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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

Avian Influenza: Whyyyy are eggs so expensive???

If you've been to the grocery store recently, you might have noticed that egg prices have skyrocketed. Why? Why has chicken meat not followed suit? The answer to that is primarily scientific, but also partially economics. Let's talk about the science first.

Highly Pathogenic Avian Influenza (HPAI) hit the United States hard in 2022, and the outbreak has lasted longer than anyone anticipated. What is HPAI? Avian Influenza is a type A influenza virus that contains a certain hemagglutinin (H) and neuraminidase (N) combination to make it pathogenic in poultry. Traditionally, the two main H types that the World Organization of Animal Health (WOAH) are concerned with are H5 and H7. The unique thing about this virus is that an H5/H7 could be either a "low pathogenicity" (LPAI) or a "high pathogenicity" (HPAI) strain, depending on its genetic makeup. Here's where things get fun (strictly from a scientist-nerding out perspective)- Wild birds such as waterfowl tend to carry and shed highly pathogenic avian influenza viruses without being affected by them (although in this outbreak we have seen numerous cases of sick and dying snow geese and other wild waterfowl). When these viruses infect domestic birds, the results are vastly different. Turkeys and chickens are specifically susceptible to the virus, but it has also been shown to cause morbidity and mortality in domestic waterfowl such as ducks and geese. The "classic" scenario, and the first case in Virginia, occurred when some domestic waterfowl co-inhabited a lake with some wild migratory birds. The domestic birds were infected and then co-housed with chickens and turkeys, resulting in

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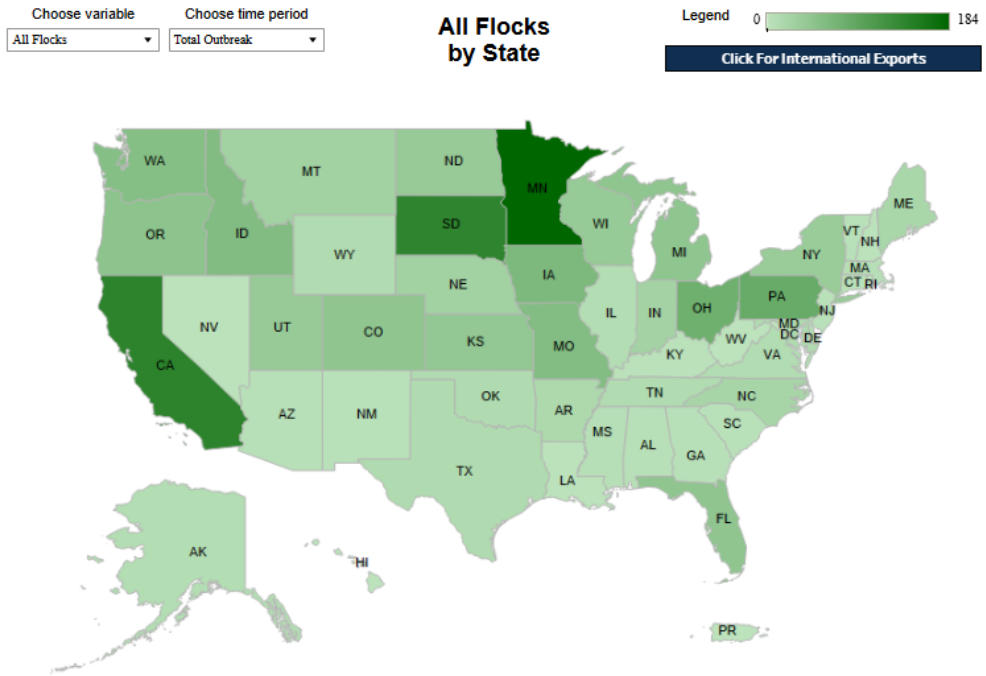
almost 100% mortality of the domestic gallinaceous birds. Biosecurity note- don't let your domestic birds play with wild ones. Since the start of the HPAI outbreak in the US on February 8th, 2022, 158.97 million birds have been affected. This has included a total of 1,566 flocks in 51 states. Of those, 744 flocks were commercial, and 822 have been backyard (USDA APHIS February 2025). The majority of these cases were the HPAI H5N1 clade 2.3.4.4b (genotypes A1-A6), which had originated from wild birds. Later cases were reported as the B3.13 genotype from dairy cattle (we will get to that!). Unfortunately, there is no treatment for HPAI, and the approach to eliminate it is to depopulate large houses of infected birds to stamp out the virus. While this is not ideal, it is more humane than the near 100% mortality that the virus can cause. Additionally, there are many levels of trade implications with "poultry" (by WOAH standards). We won't get into this, but it is a major player. Biosecurity is the number one prevention to ensure healthy flocks and protection from disease.



(Confirmations of Highly Pathogenic Avian Influenza in Commercial and Backyard Flocks | Animal and Plant Health Inspection Service)

In early 2024, things really got interesting with HPAI with the detection of the virus in lactating dairy cattle in Texas- starting out as an unknown disease affecting cows. As a response, an April 2024 Federal Order was issued that mandated the testing of lactating dairy cattle for interstate movement. Later, enhanced surveillance included bulk tank and silo level surveillance testing through a National Milk Testing Strategy (NMTS) (HPAI in Livestock | Animal and Plant Health Inspection Service). Initially, these cases were identified as the HPAI H5N1 clade 2.3.4.4b, originating from wild birds. This strain was identified as a B3.13 genotype. Epidemiologic studies showed that key transmission factors were cattle movement and both indirect and direct transmission. Biosecurity was identified as the key prevention factor (Highly Pathogenic Avian Influenza H5N1 Genotype B3.13 in Dairy Cattle: National Epidemiologic Brief). In early 2025, testing performed on dairy silos in Nevada confirmed infection with genotype D1.1, the predominant circulating genotype in wild birds. In total, there have been 968 HPAI cases in dairy cattle in 16 states. Affected dairy cattle have predominantly shown minimal clinical signs other than drops in milk production or irregular milk. Oddly, the virus tends to localize to the mammary gland.

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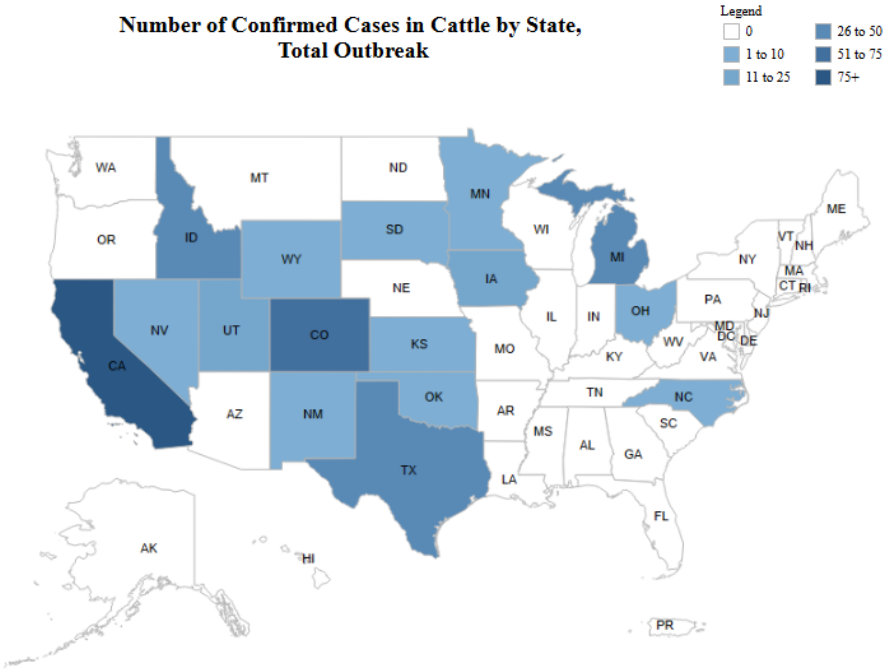
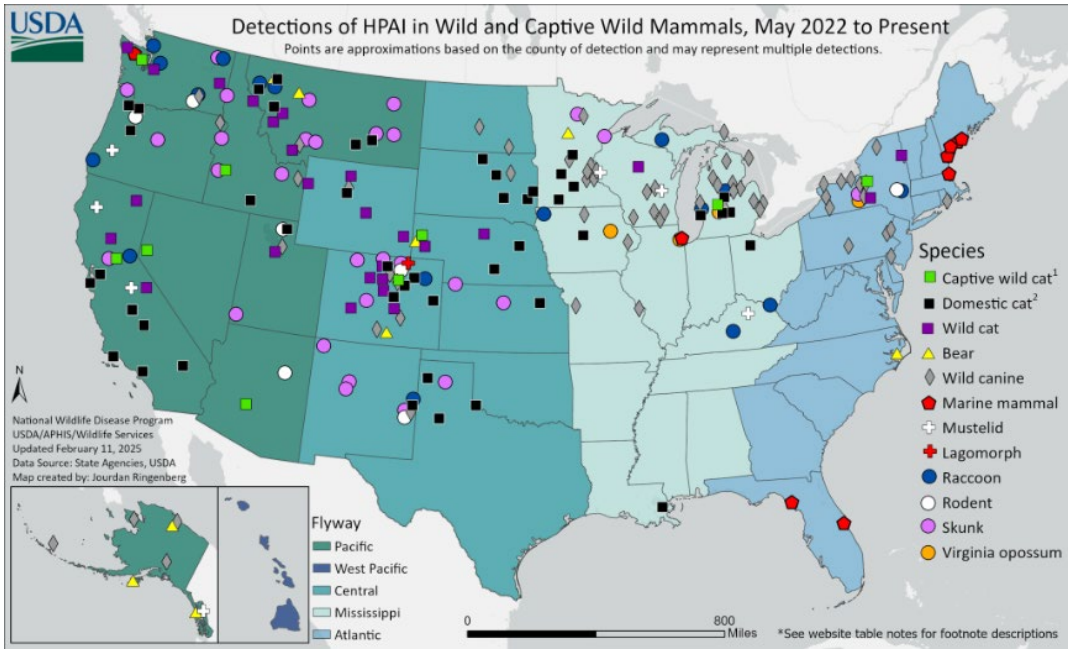


(HPAI Confirmed Cases in Livestock | Animal and Plant Health Inspection Service)

The question now becomes- HPAI in mammals? Is that a thing? Yes and no. This is the first time we have really seen HPAI in mammals that were not dead-end hosts. Lots of species are susceptible to the virus, but it has traditionally been linked to consumption of dead or diseased infected birds (including not only mammals but also birds of prey and vultures). One of the key players in recognizing HPAI on dairies was that cats were dying from the consumption of infected milk with high viral loads. While dairy cattle have been *mostly* unaffected with regards to clinical signs, many of the cases of mammals infected through consumption resulted in severe disease or death. Dairy cattle have become one of the first mammalian species to really spread the virus to others.



(HPAI Detections in Mammals)

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So now that we have geeked out about the science (although VERY briefly), let's go back to our original question: Whyyyy are eggs so expensive? Economics.

As you can see, HPAI has wreaked havoc on the poultry industry, so in a very cut and dry statement- fewer eggs = more expensive as a classic supply and demand scenario. One of the questions I hear frequently, though, is why are eggs so expensive and chicken meat is not? The simple answer to that is that those are two different production models. When a house of egg layers (millions of birds) is infected with HPAI and must be depopulated, there is a mandatory down time before replacement. When it comes time to replace these birds, egg laying breeds of chickens must mature to 20-25 weeks of production before they start laying eggs again. It takes even longer than that to be in prime production (think one egg per bird per day). For broilers (meat chickens- 25,000-50,000 per house), after the downtime, it takes only about 6 weeks to have market-ready chickens again. With regards to breeders, there are egg layer breeders and broiler breeders. Because of the differences in bird size and years of genetics, the two are not really interchangeable- so we can't use broiler chickens for eggs. When it comes to production, egg facilities are much larger than broiler facilities, so a single infected premises will be more damaging with regards to percentage of overall production. Unfortunately, it spirals from there.

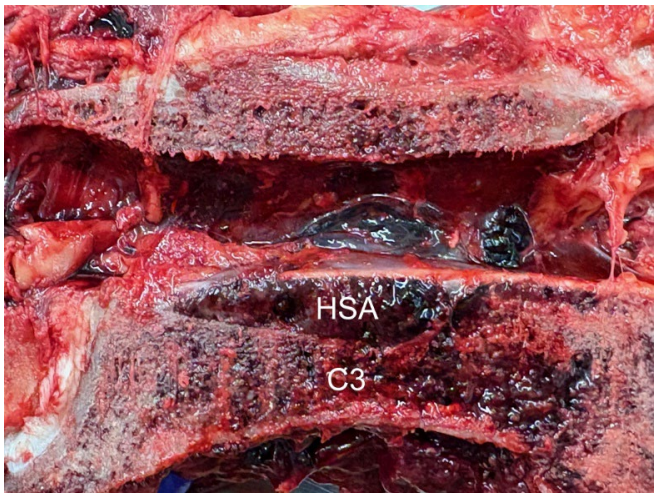
Jessica Walters, DVM, PhD, DACPV
Program Manager, Office of Laboratory Services, VDACS

Horses and Donkeys

Vertebral hemangiosarcoma in a horse

A 17-year-old mare presented with acute 4-limb ataxia and inappetence but was otherwise alert, responsive, with an adequate tongue tone and no facial problems or vision deficits. Ataxia rapidly progressed and, in 24-36 hours after the onset of the clinical signs, the mare was toe dragging and unable to lift any of the four legs off the ground when walking. When a limb was elevated manually to examine the hoof, the horse could not balance on 3 legs. The animal's condition deteriorated rapidly and humane euthanasia was elected after the animal was laterally recumbent and unable to incorporate or stand. On gross examination of the CNS, there was a red, fleshy and bloody epidural mass arising from the body of the 3rd cervical vertebra (C3) compressing the spinal cord. Histologically, this mass was compatible with a C3 hemangiosarcoma (HSA), which had caused degenerative lesions in the spinal cord associated to compression. There was no tumor metastasis in the visceral organs.

Santiago Diab DVM, DACVP - Virginia Tech



Bovine and Camelids

Chronic compressive cervical myelopathy due to intervertebral disc disease in a llama

A 17-year-old female llama was euthanized after an 8-year history of neck issues including decreased neck mobility, pain, and difficulty rising, with onset of wandering in circles immediately prior to euthanasia. On post-mortem examination, the cervical spinal canal was narrowed and distorted at the level of C3-C4 due to severe intervertebral disc degeneration, characterized by desiccation and herniation of the intervertebral disc compressing the ventral aspect of the cervical spinal cord. Similar findings were identified at the level of C5-C6. All articular facets of vertebral bodies C3-C6 were irregular, with lipping of the articular cartilage and fibrillation to eburnation of the articular surface. Histopathology of multiple spinal cord segments from C3-C6 revealed scattered areas with diameter variation of the myelin sheaths and rare axonal spheroids in the white matter. There was prominent perivascular fibrosis throughout the cervical spinal cord, which indicated previous/on-going damage, and this histologic feature is reported to be characteristic of chronic myelopathy. The gross and histologic findings were consistent with chronic compressive cervical myelopathy due to intervertebral disc disease at the level of C3-C6. Although common in dogs, intervertebral disc disease is rarely reported in other species, with only one report of chronic compressive cervical myelopathy due to intervertebral disc disease in a 12-year-old female llama.

Alexandra Reddy, DVM - Virginia Tech
Francisco R Carvallo MV, DSc, DACVP - Virginia Tech

Pneumonic mannheimiosis in cows

Multiple adult dairy cows were reported to experience acute disease characterized by recumbence, open mouth breathing, lack of rumen contractions, severe pain, inability to auscult heart sounds, and cold extremities. A 2-year-old and 4-year-old cow (Jersey and Holstein respectively) were euthanized and submitted for necropsy. Both cows had severe fibrinosuppurative and necrotizing bronchopneumonia with extensive fibrinous pleuritis. *Mannheimia haemolytica* was isolated in pure culture from the affected lung lobes of both animals. The development of severe pneumonic Mannheimiosis in multiple adult cows was an interesting and unusual feature in this case.

Thomas Cecere DVM, PhD, DACVP - Virginia Tech

Pre pubic tendon tearing in a pregnant ewe

A 3-year-old pregnant ewe was submitted for necropsy, with history of pendulous abdomen, almost touching the ground and with significant problems for deambulation. When brought to the Virginia tech large animal hospital, the ewe was not ambulatory. The owner elected a terminal c-section, and three lambs were retrieved, but two of them were not able to breathe. During necropsy, a severe tearing of the right internal and external oblique musclers was noted, with herniation of the uterus and intestine. These findings are indicative of a pre pubic tending tearing. This condition is defined as a hernia secondary to the rupture of the tendons that connect lower pelvis to the abdominal muscles. The presence of two or more fetuses and/or trauma may predispose to this condition, which has been described in horses, dogs and sheep. Unfortunately, this condition is not treatable and does not resolve.

Francisco R Carvallo MV, DSc, DACVP - Virginia Tech

Avian

Avian bornavirus in a green cheeked conure

A 7-month-old male green cheeked conure (*Pyrrhura molinae*) died the day after arriving at a pet store. The bird was in good body condition and the feathers on both wing tips were clipped. The gastrointestinal tract was mostly empty. Otherwise, the gross examination was unremarkable. Microscopic examination showed lymphocytes and macrophages infiltrating the cranial nerve ganglia adjacent to the brain and the myenteric ganglia in the walls of the proventriculus and ventriculus. The airways of the lungs contained a small amount of plant material, surrounded by multinucleated giant cells, suggestive of aspiration. These findings supported a diagnosis of avian bornaviral ganglioneuritis, leading to decreased gastrointestinal motility with regurgitation and aspiration of ingesta. Avian bornaviral ganglioneuritis, also called parrot wasting disease or proventricular dilatation disease, is caused by an RNA virus that may trigger an autoimmune response to neurons. The virus is thought to be transmitted vertically, but the exact mode of transmission is not known.

Teresa Southard DVM, PhD, DACVP - Virginia Tech

Ovarian Adenocarcinoma in a Four-Year-Old Hen

A four-year-old hen was presented with a two-week history of progressive right leg paresis, characterized by the loss of deep pain sensation and absence of reflexes. The bird also exhibited anorexia. When handled, it developed open-mouth breathing and regurgitation. Due to the severity of clinical signs, euthanasia was performed.

Postmortem examination revealed a distended coelomic cavity filled with 500 mL of green, translucent fluid. Numerous white to tan nodules were present on the intestines, liver, spleen, peritoneum, kidneys, and ovaries. The right kidney was completely replaced by these nodules.

Microscopic examination confirmed that the nodules consisted of neoplastic epithelial cells, consistent with ovarian adenocarcinoma. The presence of widespread dissemination of this tumor within the coelomic cavity is termed carcinomatosis.

Ovarian adenocarcinoma is one of the most common tumors in laying hens above 2 years old, often presenting with extensive intra-coelomic spread. The lameness observed in this case was due to sciatic nerve compression by the tumor replacing the right kidney, a well-recognized phenomenon in poultry. The coelomic effusion and respiratory distress were likely caused by tumor-induced vascular obstruction and organ compression secondary to the severe effusion respectively. This case underscores the significance of ovarian adenocarcinoma as a common neoplastic condition in older laying hens. The disease often progresses insidiously, with clinical signs becoming apparent only when significant spread has occurred. Given the anatomical relationship between the kidneys and the sciatic nerve, cases of unexplained lameness in birds warrant further investigation into potential underlying renal or neoplastic conditions.

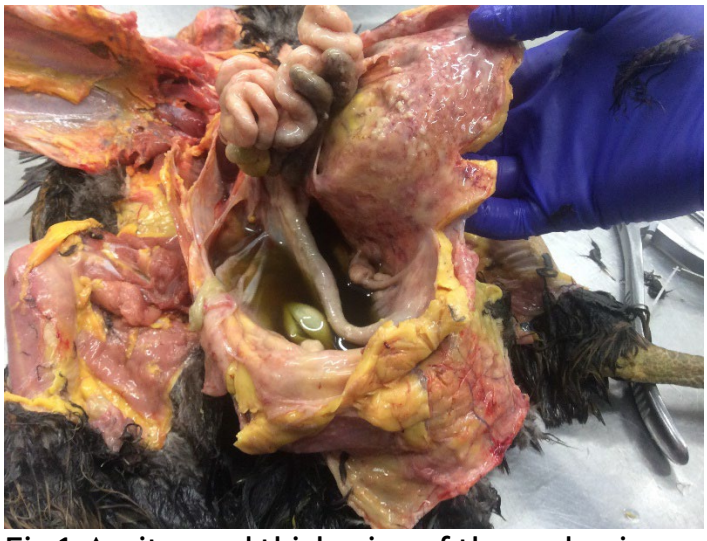


Fig 1: Ascites and thickening of the coelomic serosa.

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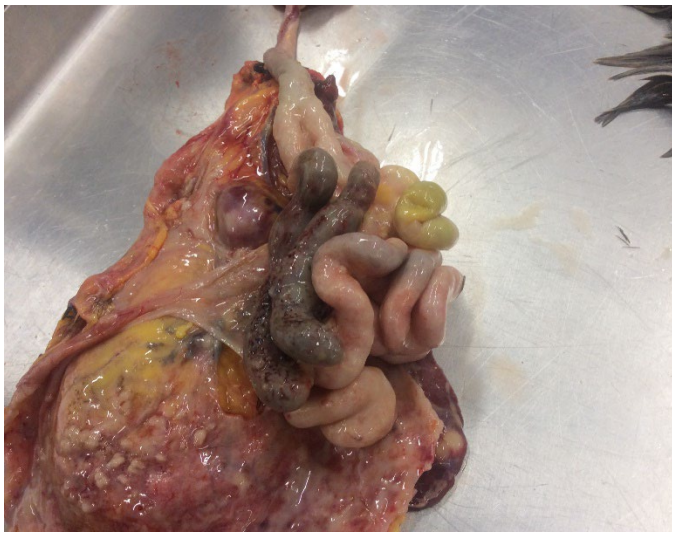

Fig 2. Intestine with multiple serosal adhesions (sequel of carcinomatosis).

Abraham Adeyemo, DVM - Virginia Tech
Santiago S. Diab DVM, DACVP - Virginia Tech

Companion Animals

Septic pasteurellosis in a rabbit

A 1.5-year-old, female spayed, New Zealand rabbit died spontaneously after a history of anorexia, lethargy, and loose stools over a 12-hour period and lost 4 pounds over the previous month. On post-mortem examination, significant findings included a red, firm area in the left cranioventral lung lobe that did not float in formalin, pinpoint white nodules throughout all liver lobes, and mucoid material in the tympanic bullae. Histopathology revealed acute necrotizing hepatitis with intralesional bacteria, which was indicative of bacterial septicemia. These findings were supported by a positive bacterial culture of *Pasteurella multocida* from the liver. Additionally, there was a regionally extensive area of acute interstitial pneumonia and vascular damage with similar intralesional bacteria, which further supported the diagnosis of bacterial septicemia due to *Pasteurella multocida*. The presence of abundant mucoid material in the tympanic bullae is a common finding with pasteurellosis. Pasteurellosis is one of the most clinically significant diseases of rabbits. Many rabbits harbor this organism within the upper respiratory tract and tympanic bullae asymptomatically and serve as carriers. This organism is transmitted through infected nasal and vaginal secretions, as well as through nursing. Clinical disease is variable but commonly includes rhinitis, otitis media, pneumonia and septicemia. Rabbits with septicemic pasteurellosis frequently die with acute to no premonitory clinical signs.

Alexandra Reddy, DVM - Virginia Tech
Francisco R Carvallo MV, DSc, DACVP - Virginia Tech

Secondary copper-associated hepatopathy due to chronic cholangiopathy in a cat

A 4-year-old cat was euthanized after an acute onset of hyporexia, listlessness, tremors, and seizures and a recent history of getting into the garbage with potential access to human medications. Ghost cells, severe hyperbilirubinemia, and mostly unremarkable liver enzymes were identified on CBC/CHEM. The most significant findings on gross and histologic evaluation included diffuse icterus, end-stage liver disease (cirrhosis), and chronic peri-cholangitis with ductular reaction. Special histochemical stains were performed to enhance our understanding of potential causes for the severe hepatobiliary changes: 1) copper stain, which highlighted abundant copper granules within hepatocytes and macrophages, 2) iron stain, which highlighted iron pigment within macrophages, and 3) trichrome stain, which highlighted dense bands of fibrosis dissecting throughout the hepatic parenchyma. In combination, these severe hepatobiliary changes were compatible with secondary copper-associated hepatopathy. Although uncommonly reported, this disease has been reported in cats secondary to chronic hepatobiliary disorders including chronic biliary obstruction and chronic cholangitis-cholangiohepatitis. This patient's

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history of previous gallbladder obstruction was consistent with the chronic hepatobiliary changes, and it is likely that this animal developed a secondary copper-associated hepatopathy as a sequela to chronic cholangiopathy. Therefore, these findings were unrelated to the patient's clinical history of recent dietary indiscretion. Although there were no significant histologic findings in the brain, the patient's clinical history of seizures and tremors can likely be attributed to hepatic encephalopathy secondary to hepatic insufficiency.

Alexandra Reddy, DVM - Virginia Tech
Francisco R Carvallo MV, DSc, DACVP - Virginia Tech

Laboratory News

ViTALS

Sheryl Coutermarsh-Ott us returning to the pathology team in ViTALS, and will be continuing to support the VDACS pathology service. We are excited to have her back in a full-time capacity and look forward to further enhanced collaboration with diagnosticians in the RAHLs.

We also want to welcome Christina Pacholec to the Clinical Pathology team. You may have interacted with Christina while she was doing her clinical pathology residency in ViTALS and as a clinical instructor after passing her board exam. She will be joining us full time in May. Anna Hassebroek accepted the NBAF scientist position, and has been serving in that role since December. In that role, she will provide disease investigation and pathology support to regional labs.

Tanya LeRoith DVM, PhD, DACVP. ViTALS Director.

VDACS

The VDACS Office of Laboratory Services has converted to a new LIMS system! CoreOne for Labs went live on February 3rd, and we are excited for the ability to allow clients to pay invoices, create accessions, and log into and track samples/results through the new client portal. The billing portal will open on March 3rd due to necessities in the finance transition.

VDACS and ViTALS are excited to enhance collaboration in histopathology services. With the return of Dr. Sheryl Coutermarsh-Ott to VMCVM, VDACS has expanded their partnership with the veterinary college to source all histopathology and pathology subject matter expertise through ViTALS.

Bulk tank surveillance testing for Highly Pathogenic Avian Influenza Virus in milk began in January at the Harrisonburg Regional Animal Health Laboratory. Over 250 bulk tank samples have been tested so far with over 650 samples tested to date (all testing negative). The laboratory is expecting to continue to enhance surveillance numbers with changing federal orders. In Warrenton, analysts are successfully working on bringing in CL SHI testing capabilities and have been working to bring in Avian Influenza testing. The Wytheville Laboratory is in the process of bringing up confirmatory testing for Viral Hemorrhagic Septicemia Virus in fish and are preparing for the yearly fish screening testing for the Department of Wildlife Resources. The Lynchburg Laboratory is now approved to test for Foot and Mouth Disease and both Avian Influenza and Newcastle Disease as part of the National Animal Health Laboratory Scope expansion.

We are always open to feedback and ideas for testing to further assist our agricultural community. It is our goal to make this lab system the best it can be to further serve our clients. Our partnership with the Virginia Tech Animal Laboratory Services Lab (ViTALS) continues to evaluate ways to collaborate and expand testing services. Whether by utilizing data for reports or enhancing testing capabilities, we look forward to continuing to work together and with all of you.

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

LABORATORY LOCATIONS

ViTALS Virginia Tech Animal Laboratory Services

VIRGINIA TECH ANIMAL LABORATORY SERVICES
205 Duck Pond Drive
Blacksburg, VA 24061
540-231-7666
lcrvth@vt.edu

RAHLS: Regional Animal Health Laboratory System

HARRISONBURG
261 Mt. Clinton Pike
Harrisonburg, VA 22802
540-209-9130
RAHLHarrisonburg@vdacs.virginia.gov

WARRENTON
272 Academy Hill Rd.
Warrenton, VA 20186
540-316-6543
RAHLWarrenton@vdacs.virginia.gov

LYNCHBURG
4832 Tyreeanna Rd.
Lynchburg, VA 24504
434-200-9988
RAHLLynchburg@vdacs.virginia.gov

WYTHEVILLE
250 Cassell Rd.
Wytheville, VA 24382
276-228-5501
RAHLWytheville@vdacs.virginia.gov

VIRGINIA DEPARTMENT OF AGRICULTURE AND CONSUMER SERVICES

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