutaneous tissue, skeletal muscle and mucus membranes. Petechia and ecchymosis were present in the lungs, on the epicardial and pericardial surfaces, on the splenic capsule and in the trachea and esophagus. Paintbrush to diffuse hemorrhages were distributed throughout the gastrointestinal viscera. Tongue and oral mucosa were dark purple and had multiple erosions. There was mild to moderate pericardial accumulation of clear, watery fluid. Tissue samples submitted for virus isolation included lung, liver, spleen. Viral cytopathic effect was observed with Vero cells inoculated with spleen. PCR at NVSL confirmed Epizootic Hemorrhagic Disease virus serotype 6.

Epizootic Hemorrhagic Disease and Blue Tongue are diseases of wild and domestic ruminants caused by a group of closely related orbiviruses. Epizootic Hemorrhagic Disease is widespread in white-tailed deer in North America and causes the most severe disease in this species. Transmission is by *Culicoides* sp, a biting midge, and outbreaks are caused by exposure to the midge rather than deer to deer transmission. The disease can present in the peracute to acute form, such as seen in these specimens, or in the chronic form where damage to hooves and distal limbs can be seen. The incubation period is 5 - 10 days. In acute cases, the disease progression from onset of clinical signs to death is short, approximately 8 - 36 hours. Viral infection of endothelial cells and subsequent vascular damage results in the vascular leakage with hemorrhage and edema that are the hallmark of acute infection. If the animal survives, tissue ischemia and necrosis subsequent to the vascular damage have long-term effects seen in the chronic form.

Epizootic Hemorrhagic Disease serotypes 1, 2 and 6 have been identified in wild deer in North and South Carolina in 2014, according to the Southeastern Cooperative Wildlife Disease Study and have been identified in wild deer in the central US according to NVSL.

For additional information, see

http://www.cfsph.iastate.edu/Factsheets/pdfs/epizootic_hemorrhagic_disease.pdf

<http://www.ncwildlife.org/Hunting/AftertheHunt/DeerDiseases.aspx>

<http://www.vet.uga.edu/scwds/>

Short Cuts

COMPANION ANIMAL

Feline

A 2.5 year old female spayed indoor DSH was submitted. External exam of the 5.2kg 2.5 year old animal revealed hyperemic mucous membranes, a small amount of hemorrhage from the oral cavity and a BCS of 7/9. The lungs were firm and rubbery on palpation and had a white sheen to the pleurae with areas of petechia and palpable nodules ranging in size from .25-.5cm in diameter. Cut surface of the lobes revealed tan mucus filled airways. A small amount of hemorrhage was identified in the trachea. The heart weighed 19g and the myocardium was hyperemic. Three adult *Dirofilaria immitis* were evident in the right atria (Figure 1). Histopathology sections identified eosinophilic endarteritis in the lungs that was characterized as proliferative and villous, chronic, multifocal, moderate to marked. Lung lesions included an eosinophilic, histiocytic, and lymphoplasmacytic pneumonia that was characterized as diffuse, moderate to marked with occasional granulomas and diffuse, moderate pulmonary fibrosis and smooth muscle hypertrophy. A lymphocytic myocarditis was evident. Hepatic centrilobular cord atrophy and degeneration were also noted on histo. The findings in the lungs were likely secondary to the **heartworm disease**. The centrilobular cord atrophy and degeneration observed in the liver is likely a result of hypoxia from the severe pulmonary disease. Significant disease can occur in cats with a very small number of heartworms due to the small size of the cat's heart.

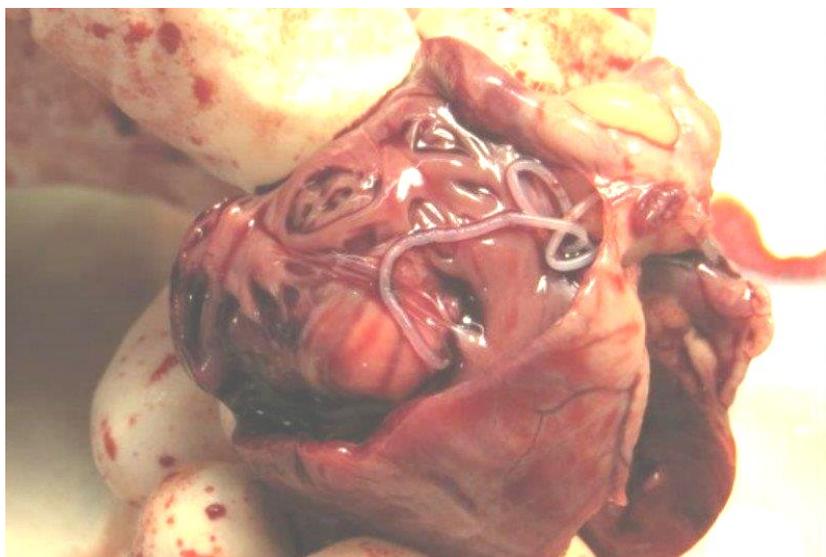


Figure 1: *Dirofilaria immitis* present in the right ventricular lumen

Dr. Kim Hagans

COMPANION ANIMAL, CONTINUED

The body of a 1 year old neutered male domestic short haired cat was presented for post mortem examination. The provided history stated the cat was euthanized and the referring veterinarian reported a week long history of inappetence, weight loss, and lethargy. Blood work showed hyperbilirubinemia, elevated amylase and globulins. The cat was alert, but dehydrated and febrile. Pancreatitis test was negative. Feline Infectious Peritonitis screening test negative. They treated the cat with intravenous fluids and antibiotics. The cat began to have seizures and developed ataxia. They treated the cat with Metronidazole, Clavamox, and Baytril, but the seizures progressed.

On post mortem examination, the cat weighed 3.60 kg, and had a BCS of 2.5/5. The heart weighed 14.9 grams. The lungs were purple to red in color, rubbery on palpation with < 1 mm wide white foci throughout the lung tissue. There were coalescing 2+ mm wide white foci throughout the liver tissue. The urine tested negative for glucose and ketones using dip stick testing. There was a 1.5 cm by 1.5 cm slightly raised, white colored mass on the medial surface of the left kidney and a 0.5 cm by 0.5 cm similar mass on the caudal pole of the right kidney. Multiple enlarged mediastinal and bronchiolar lymph nodes. Based upon the gross necropsy finding, Lymphoma was initially suspected.

Brain tissue was submitted for Rabies testing, and was negative.

Histopathology revealed disseminated (Lung, Liver, Kidney, Intestine, Pancreas and Lymph Node) pyogranulomatous inflammation with vasculitis.

Sections of liver were positive for immunoreactivity to the Feline Coronavirus by immunohistochemistry tests.

The diffuse pyogranulomatous inflammation coupled with the positive Feline Coronavirus test were diagnostic for **Feline Infectious Peritonitis virus infection (FIP)**.

This is one of those cases where the in-clinic test for FIP was negative, yet the cat has advanced evidence of the viral infection.

Dr. David Drum

LIVESTOCK

Cattle

A 5 month old Holstein calf was submitted. The owner reported the calf had been bottle fed but was turned out with three Angus heifers 3 weeks ago. Three days ago he noticed the calf was standing in one spot and not opening its mouth. It was unstable and moving its head from side to side. The veterinarian examined the calf and treated it with an antibiotic, Thiamine and an anti-inflammatory. The calf was dead the next morning. The 118kg, 5 month old Holstein calf was submitted and external exam revealed mild dehydration and mild to moderate autolysis. The cerebral spinal fluid was dark straw in color. The brain was submitted for Rabies and after a Negative result was reported, the brain was examined further. The meninges were hyperemic and congested. Sections of cerebrum were swollen with flattening of the gyri and multiple sections of cerebrum fluoresced under ultraviolet illumination (Figure 1). No other abnormalities were identified on gross exam.

The fluorescence under ultraviolet illumination along with histopathology lesions of marked acute laminar cortical necrosis in the brain was diagnostic for **Polioencephalomalacia**.

LIVESTOCK, CONTINUED

Polioencephalomalacia is associated with altered thiamine status or increased sulfur intake. Young animals are more commonly affected than adults. Treatment with Thiamine must be started early in the clinical course to see improvement in clinical disease



Figure 1: Cerebral Fluorescence under ultraviolet illumination

Dr. Kim Hagans

Equine

A 25-year-old female Welsh pony developed colic and progressively deteriorated over the next 24 hours. An impaction in the left ventral quadrant was suspected. The horse was euthanized. This horse had a previous colic episode 3 to 4 weeks earlier. On gross examination, the left and right thorax contained 1 liter and 2 liters yellow fluid with abundant chunks of fibrin, respectively. Variable amounts of fibrin were diffusely adhered to the costal, diaphragmatic and pulmonary pleura. A 23 cm section of the distal esophagus was markedly thickened and measured 8 cm in diameter. There was marked hypertrophy of the esophageal smooth muscle that measured 2.5 cm in thickness. The mucosa appeared normal with exception of a 4 cm perforation located along the left distal esophagus that measured 3 cm from the gastroesophageal junction. (Figure 1) There was marked inflammation along the adventitia of the esophagus, characterized by abundant fibrin, petechiation, necrosis, and adherence of forage particles to the surface.

The cause of discomfort in this horse was a result of an **esophageal perforation with bilateral pleural effusion and severe pleuritis**. Histopathologic examination findings were reflective of the necropsy findings. There was a focally extensive transmural fibrinosuppurative and hemorrhagic esophagitis with intralesional plant material and myriad bacterial colonies. Also associated with the perforation was mild to moderate myocyte degeneration and necrosis and mild muscularis fibrosis. The exact cause of the perforation was unknown. Given the history of prior colic and the chronicity of the esophageal lesion, a previous choke episode could not be ruled out. **Esophageal muscular hypertrophy** has been commonly reported in horses and is usually an incidental finding. The cause of the hypertrophy is unknown.

LIVESTOCK, CONTINUED

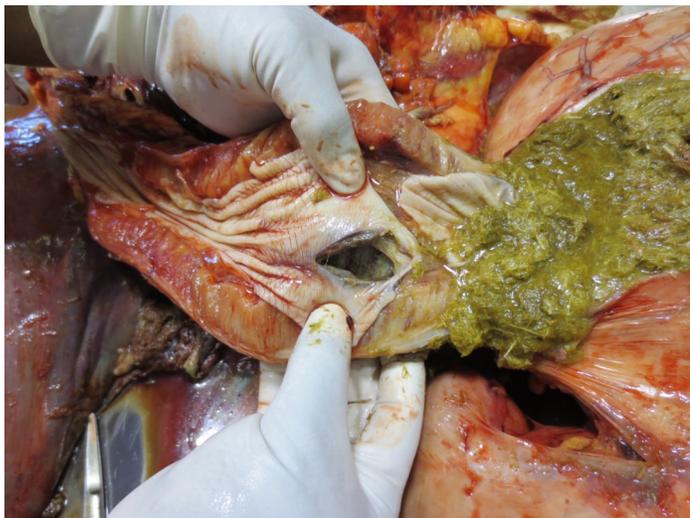


Figure 1: Esophageal perforation; Note the marked hypertrophy of the smooth muscle.

Dr. Mahogany Wade

Porcine

An organic pig producer purchased four 4-month-old crossbred pigs from a random source. One week later, coughing was observed in the pigs. An improvement was initially seen following treatment with Enrofloxacin and Liquamycin; however, all four pigs died around one week post-treatment. One female pig was submitted for necropsy. Necropsy examination revealed moderate multifocal to coalescing fibrosis of the capsular surface of the liver (interpreted as milk spots). On multiple cut sections, several blood vessels contained roundworms. (Figure 1) There was moderate edema and hemorrhage of the hepatic lymph nodes. The common bile duct was markedly distended with 24 roundworms; several worms had also migrated into the hepatic ducts (Figure 2). The bile duct epithelium was diffusely hyperemic with multifocal to coalescing fibrinous plaques. (Figure 3) The stomach and small intestines contained 10 and 90 roundworms, respectively. Approximately 95% of the cecal and colonic mucosa was markedly effaced by multifocal to coalescing ulcers with abundant fibrinonecrotic debris. (Figure 4) Many of the ulcers had dark brown to black umbilicated centers surrounded by fibrinonecrotic debris along the edges. Some of the ulcers penetrated beyond the submucosa, which corresponded to circular hyperemic to white lesions identified along the serosa. Other gross findings included lungworms in one of the bronchioles and mild serous fat atrophy of the heart and bone marrow.

Histopathologic examination revealed multifocal suppurative and lymphoplasmacytic cholangiohepatitis with biliary hyperplasia, bile duct ectasia, and intraductal nematodes (roundworms), mild hepatic fibrosis, fibrinosuppurative cholecystitis with intralesional nematodes eggs that were consistent with **ascariasis**. Also identified was marked multifocal fibrinosuppurative and ulcerative **typhlocolitis**, and multifocal fibrinosuppurative, granulomatous, and eosinophilic bronchointerstitial pneumonia with intra-bronchi and intra-alveolar nematodes (lungworms).

The cause of death and debility in this pig was due to a combination of parasitism, typhlocolitis, and malnutrition. A bacterial etiology was suspected as the cause of typhlocolitis; the "button ulcers" were highly suggestive of salmonellosis. No organisms were isolated on bacterial cultures, which was likely due to recent antibiotic therapy. Ascariasis, a disease caused by roundworms (*Ascaris suum*), can cause pneumonia, hepatitis, and ill thrift. Metastrongylosis is a disease caused by lungworms (*Metastrongylus* sp.), which can cause chronic coughing and may lead to generalized unthriftiness. These two parasitic infections can occur concurrently in heavily contaminated pastures; this a reminder of the need for parasite control on organic pig farms.



Figure 1: Liver; intravascular roundworms

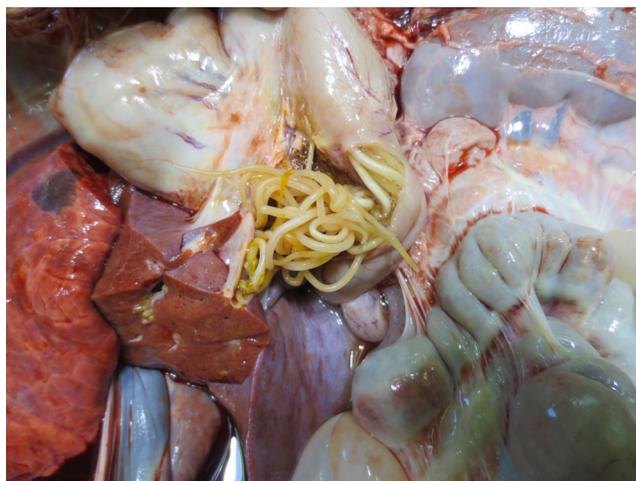


Figure 2: Common bile duct; marked distension with abundant roundworms



Figure 3: Common bile duct; fibrinosuppurative cholecystitis



Figure 4: Cecum, colon; marked fibrinosuppurative and ulcerative typhlocolitis

Dr. Mahogany Wade

LIVESTOCK, CONTINUED**Caprine**

A 3 year old Nubian doe was submitted with a history of arriving on the farm for breeding 3 weeks earlier. The doe went off feed, had diarrhea and was treated with 12 ml of Pepto-bismol® one morning and died later that day. She was on mixed grass pasture and had not been vaccinated. The 65kg 3 year old Nubian doe was submitted and external exam revealed obese body condition. The rumen was distended with forage and grain was visible. The rumen pH was 6.0 and microbes were nonmotile. Large hard abdominal, omental and perirenal adipose deposits were evident. The right adrenal gland was hemorrhagic. Mucosal fold hyperemia was noted in the abomasum. The small intestines were thin walled and autolytic with a green tint and red green liquid filled the lumen. The cecum was very distended with tan red liquid and the mucosa was thin. The mesenteric lymph nodes were pale, enlarged and edematous. No feces remained in the distal colon or rectum. The spleen was mildly congested. No urine remained in the bladder. The liver and kidneys were hyperemic. The lungs were pink with small areas of mild congestion. The myocardium was hyperemic. The meninges were congested. Bacterial cultures of the large intestine isolated heavy growth of *Clostridium perfringens*. This isolate was genotyped by multiplex PCR as **Type D** which is associated with Enterotoxemia in goats. This infection is preventable with proper vaccination.

Dr. Kim Hagans

Wildlife and Exotics

Two deceased rabbits were recently submitted to the Griffin Laboratory. The submitting small meat rabbit operation of approximately 75 rabbits had experienced illness and death of 12 adult and 2 young rabbits over a 2 to 3 week period. Lethargy and diarrhea had been observed in many of the affected rabbits before death.

The rabbits examined were adult does which had been nursing average size litters of healthy kits. Greenish-brown watery feces stained the fur around the rectum and on the hind legs of one of the does. The most significant gross necropsy finding was thick, gelatinous mucus distending the colon (Figures 1 and 2).

Goblet cell hyperplasia was found in intestinal histopathology sections. The findings were characteristic of **Mucoid Enteropathy**.

Mucoid Enteropathy is a disease of rabbits characterized by the accumulation of mucus in the intestines. Lethargy, anorexia, diarrhea and bloating of the abdomen are often observed. Dietary changes and other various predisposing factors have been associated with this disease. The commercial pelleted rabbit feed had been changed about 2 weeks prior to the disease onset.



Figure 1: Gelatinous mucus distending the colon



Figure 2: Gelatinous mucus distending the colon

Dr. Reginald Ridenhour

POULTRY

The body of a 6 year old female Black Silkie chicken was presented for post mortem examination. The provided history stated the bird had “cauliflower like” tissue build up on the face and it was spreading fast. The changes started 2 weeks ago and has gotten worse over the last week. This bird had been eating yesterday and laid an egg a few days ago. The owners lost another bird to the same condition a week previously. They obtained 8 new birds in the spring. The birds were fine until 2 weeks ago. The owner reported a number of the birds in the flock “just don't look well”. Another bird in the flock had black scabs on the comb, and swelling around the eyes. On post mortem examination the bird weighed ~ 850 grams and was of thin body condition. Brown to tan colored, crusty, raised papillomatous masses surrounded the eyes; and spread over the sides of the face, under the throat, then around and over the back of the head and neck (Figure 1). There were also similar lesions on the inside surface of the wings (Figure 2). A raised mass was seen on the back of the tongue and in the area of the choana.

Histopathological lesions included - Skin: Marked epidermal hyperplasia with hydropic swelling of epidermal cells and intracytoplasmic inclusion bodies characteristic of poxvirus infection. A thick crust of proteinaceous and necrotic debris containing enormous numbers of bacteria on the skin surface. Loss of the epidermis in some areas, with a thick crust covering the underlying dermis, which had marked morphonuclear inflammatory infiltrates. No lesions, or changes of diagnostic significance were seen on examination of section of other submitted tissues.

This was a case of **Fowl Pox Virus**.

POULTRY, CONTINUED



Figure 1: Lesions on the head and face



Figure 2: Lesions under the wings

Dr. David Drum

DEPARTMENTAL NEWS

ROLLINS LABORATORY

Rollins Laboratory New Hires:

Ashlee Zack, Medical Laboratory Technologist II, Molecular Diagnostics

Excel Swann, Medical Laboratory Technologist I, Virology

Dr. Neeti Dahal, Medical Laboratory Technologist II, Molecular Diagnostics

Talley Ouro, Business & Technology Applications Analyst

Tina Buffington, Virology Laboratory Section Supervisor

Younghee Lee, Medical Laboratory Technologist II, Molecular Diagnostics

NORTHWESTERN LABORATORY

New Hires at the Northwestern Laboratory:

Cindy Smith, Processing Assistant IV

CE ATTENDANCE

Drs. Post, Mock, Ridenhour, Rushton and Trybus attended the AAVLD / USAHA Convention in Kansas City, KS during October 16-22, 2014.

Dr. David Drum attended the Veterinary Biomedical and Diagnostics Meeting at the University of Tennessee in Knoxville, TN on December 4, 2014.

DIRECTORY

Rollins Laboratory - 919-733-3986

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Raleigh, NC 27607

Director, NCVCLS

Dr. Karen Post

Assistant Director, NCVCLS

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Dr. James Trybus (Pathology Services Coordinator)

Dr. Tahseen Abdul-Aziz (Avian)

Dr. Alison Boone (Anatomic)

Dr. Steven Rushton (Anatomic)

Dr. Alison Tucker (Anatomic)

Veterinary Diagnosticians

Dr. Mahogany Caesar

Dr. Jennifer Haugland

Dr. Stacy Robinson

Microbiologists

Dr. Karen Post

Dr. Richard Mock

Dr. Chad Cecil

Laboratory Section Supervisors

Tina Buffington—Virology

Sandy Murphy—Bacteriology

Mary Baker—Histopathology

Dr. Kristen Crook—Serology

Beverly Wood—Molecular Diagnostics

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Veterinary Diagnostician

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Resident Director

Dr. Kim Hagans

Veterinary Diagnostician

Dr. Reg Ridenhour

Diagnostic Laboratory Advisory Committee

Dr. Allen Cannedy	Small Ruminant/Camelid Practitioner
Dr. Eric Gonder	Corporate Poultry Practitioner — Butterball (turkeys)
Dr. Jennifer Haugland	Veterinary Diagnostician — NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Shannon Jennings	Corporate Poultry Practitioner — Nash Johnson Farms (chickens/turkeys)
Dr. Randy Jones	Private Veterinary Practitioner — Livestock Veterinary Services (swine)
Dr. Richard Kirkman	Private Veterinary Practitioner — Large Animal
Dr. R. Douglas Meckes	State Veterinarian — NCDA&CS Veterinary Division
Dr. Karen Post	Director of Laboratories — NCVCLS
Dr. Rick Sharpton	Corporate Poultry Practitioner — Perdue, Inc. (chickens)
Dr. Betsy Sigmon	Small Animal Practitioner—Creature Comforts Animal Hospital
Mr. Larry Wooten	NC Farm Bureau