Laboratory Services Division
Animal Health Laboratory

AHL Newsletter

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In this issue:

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Editor: **Maria Spinato,** DVM, DVSc, Diplomate ACVP, MBA
Editorial Assistants: **Helen Oliver, Sofija Jelacic**

The *AHL Newsletter* is published quarterly (March, June, September, December) by the Animal Health Laboratory, Laboratory Services Division, University of Guelph.

Its mission is to inform AHL clients and partners about AHL current activities, and laboratory-based animal disease events and disease trends. *Ideas and opinions expressed herein do not necessarily reflect the opinions of the University or the Editor.*

Mailing address & contact information:
Animal Health Laboratory
Laboratory Services Division, University of Guelph
Box 3612, Guelph, Ontario, Canada N1H 6R8
Phone: (519) 824-4120 ext. 54538; fax: (519) 821-8072

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Update from the Director

The view from the Director's office

The end of summer is approaching, and while our neighbours in Western and Eastern Canada have experienced many devastating weather-related events, those of us in Ontario can only complain about too many soggy days and a few hail and wind storms. The insects have flourished in this weather as well, and it is that time of year when vector-borne diseases such as EEE and West Nile virus are increasing in numbers, in addition to Lyme disease and *Anaplasma phagocytophilum*. Check out this month's articles on best practices for testing for Eastern equine encephalitis virus and West Nile virus encephalopathies, leucocytozoonosis in multiple avian species, as well as the OAHN companion animal network's recently-released infographic on the distribution of Lyme disease and its associated tick vector.

It is very important to stop and take some time during our hectic schedules to celebrate our successes. We are pleased to announce that Ms. Joanna Sawicki, AHL's Virology Technical Supervisor, has been awarded the University of Guelph President's Exemplary Staff Award in the category of Service Excellence. This award is in recognition of the superb service that Joanna provided to CFIA and industry clients during the HPAI outbreaks during 2022 and 2023. Joanna's leadership and communication skills have been praised by those who worked with her during these outbreaks. Congratulations on this well-deserved award Joanna!

I hope you have been able to take some time off this summer, and are feeling revitalized and prepared for the busy fall ahead.

Maria Spinato, Director

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Client Portal

Josie Given, Client Outreach Technician

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2023;27(3):3.

Interested in our AHL Client Portal? If your clinic submits samples to the Animal Health Laboratory on a regular basis (2-3 x/week) and you want to streamline your AHL experience, please let us know! The AHL Client Portal offers our clients the ability to integrate their client database, and ensure proper spelling and demographic information on each case submitted. Our Client Services team will work with your clinic to customize your portal experience. Features of the AHL Client Portal include:

- Fully responsive (works on PC, phone, tablet);
- Create your submissions in-clinic, ensuring proper spelling, PIDs and demographics;
- Edit/cancel details until the specimen is formally received at AHL;
- Know your case # prior to sending to AHL, and track its progress as results are made available;
- Advanced search capabilities, and the ability to design reports based on animal ID, farm, owner (**Fig. 1**). *AHL*

Figure 1. Example of AHL client dashboard.

New PCR tests at the AHL

Tim Pasma

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2023;27(3):4.

The following new virology PCR tests have recently been validated and are now available at the AHL:

1. Equine respiratory triplex PCR: Influenza A virus/Equine rhinovirus A/Equine Rhinovirus B – code **inflerv** - $60.00
   Sample to submit: live: nasal swab, oral fluid; dead: trachea, lung

2. Porcine sapovirus PCR – code **psapopc** - $40.00
   Sample to submit: live: feces, fecal swab; dead: intestine

3. Cache Valley virus PCR – code **cvvpcr -** $40.00
   Sample to submit: placenta, brain

Collect swabs into virus transport medium (VTM). Tissues should be submitted in leakproof containers.

We are happy to discuss any testing questions and concerns. Please feel free to reach out!

ahlinfo@uoguelph.ca or tpasma@uoguelph.ca; 519-824-4120 extension 54530.

Determination of serum/plasma vitamin A and E testing at the AHL

Felipe Reggeti, Nick Schrier, Tracy Van Raaij

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2023;27(3):5.

Vitamin A is an essential nutrient that is necessary for dim-light vision, normal bone growth and maintenance of epithelial cells, and is not produced by ruminal bacteria. Clinical signs of deficiencies result from impairment of these functions, but the most critical economical impact is related to the effects on reproductive function. Pro-vitamin A carotenoids are abundant in green plants/grasses, and are converted to active retinol in the intestinal epithelium and liver. Deficiency may occur when animals are fed hay/straw without vitamin A supplementation (e.g., winter months), as carotenes progressively deteriorate upon storage, as well as in association with conditions causing malabsorption. Clinical signs of vitamin A deficiency may include:

- night blindness and papilledema (usually the first clinical signed noted);
- bone growth defects;
- poor maintenance of epithelial cells resulting in squamous metaplasia of glandular epithelium, reduced thyroxin secretion, xerophthalmia, corneal keratinization;
- skin problems such as scaly skin, dry hair-coat, scaly hooves with vertical cracks;
- neurological signs such as syncope and convulsions caused by elevated CSF pressure due to impaired fluid absorption;
- reproductive problems such as infertility (reduced count of motile sperm cells), abortions and placental retention;
- congenital defects such as optic nerve damage, retinal dysplasia, malformation of cranial bones;
- impaired immune system;
- weight loss (may be due to overall poor nutrition).

Vitamin E is an essential antioxidant that helps maintain cell health by preventing lipid membrane peroxidation from continued exposure to free radicals, a process that works in conjunction with the action of the selenium-containing enzyme glutathione peroxidase. Vitamin E is abundant in green forages, and similar to vitamin A, deficiency may occur when animals are fed poor quality hay or straw without vitamin E supplementation. Conditions known to respond to vitamin E (and/or Se) supplementation include nutritional muscular dystrophy (multiple species), mulberry heart disease (pigs), reproductive inefficiency and decreased resistance to infections.

Some of these health issues may be prevented by quantification of vitamin A and E concentrations in appropriate samples, and correcting suboptimal levels via supplementation. Determination of plasma/serum concentrations is practical and commonly used to diagnose nutritional imbalances, although liver concentrations are better indicators of body reserves. The AHL now offers quantification of the concentration of vitamins A and E in serum or plasma samples from multiple species by HPLC, and we are currently working on developing methods to assess concentrations in tissues. The volume of serum/plasma required for testing is less than 1 ml. For sample collection, we recommend separating the serum after clot formation (~1 hour), or centrifuging the blood to obtain the plasma soon after venipuncture. Do not use tubes with gel separators and avoid hemolysis. Transfer the sample to a transport tube, protect from light exposure, and ship refrigerated or frozen.

References
1. Radostits OM, Gay CC, Hinchcliff KW, Contstable, PD. Chapter 30 - Diseases associated with nutritional deficiencies. In: Veterinary Medicine: A textbook of the diseases of cattle, horses, sheep, pigs and goats, 10th ed. Radostits OM et al, eds. Elsevier, 2007:1771-1777.

Staff highlights

Ms. Joanna Sawicki, Technical Supervisor Virology Laboratory Section at the AHL, has been awarded the University of Guelph President's Exemplary Staff Service award in the category of Service Excellence. This award is in recognition of the superb service that Joanna provided during the HPAI outbreaks in 2022 and 2023. While managing a group of over 20 technicians and students, Joanna reorganized lab operations to improve efficiency and reduce turnaround times, often providing same day test results. Joanna's leadership and communication skills were recognized by CFIA and the poultry industry who expressed gratitude for the excellent service provided by AHL throughout the outbreaks. It was Joanna who often worked late in the evenings or on weekends to ensure that clients continued to receive timely results. We are happy that her contributions have been recognized by the President of the University of Guelph. Congratulations Joanna!

OAHN update – September 2023

Mike Deane, Tanya Rossi

Animal Health Laboratory, University of Guelph, Guelph, ON

This summer, the OAHN networks completed several research projects, held multiple species-specific meetings, and created new resources for vets and animal owners. Read on to find out what new communications and resources are now available. To view any of our network reports and research projects, go to OAHN.ca and navigate to the species you are interested in. Free memberships are available to veterinarians, students, researchers, and veterinary technicians.

New Resources and Completed Research Projects:

- Infographic: Ticks and Lyme disease in Ontario – 2023 UPDATE
- OAHN equine podcast: Exposure of Ontario broodmares to the parasites *Neospora caninum* and *Neospora hughesi*
- OAHN bovine podcast: Johne's disease control on Ontario dairy farms with Dr. Jamie Imada
- OAHN equine project: Equine network webinar series
- OAHN equine network research project: *Neorickettsia risticii* in Ontario: Identifying emerging strains, their diagnosis and environmental risk factors for disease development
- Wildfires and emergency management resources
- VIDEO: Trace mineral status in Ontario beef herds

Rabbit Hemorrhagic Disease Surveillance Project

The OAHN wildlife and companion animal networks have received approval for a joint project to provide subsidized testing for cases in deceased domestic rabbits that meet certain high-risk criteria for RHD, as well as testing for high-risk cases from rehabilitation facilities. This surveillance project is currently underway and open to rabbit submissions from veterinarians and wildlife rehabbers. Watch oahn.ca and OAHN social media for more details. If you have a question about a potential RHD case during regular business hours, please contact Dr. Alexandra Reid, co-lead of the OAHN wildlife network, at alexandra.reid@ontario.ca, or the OMAFRA AICC at 877-424-1300. Learn more about this project here: https://www.oahn.ca/resources/rhdv-project/

New Reports

Most OAHN networks create reports once per quarter. To view any of the veterinary reports below, please click on the OAHN icon for each network, or go to OAHN.ca and navigate to the species in which you are interested.

- OAHN survey: Parasite-palooza
- High path avian flu in pets
- NEW! Acute diarrhea infographic
- Rabies reg update July 1; blasto info

- National Poultry Network for Collaborative Solutions to ‘Wicked’ Problems
- Poultry veterinary survey highlights – Q2 2023
- Events and news

- Bits ‘N Snips
  - Chewing lice (*Werneckiella equi*)
  - Ergot alkaloid toxicity
- Network member reports
- Syndromic and lab surveillance dashboard

- Look out for Lead!
- Trace mineral project webinar recording and summary
- Case report: *Mycoplamsa wenyonii* in dairy cattle
- Surveillance: Q1 Animal Health Laboratory data
- Latest podcast: Johne’s disease control on Ontario dairy farms

- Disease surveillance discussion
- Animal Health Laboratory reports
- Ontario slaughter statistics
- CanSpot ASF surveillance update
- International disease surveillance topics

Lead intoxication in a herd of beef cattle

Dominique Comeau

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2023;27(3):9.

Three cattle from a group of approximately 20 Angus yearlings became ill with acute neurologic signs. Clinical signs included central blindness, a hypermetric gait, teeth grinding, twitching ears and dehydration, as well as anorexia. One of these animals was euthanized for a broken leg, and another showed signs of improvement with supportive therapy including selenium, thiamine, and penicillin, based on the top differential diagnoses of selenium deficiency, blackleg, and polioencephalomalacia. The third animal demonstrated progressively worsening neurologic signs and was euthanized and submitted to the Animal Health Laboratory for postmortem.

On gross examination of the 15-month-old heifer, there were no lesions, excluding those associated with the method of euthanasia. On histologic examination, there was multifocal vacuolation of the grey matter in the middle cortical lamina (**Fig 1**). There were also hypereosinophilic shrunken ("red dead") neurons, and frequent swollen glial cells in these affected areas, especially swollen astrocytes (**Fig 2**). The endothelial cells of vessels in affected areas were plump and reactive. These changes are all classic lesions of polioencephalomalacia.

A blood sample was submitted for testing of selenium levels. The heavy metal screen found markedly elevated lead levels in the affected animal. An extremely high lead level was confirmed by testing of the liver which had 17 ppm lead (normal reference level is less than 0.5 ppm). On further review of the history, it was determined that there was a dilapidated car in the field the cows were grazing, with access to the car battery.

Polioencephalomalacia or cerebrocortical necrosis (CCN) is a common neurologic presentation in cattle. It is most commonly associated with low levels of thiamine. In adult cattle, this vitamin is produced by ruminal microbes. Low levels of thiamine occur either due to lack of production due to alterations in ruminal microbes, or due to ingestion of plant enzymes or production of bacterial enzymes which break down thiamine. A commonly-cited example of a plant with high levels of thiaminase is bracken fern. Alternatively, CCN can be caused by high intake of sulphur, which leads to production of abundant sulphide in the rumen which alters the ruminal microflora. Increased dietary sulphur can come from water, feed additives, or forage.

Lead intoxication is one of the most common neurologic intoxications in cattle, but it is not always considered in cases of CCN. Diagnosis is essential as specific chelating agents are needed for treatment, and animals with high levels of lead cannot enter the food chain due to the risk of human exposure. The classical clinical signs are central blindness, ataxia, twitching of the ears and eyelids, and bruxism; however, these overlap extensively with the signs of CCN from other causes. In subacute lead poisoning, gastrointestinal signs may predominate early in the course of disease, and include anorexia, ruminal stasis, and constipation. Histologic lesions of cerebrocortical injury are not apparent in every case. Inclusion bodies can often be seen in the kidney in subacute or chronic cases of lead toxicosis. Common sources of exposure for cattle include old car or tractor batteries, lead paint, roofing materials, or

contamination of foliage in areas exposure to industrial waste. Young animals are more likely to consume these materials, and are more commonly affected.

The remaining animals in this herd were tested for lead exposure. Approximately 65 % of the animals had elevated lead levels in the blood. This case is a reminder that lead toxicity remains an important differential in cases presenting as polioencephalomalacia if there is any history of possible exposure.
AHL

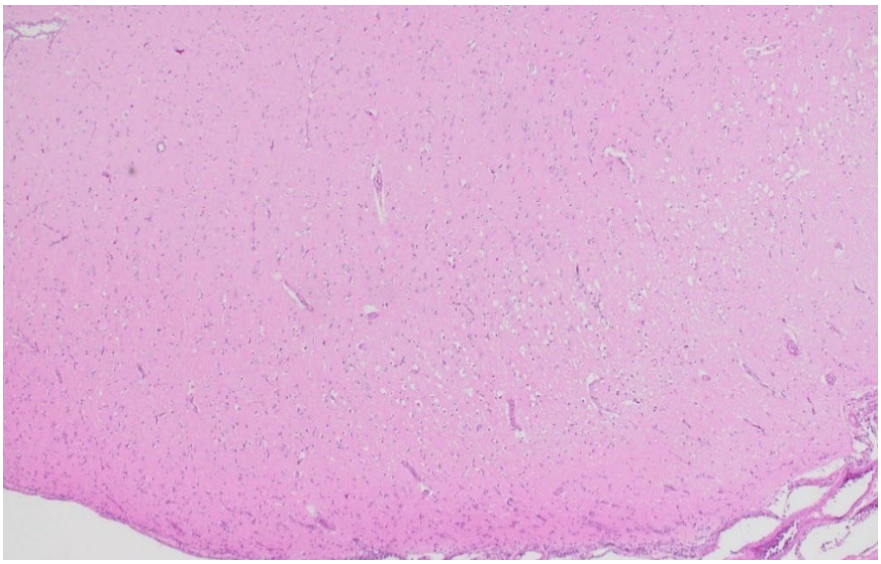

Figure 1. Vacuolation of the grey matter of the middle cortical lamina is evident on the right-hand side of the image. This is characterized by clear (white) spaces within the background of the neuropil (pink). The tissue on the left-hand side of the image is unaffected.

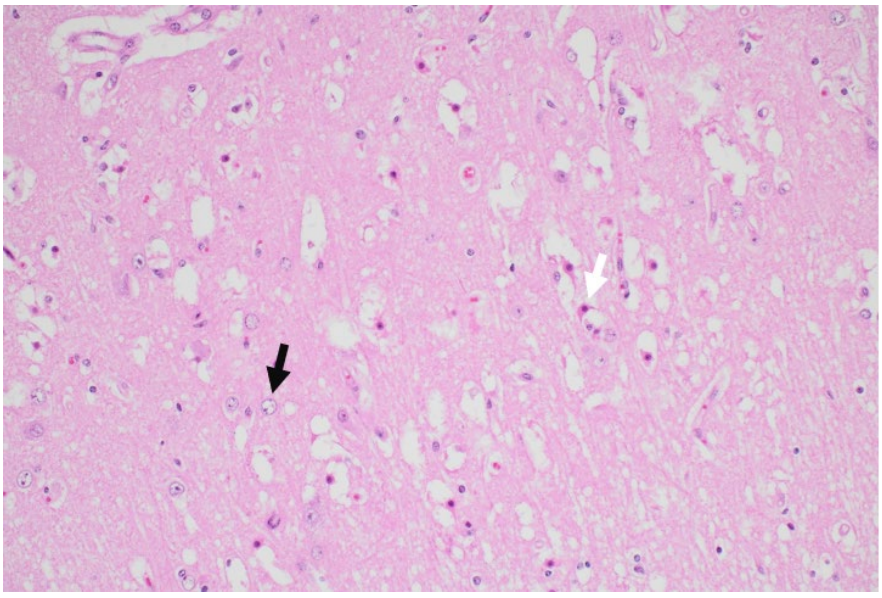

Figure 2: Higher magnificent view of an area of vacuolation showing swollen astrocytes (black arrow) and shrunken, dead neurons (white arrow).

Reference

1. Osweiler GO, et al. Epidemiology of lead poisoning in cattle– A five-year study in Iowa. Clin Toxicol 1973;6(3):367-376.

Infection of pigs with influenza A virus H3.2010.1 clade in Ontario

Davor Ojkic, Tim Pasma, Kevin Vilaca, Meegan Larsen, Yohannes Berhane.

Animal Health Laboratory, University of Guelph, Guelph, ON (Ojkic, Pasma, Larsen); South West Veterinarians, Stratford, ON (Vilaca); National Center for Foreign Animal Disease, Canadian Food Inspection Agency, Winnipeg, MB (Berhane)

AHL Newsletter 2023;27(3):12.

In April 2023, samples from four 7-week old piglets were submitted to the University of Guelph Animal Health Laboratory (AHL). The herd had a history of respiratory distress and an increase in mortality. Samples included formalin-fixed tissues for histologic examination as well as 4 nasal swabs for PRRSV and influenza A virus PCR tests. Histologic examination found lung lesions consistent with influenza infection (**Fig. 1**), and influenza A virus antigen was detected in bronchial and bronchiolar epithelial cells in affected areas using immunohistochemistry (**Fig. 2**). A diagnosis of influenza was also supported by strong positive PCR test results for influenza A virus (Ct=22.21-28.6) on all four swabs. One swab was also weakly positive for PRRSV (Ct=35.54); however, microscopic lesions suggestive of PRRSV infection were not seen in lungs.

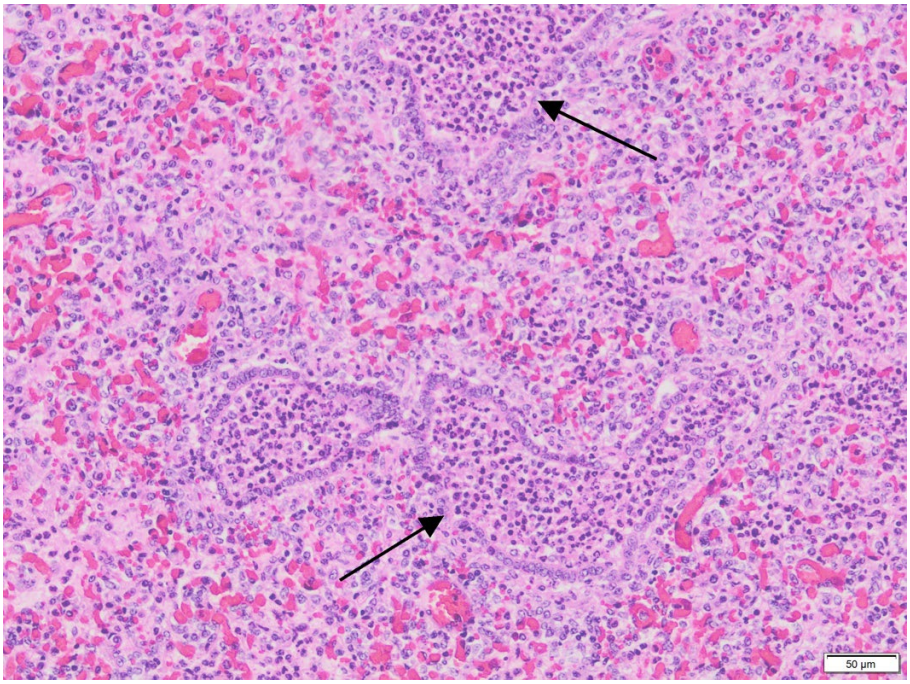

Figure 1. Porcine lung with lesions of suppurative bronchointerstitial pneumonia. Bronchioles and adjacent alveoli contain large numbers of neutrophils, and there is patchy attenuation and loss of bronchiolar epithelium (black arrows). (H&E stain)

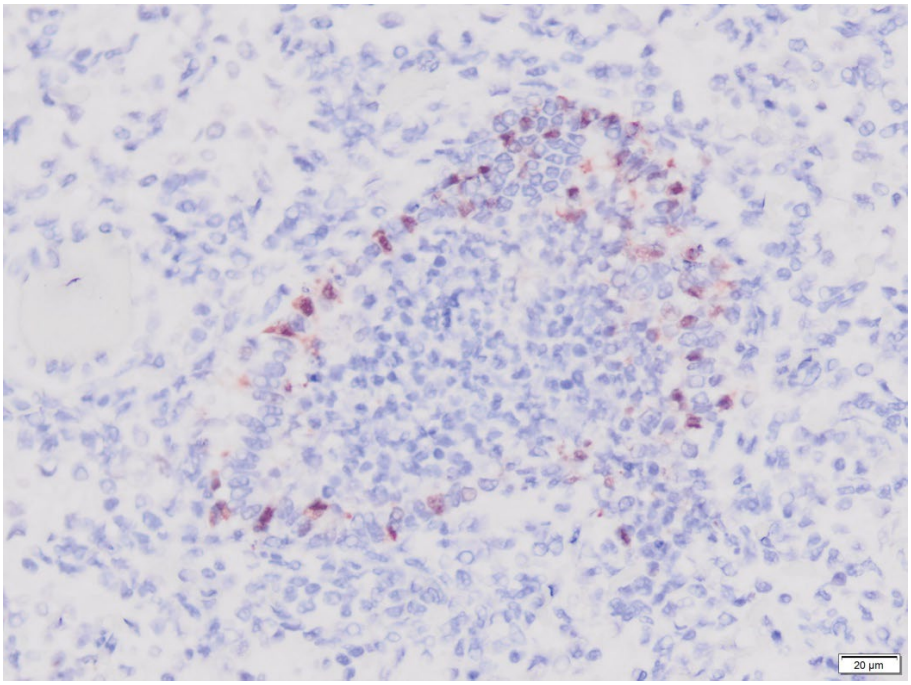

Figure 2. Porcine lung with positive (brown) cytoplasmic staining of bronchiolar epithelial cells lining affected bronchioles. (Influenza A immunohistochemical stain)

Hemagglutinin gene sequencing of one positive sample revealed the virus belonged to influenza A virus subtype H3N2 clade 2010.1 (H3.2010.1). This clade was identified in Ontario swine previously in 2017, when four cases from the same owner were detected, but that outbreak did not spread to other farms. No other detection of H3.2010.1 clade was documented until 2023. Interestingly, in 2022 H1N1/H1N2 subtype viruses were the most frequently detected influenza A subtype in Ontario samples: 61% of genotyped viruses were H1N1/H1N2 subtype and 39% were H3N2 subtype (**Table 1**).

Table 1. Genotyping results for influenza A viruses from pigs in Ontario (January 1, 2022-December 31, 2022).

Clade	H1N1	H1N2	H3N2
alpha	4	28	
beta	14		
IV			13
IVb			18
IVc			1
IVx1			4
IVx2			3
pdm	15		
Total	33	28	39

However, since April 2023, the prevalence of H3N2 viruses has increased markedly: 78.6% have been genotyped as H3N2 and only 21.4% were H1N1/H1N2. The H3.2010.1 clade has become the dominant

H3N2 strain in Ontario, and since its initial detection in April 2023, H3.2010.1 represents 84% of H3N2 influenza A viruses from pigs (**Table 2**). The whole genome sequences were obtained for 5 recent cases, and they all showed a remarkably high identity among themselves, and a very high level of identity to the US viruses.

First detected in humans during the 2010-11 flu season, genetically-related swine H3.2010.1 viruses have become established in swine populations in the US, and have been the most common clade detected in several US states. Ontario pig herds appear to be going through a rapid transmission cycle, but it is not yet known if the virus has appeared in other Canadian provinces. *AHL*

Table 2. Genotyping results for influenza A viruses from pigs in Ontario (April 15, 2023-July 26, 2023).

Clade	H1N1	H1N2	H3N2
2010.1			28
alpha		5	
beta	1		
IV			1
IVb			3
IVx1			1
pdm	3		
Total	4	5	33

References
1. Rajão DS, et al. Novel reassortant human-like H3N2 and H3N1 influenza A viruses detected in pigs are virulent and antigenically distinct from swine viruses endemic to the United States. J Virol 2015;89(22):11213-22.
2. Powell JD, et al. Characterization of contemporary 2010.1 H3N2 swine influenza A viruses circulating in United States pigs. Virology 2021;553:94-101.

CanSpot ASF surveillance testing is ongoing – Submit your samples

Josepha DeLay

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2023; 27:14.

The CanSpot ASF program remains open for African swine fever (ASF) surveillance testing of eligible swine cases submitted to the AHL. The eligibility criteria for testing include a broad range of clinical and pathologic scenarios (**Table 1**). To date in 2023, these criteria have been met for an average of 54% of swine pathology submissions to the AHL. Unfortunately, only 35% of eligible cases included appropriate tissue samples (fresh spleen) for ASF PCR testing. Surveillance testing could not be pursued on the remaining 65% of eligible cases due to the lack of appropriate samples.

A proactive surveillance program is only as successful as the quality and magnitude of sampling. More robust ASF surveillance testing in Ontario swine could be accomplished if fresh spleen were included

with **every** field postmortem case submitted to the AHL. In many cases, eligibility for ASF surveillance testing is not suspected based on the clinical scenario, although subsequent histopathology findings often do meet eligibility criteria. For example, nursery pigs may have clinical pneumonia without a significant increase in mortality, in which case the group would not be eligible for CanSpot ASF testing. However, the same group may have histologic evidence of septicemia or fibrinous pleuritis that would qualify for testing. Sampling spleen at the time of field necropsy in every case would allow ASF surveillance testing to proceed for all eligible cases, including those in which eligibility was not expected based on clinical findings.

Please remember to include pertinent clinical history and gross postmortem findings on the submission form, such as increased mortality, fibrinous pleuritis or pericarditis, or splenic torsion. These findings will influence eligibility for ASF surveillance testing.

Trace-back information (PID, or the physical address of the herd) must also be available in order to proceed with CanSpot ASF surveillance testing.

Thank you to all swine practitioners who have submitted samples and agreed to ASF surveillance testing. **Please continue to include fresh spleen with all field necropsy cases.** *AHL*

Table 1. Clinicopathological presentations eligible for CanSpot ASF testing in swine.

Septicemia and/or multiorgan hemorrhage such as caused by *E. rhusiopathiae*, *S. suis, S. zooepidemicus, A. suis, S. cholerasuis,* or other bacteria
Porcine Reproductive and Respiratory Syndrome virus (PRRSV), especially when it causes cyanotic skin
Porcine Dermatitis and Nephropathy Syndrome (PDNS) and vasculitis that can be caused by PCV 2, PCV 3 or other pathogens
Hemorrhagic diarrhea / **necrotizing enterocolitis** such as caused by *Salmonella* spp; *L. intracellularis, B. hyodysenteriae, B. hampsonii*
Fibrinous pleuritis / pericarditis / hydropericardium such as caused by *H. parasuis, S .suis*
Mulberry heart disease
Splenic torsion
Abortion above historical trend for herd
Mortality above historical trend for herd

Lead toxicosis in a captive urban rock pigeon (*Columba livia)*

Emily Brouwer, Markus Luckwaldt

Animal Health Laboratory, University of Guelph, Guelph, ON (Brouwer); Greenwood Park Animal Hospital, Toronto, ON (Luckwaldt)

AHL Newsletter 2023; 27(3):16.

A captive wild rock pigeon (*Columba livia)* was submitted to the Animal Health Laboratory for postmortem examination. The bird was initially examined six days prior to death for severe crop stasis and distension. At the time of examination, the bird was markedly underweight with pectoral muscle atrophy and a distended crop. Cytology of crop contents identified large numbers of spore-forming bacilli. A wet mount of the crop wash was negative for Gram positive bacilli and trichomonads. Based on these findings, the bird was prescribed a course of amoxicillin/clavulanic acid and trimethoprim sulfamethoxazole. At a recheck examination two days later, there was no improvement in the bird's condition and a bacterial culture swab was collected and submitted for aerobic and anaerobic culture. The bird died four days following the recheck examination.

On external postmortem examination, the bird was noted to be in poor body condition, with markedly atrophic pectoral muscle mass. The crop was markedly distended, and tan mash-type feed was adherent to the beak and the cere. There was green staining and mild urate pasting around the vent.

Internally, the crop was thin walled and contained abundant mash-type feed. The proventriculus was flaccid and empty. The gizzard contained several small smooth fragments of metal, a small screw, and a piece of thin-gauge copper wire that was embedded into the gizzard and surrounded by fibrosis (**Fig. 1**). The koilin was stained dark green. Internal fat stores were absent. Based on the gross findings of multiple metallic foreign bodies in combination with presumptive neuropathic paralysis of the crop, liver was sent for quantitative lead levels and routine tissues were sampled for histology.

Figure 1. Multiple foreign bodies removed from ventriculus.

On microscopic examination, the renal tubular epithelium frequently contained round, amphophilic and acid-fast intranuclear inclusion bodies, typical of lead (**Fig. 2)**. There was accumulation of abundant brown granular pigment within hepatocytes. There was also a focus of inflammation in one section of lung that was compatible with aspiration pneumonia. Subtle vacuolation was noted in the brain, as well as dilation of myelin sheaths in the peripheral nerves. Toxicological testing identified significantly elevated liver lead levels at 73 micrograms/g (normal reference intervals in chickens 0.1-0.5 mg/g), and therefore, lead toxicosis was confirmed.

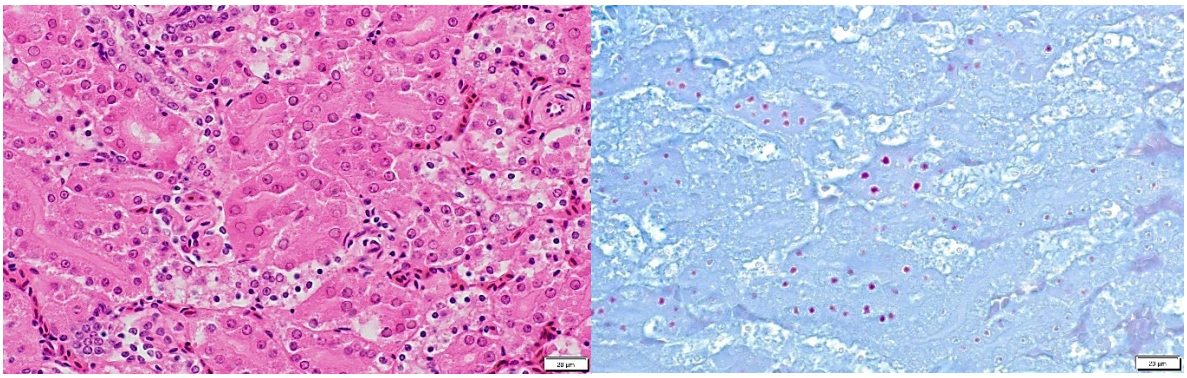

Figure 2. Kidney, H&E, 60X. Intranuclear inclusion bodies in renal tubular epithelium (left).
Kidney, Ziehl-Neelsen, 60X. Acid-fast intranuclear inclusion bodies in renal tubular epithelium (right).

Lead poisoning is well described in avian species who consume lead in the form of various environmental contaminants, including: paint, lead ammunition, fishing weights, grease, or contaminated foliage (typically near smelters, mines, waste dumps, or along roadsides). It is suspected that columbiform birds consume metallic fragments from the environment to serve as grit, as opposed to raptor species who tend to ingest metallic fragments from the gut of prey species. Once ingested, the lead

is rapidly absorbed into the bloodstream, redistributed to the soft tissues, particularly the kidneys and liver, and subsequently deposited in bone.

The elevated blood lead levels impact various metabolic functions, including sulfhydryl-containing enzyme activity and mitochondrial function, eventually leading to neurotoxicity, anemia and gastrointestinal dysfunction. Clinical signs of lead intoxication in birds include weakness, altered mentation, tremors/seizures, regurgitation, loss of condition, muscular atrophy, and green discolouration of the urates. Antemortem diagnosis can be made by assessing blood lead levels, and radiographs can be performed to identify metallic foreign bodies in the gastrointestinal tract. Treatment typically involves removing the source of lead from the gastrointestinal tract, followed by chelation. When there is extensive tissue damage, particularly with neurological involvement, treatment is typically unsuccessful. *AHL*

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2. DeMent SH, et al. Toxic lead exposure in the urban rock dove. J Wildl Dis 1987;23(2):273-278.
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Egg Drop Syndrome ’76: Keep on your rule out list

Emily Martin, Davor Ojkic

Animal Health Laboratory, University of Guelph, Guelph, ON.

AHL Newsletter 2023;27(3):18.

A disease that was originally described as “Egg Drop Syndrome ’76” is caused by duck adenovirus 1 (DAdV-1) infection. The agent, DAdV-1, is still often referred to as “egg drop syndrome virus” – EDSV. Previously DAdV-1 was called “group III avian adenovirus” but is now classified as a member of *Duck atadenovirus A* species of the genus *Atadenovirus*.

Since 2019 there have been multiple outbreaks of “Egg Drop Syndrome” associated with DAdV-1 infection in the USA affecting commercial layer and broiler breeder chickens. EDS has also been previously identified in commercial ducks in Ontario (2009) and Quebec (2019). The identification of this virus in the USA and Quebec since 2019 is concerning given that this virus is known to exist in wildlife, therefore, this virus should continue to be a rule out in cases of egg production drops. Infection primarily affects the oviduct of laying chickens replicating in the oviduct, nasal mucosa and spleen. This results in the production of pale, thin-shelled, soft-shelled or shell-less eggs. There is also a drop in egg production. Shell-less eggs may be missed if they rupture (leaving only membranes) or if they are eaten by the birds.

Clinical presentation: The affected birds typically have no other overt clinical signs other than poor egg quality and production problems. The fall in egg production can be variable (rapid or weeks, up to 40% drop), outbreaks last 4-10 weeks, and there is no effect on fertility or hatchability. The age of initial infection can affect the clinical signs (i.e., vertical, day of age, in lay,) resulting in 3 possible presentations (**Table 1**).

Table 1. Egg Drop Syndrome '76 patterns of disease.

Classic	Primary breeding flock. Virus transmitted vertically through egg. Latent infection until onset of egg production. Virus shed in eggs and droppings – infect susceptible contacts.
Endemic	Horizontal infection during lay. Usually commercial egg layers. Horizontal transmission by egg trays between flocks. Outbreaks often associated with shared egg-packing station.
Sporadic	Direct or indirect contact with: - domestic ducks or geese - contaminated water supply (wild waterfowl droppings) May lead to endemic disease.

Transmission: Vertical transmission can result in a latent infection. When birds come into egg production the virus can be reactivated, excreted and disseminated through lateral spread. The virus can also be spread by fomites (equipment, egg trays), or reused needles during vaccination. Sporadic spread can occur from domestic or wild ducks or geese through contaminated droppings or drinking water. The incubation period is 7-9 days.

Gross lesions: Potential lesions include mucosal edema and exudate in the shell gland, mild splenomegaly, flaccid ovules, and multiple eggs in various stages of development in the celomic cavity. Alternatively, birds could have regressed ovaries and oviducts.

Diagnosis: It may be difficult to select birds for examination due to the lack of clinical signs, slow spread through flock, and short duration of virus replication/lesions. DAdV-1 (EDSV) PCR testing is available at the AHL; suitable samples include shell membrane of shell-less eggs or cloacal swabs. A positive PCR and detection of antibodies coincident with a drop in egg production or shell abnormalities is considered diagnostic. As serological testing is not available at the AHL, serum samples for a hemagglutination inhibition test will be forwarded to another commercial laboratory.

Since December of 2012, when the DAdV-1 (EDSV) PCR was introduced, 200 samples have been tested and only 2 commercial duck samples have been positive (**Table 2**).

Table 2. AHL DAdV-1 PCR results since 2012.

Count of Sample ID	**DAdV-1**	
Commodity	**Negative**	**POSITIVE**
Chicken	12	
Chicken, broiler-breeder	90	
Chicken, layer	25	
Chicken, layer-breeders	10	
Duck	19	2
Turkey, breeder	42	
Grand Total	**198**	**2**

Out of these 200 samples, 126 were also tested for IBV, another agent commonly suspected in egg production issues, and 90 (71.4%) were IBV-reactive (e.g., inconclusive and positive).

Table 3. DAdV-1 samples from Table 2 also tested for IBV.

Count of Sample ID	**IBV Result**

Commodity	Inconclusive	Negative	Not tested	POSITIVE	Total
Chicken			2	10	12
Chicken, broiler-breeder	4	28	22	36	90
Chicken, layer	6	8	2	9	25
Chicken, layer-breeders	6			4	10
Duck			21		21
Turkey, breeder		15	27		42
Grand Total	**16**	**51**	**74**	**59**	**200**

Control: There is no treatment available. Control is by eradication or vaccination of the breeder flocks. Laying hens provide maternal antibodies to the chicks. Ensure birds are not exposed to ducks, geese or open untreated water sources (ponds, rivers).

All species and ages of chickens are susceptible to developing disease. Other species that can be infected include quail and guinea fowl. The virus is resistant to inactivation and resistant to pH range 3–10. Treatment with 0.5% formaldehyde and 0.5% glutaraldehyde could neutralize infectivity. EDS '76 is immediately notifiable under the Ontario provincial Animal Health Act. *AHL*

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Leucocytozoonosis in multiple avian species

Emily Martin, Andrew Brooks, Felipe Reggeti, John R Barta, Natasha Slawnych, Perryn Kruth

Animal Health Laboratory, University of Guelph, Guelph, ON (Martin, Brooks, Reggeti); Department of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, ON (Barta, Slawnych, Kruth)

AHL Newsletter 2023;27(3):20.

Since the spring of 2023, the AHL has received 3 submissions of different avian species with a history of sudden death (cygnets, hawk, duckling) that are either confirmed or suspicious for leucocytozoonosis, a blood parasite of birds in the phylum *Apicomplexa*. The life cycle requires 2 hosts; black flies (*Simuliidae*) are the definitive host and transmit the parasite to birds who are the intermediate host. It is rare that we determine this parasite as the primary cause of death, as most cases have an underlying disease; however, in many areas of southern Ontario, the rainfall is normal to above average for this time of year. This provides an environment for black fly development since they lay their eggs in moving water. Once flies emerge, they have an average life span of 3 weeks.

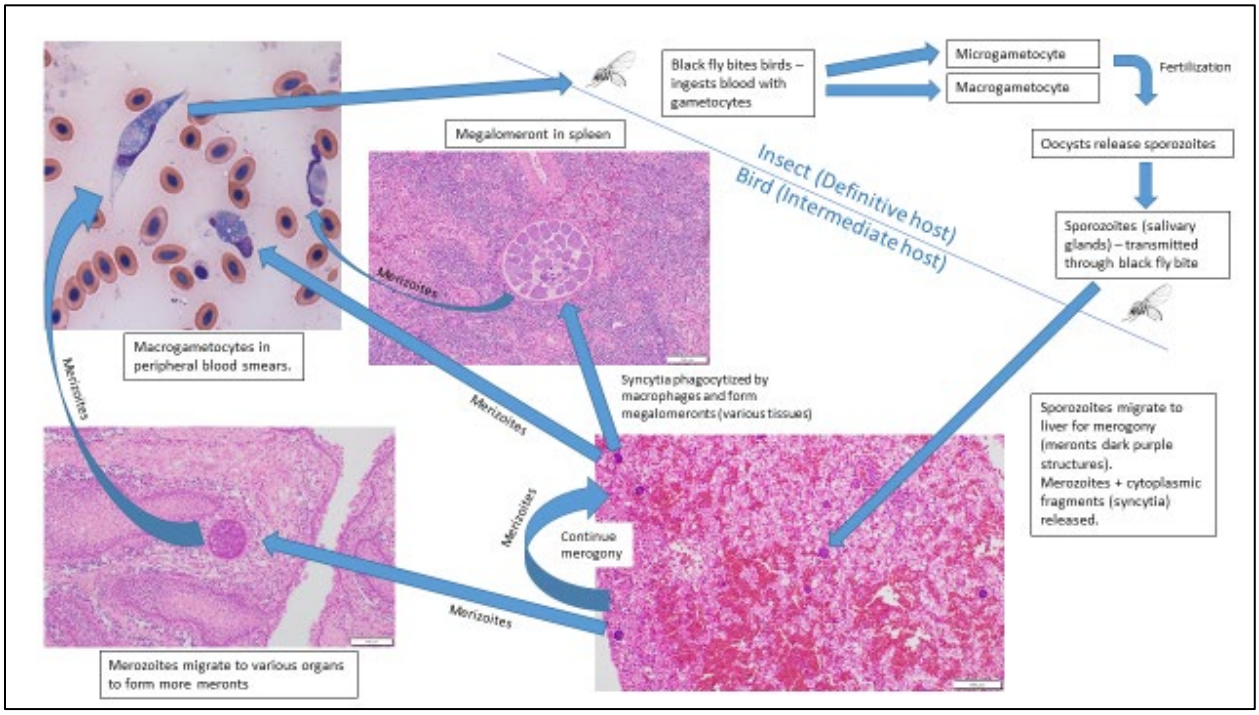

Figure 1. Life cycle of *Leucocytozoon* spp.

Clinical signs of leucocytozoonosis in birds can be related to the location of the developing parasite (**Fig. 1**):

- listlessness, pallor (anemia - parasite produces antierythrocytic factors);
- dyspnea (large gametocytes occlude pulmonary capillaries);
- CNS signs (megaloschizonts form in vessel endothelium that could occlude vessels causing multifocal necrosis – multiple organs).

Clinical signs are observed approximately 1 week after infection during the onset of parasitemia. Mortality can vary with strain, species, degree of exposure, age, and immune status. Death can occur within 24 hours of onset of clinical signs. Mortality can be up to 70% in ducklings, but is low in adult birds that may have decreased egg production and hatchability. Approximately 60% of deaths occur 11-19 days post exposure. Death can occur in commercial species as well, including turkeys, ducks, geese and chickens.

Acute disease is more common in areas with high black fly populations and in young birds with high parasitemia. Subacute or chronic disease develops in young birds during low insect exposure or in older birds (low parasitemia). NOTE: Recovered birds remain carriers and a reservoir of infection for young susceptible birds. On postmortem examination the blood can appear thin and watery. There may be splenomegaly, hepatomegaly, and hemorrhages as well as visible white foci in multiple organs (megaloschizonts).

Leucocytozoon spp. cannot parasitize other vertebrates, and have wide genetic diversity making diagnosis challenging. Diagnosis is by identification of gametocytes on blood smears, schizonts in tissue sections, or PCR. There is no treatment, and control aims to reduce or eliminate the insect vector from the environment of the birds.

In areas with high black fly populations and death primarily in young birds on a property, remember to include this disease on your list of rule outs. *AHL*

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HORSES

Testing for Eastern equine encephalitis virus and West Nile virus encephalopathies

Tim Pasma, Alison Moore

Animal Health Laboratory, University of Guelph, Guelph, ON (Pasma); Ontario Ministry of Agriculture, Food and Rural Affairs, Guelph, ON (Moore)

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Summer brings mosquitoes and unfortunately, they may be infected with West Nile virus (WNV) and Eastern equine encephalitis virus (EEEV). Depending on where you live in the province, EEEV or WNV may be endemic in your area. To see where previous cases of these diseases have occurred in Ontario and Canada, please refer to the Canadian Animal Health Surveillance System (CAHSS) equine dashboard: https://app.powerbi.com/view?r=eyJrIjoiOGFiMTAwYjAtZDkzZC00ZmZhLTk4ZTgtNGJjYmNjZjU3MmE4IiwidCI6IjE4YjVhNWVkLTFkODYtNDFkMy05NGEwLWJjMjdkYWUzMmFiMiJ9.

Although horses with either disease present with neurologic signs, the clinical signs of EEE are typically more acute and severe with death occurring within 24-48 hours. Reported clinical signs include depression, ataxia, circling, and/or head pressing, with or without fever, progressing to recumbency and frequently followed by seizure activity. WNV is typically less severe with a much lower mortality rate. Clinical signs may include ataxia, muscle fasciculations, and mild depression, with or without fever. However, in rare cases, horses progress rapidly to recumbency and require euthanasia within 48 hours of clinical onset.

The most reliable antemortem test which confirms these encephalitic infections is the serum EEEV IgM ELISA (code xeeevme) and WNV IgM ELISA (code xwnveq), respectively. However, vaccination for either virus within the previous month will interfere with interpretation of the test result, as recently vaccinated horses will also test positive. For postmortem testing, the brain can be submitted for EEEV or WNV PCR testing.

Since both of these diseases usually occur in unvaccinated horses, rabies should also be on the differential list. If rabies is suspected, contact the local public health unit if there is human contact with the saliva of the horse. If there is animal involvement only, contact the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA) for assistance with risk assessment, rabies sample submission and management of rabies exposure cases. The OMAFRA Rabies Response Request form is available at https://survey123.arcgis.com/share/f24efaeca671431a8f8555f0400644d1 or by phone at 1-877-424-1300.

If the brain is collected for rabies testing, one can ask the technician or pathologist to retain a piece of brain for EEEV/WNV PCR testing, should the rabies test be negative. Depending on the clinical signs, EHV-1 should also be on the differential list for acutely neurologic horses. The preferred samples for EHV-1 PCR testing are nasal swab and whole blood (live), or brain tissue (dead). *AHL*

Canine distemper virus infection of vaccinal origin in a 14-week-old puppy

Emily Rätsep[1] and Davor Ojkic[2]

Animal Health Laboratory, University of Guelph, Kemptville, ON[1] and Guelph, ON[2]

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Canine distemper virus (CDV) is a morbillivirus in the *Paramyxoviridae* family, causing subacute to acute systemic and/or neurological disease in dogs and other carnivorous species. With the increased use of a modified live-virus vaccine against CDV, infections in domestic animals have decreased. Although uncommon, CDV infections can still occur in vaccinated animals. Emergence of new wild type strains resulting in vaccine failure, complications by lingering maternal immunity, and reversion to virulence of a modified life vaccine have all been reported.

A 14-week-old puppy presented for postmortem following a history of intermittent issues with anorexia and lethargy since 10 weeks of age. Various potential causes including intussusception, intestinal foreign body, hypothyroidism, hepatic shunt and cardiac disease were ruled out with no change in presentation. Initial vaccines (canine morbillivirus, adenovirus, parainfluenza and parvovirus) were administered at 7 and 12 weeks. The 12-week vaccine was later than desirable as the puppy was too sick for vaccination at an earlier date. The puppy had not yet been vaccinated for rabies. The puppy's clinical signs progressed to pyrexia and lethargy with pruritic skin rash, green mucoid nasal discharge, decreased mentation, dysphagia, muscle twitches and ventral strabismus of the right eye within 48 hours of euthanasia. Focal seizures (4 in one day) were the ultimate reason for electing euthanasia.

On gross postmortem examination, a mucopurulent rhinitis and pulmonary edema were noted. Histologically, changes in the lungs were far more florid and consisted of marked necrosis with fibrin deposition and abundant neutrophilic inflammation (**Fig. 1**). A neutrophilic and lymphohistiocytic rhinitis was confirmed. The cortical grey and white matter of the brain contained rare small glial nodules (**Fig. 2**). Rare individual neutrophils were occasionally seen in the choroid. Other histological findings included tracheitis (presumed secondary to the pulmonary changes), and a portal bridging fibrosis with mild biliary hyperplasia (significance undetermined). The combination of the initial gross and histological findings suggested acute septicemia and aspiration pneumonia were possible causes of the decline in this puppy. While bronchopneumonia was present, no distinct syncytial cells or viral inclusions were observed in any tissue, nor were any histological changes supportive of demyelinating leukoencephalitis observed. Other findings typically associated with canine distemper such as enamel hypoplasia, hyperkeratosis of the footpads and conjunctivitis were also not observed.

Considering the clinical history and change in mentation, testing for canine distemper virus (CDV) by PCR was carried out on lung and brain tissue, and both were PCR-positive for CDV with cycle threshold numbers of 22.09 and 25.26, respectively. Immunohistochemistry was performed on brain for further confirmation, and CDV was detected (**Fig. 3).** Moderate growths (2+) of *Staphylococcus pseudintermedius* were cultured in both tissues also, suggesting that a concurrent bacterial infection was present, possibly secondary to aspiration and systemic spread, explaining some of the lesions observed in the lung and brain.

The PCR-positive sample from the brain was genotyped, and was a 99.9% match to the Rockborn strain of CDV (**Fig 4**), providing evidence to support the hypothesis that the disease in this case was vaccinal in

origin. The Rockborn CDV strain was originally isolated in Sweden and attenuated in canine kidney cells, and has been used since the 1960s in several live-attenuated CDV vaccines.

Vaccine-associated canine distemper infections have been reported in multiple breeds, usually following vaccination with attenuated or modified-live canine distemper virus vaccines. Clinical signs typically develop within 3 weeks of vaccination, and can include seizures and circling, or changes in behaviour. Reversion to virulence most commonly occurs in immunosuppressed animals (immunocompromised or animals with concurrent infections, including parvovirus), or is demonstrated in isolated cases as a post-vaccinal encephalitis. In these and in peracute cases, characteristic inclusion bodies or prominent demyelination may be absent; therefore, demonstration of the CDV antigen in neurons is required to confirm the diagnosis.

In this case, CDV antigen was expressed in neural tissue (IHC) and confirmed by PCR testing of the lung and the brain. Additionally, CDV hemagglutinin gene sequencing allowed for confirmation that the virus was identical to a vaccine strain. This case serves as a reminder of the possibility of reversion to virulence when using modified-live vaccines, and the importance of assessing general health and wellness of an individual prior to vaccination. *AHL*

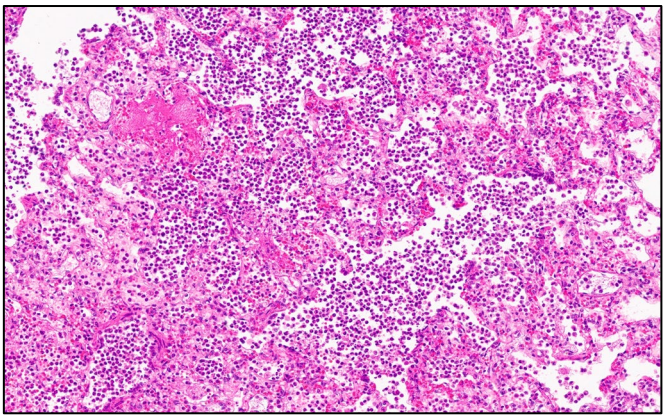

Figure 1. Lung from a 14-week-old puppy. There is necrosis of alveolar walls with fibrin deposition and numerous macrophages and degenerate neutrophils (H&E stain, 10x)

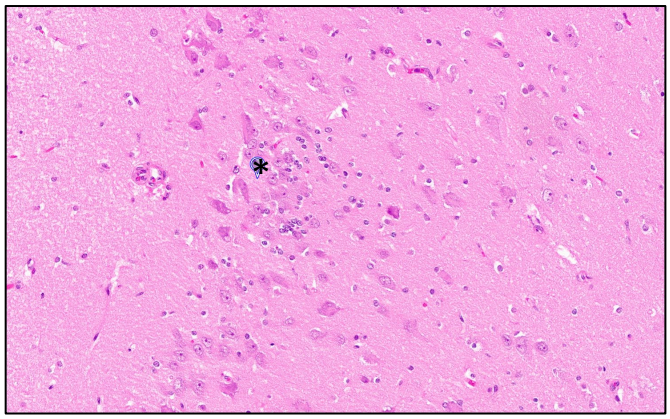

Figure 2. Brain from a 14-week-old puppy. There are rare clusters of increased glial cells (*) in the cortical grey mater (H&E stain, 20x)

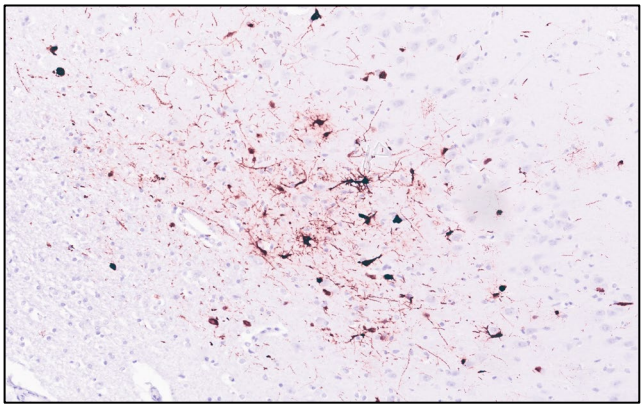

Figure 3. Brain (hippocampus) from a 14-week-old puppy. Clusters of neuronal cell bodies and nerve processes are intensely positive for expression of the canine distemper virus antigen (IHC stain canine distemper virus, 20x)

Percent Identity

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	
1		92.1	99.9	91.2	92.2	99.9	92.1	92.0	91.8	92.0	99.8	91.9	99.9	99.9	99.9	92.0	92.1	1
2	7.8		92.6	99.2	99.5	92.6	99.5	98.9	99.5	99.4	92.6	98.8	92.7	92.7	92.7	98.8	97.0	2
3	0.1	7.4		91.8	92.6	99.7	92.6	92.4	92.2	92.5	99.9	92.2	99.9	99.9	99.9	92.5	92.4	3
4	8.5	0.8	8.0		99.0	91.8	99.0	98.2	98.6	98.9	91.8	98.1	92.0	91.9	91.9	98.1	96.3	4
5	7.8	0.5	7.5	1.0		92.6	99.7	98.8	99.2	99.7	92.6	98.7	92.7	92.7	92.7	98.7	96.9	5
6	0.1	7.5	0.3	8.1	7.5		92.5	92.3	92.2	92.4	99.6	92.2	99.7	99.7	99.7	92.4	92.4	6
7	7.9	0.5	7.5	1.1	0.3	7.6		98.8	99.1	99.7	92.5	98.6	92.7	92.6	92.6	98.6	96.9	7
8	7.9	1.1	7.6	1.8	1.2	7.7	1.3		98.6	98.7	92.3	99.6	92.5	92.4	92.4	98.8	97.2	8
9	8.1	0.5	7.8	1.3	0.8	7.8	0.9	1.5		99.0	92.2	98.4	92.4	92.3	92.3	98.4	96.7	9
10	8.0	0.6	7.6	1.1	0.3	7.7	0.3	1.3	0.9		92.4	98.6	92.6	92.6	92.6	98.6	96.8	10
11	0.1	7.5	0.1	8.1	7.5	0.4	7.6	7.7	7.8	7.7		92.2	99.8	99.9	99.9	92.4	92.4	11
12	8.0	1.3	7.8	1.9	1.3	7.8	1.4	0.4	1.6	1.5	7.8		92.4	92.3	92.3	98.7	96.9	12
13	0.0	7.3	0.1	7.8	7.3	0.3	7.4	7.5	7.6	7.5	0.2	7.6		99.9	99.9	92.6	92.6	13
14	0.0	7.3	0.1	7.9	7.4	0.3	7.5	7.6	7.7	7.5	0.1	7.7	0.1		100.0	92.6	92.5	14
15	0.0	7.3	0.1	7.9	7.4	0.3	7.5	7.6	7.7	7.5	0.1	7.7	0.1	0.0		92.6	92.5	15
16	7.9	1.3	7.6	1.9	1.3	7.6	1.4	1.2	1.6	1.5	7.6	1.3	7.4	7.5	7.5		97.2	16
17	7.9	2.9	7.7	3.6	2.9	7.8	3.0	2.8	3.2	3.1	7.8	2.9	7.6	7.6	7.6	2.8		17
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	

1 Brain_puppy
2 DQ903854_CDV_Lederle
3 EF095750_CDV_vaccine-Hungary
4 EU143737_CDV_Onderstepoort
5 FJ461701_CDV_NobivacDHPPI
6 FJ461702_CDV_VanguardPlus
7 FJ461709_CDV_NobivacPuppyDP
8 FJ46170_CDV_GalaxyDA2PPV
9 FJ461710_CDV_Canigen-DHPPI
10 FJ705237_CDV_Vacc-N
11 FJ705238_CDV_Vacc-P
12 FJ705239_CDV_Vacc-Q
13 GU266280_CDV_Rockborn-Candur
14 GU810819_CDV_Rockborn
15 MN702774_CDV_CanishotK5
16 Z35493_CDV_Convac
17 AF259552_CDV_SnyderHill

Figure 4. Sequence distances comparing the clinical sample with CDV hemagglutinin gene sequences from GenBank. Percent identity in upper triangle; percent divergence in lower triangle.

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Infection with highly pathogenic avian influenza (HPAI) H5N1 in an Ontario dog

Josepha DeLay, Davor Ojkic, Yohannes Berhane, Carissa Embury-Hyatt, Cindy Boeve

Animal Health Laboratory, University of Guelph, Guelph, ON (DeLay, Ojkic); National Center for Foreign Animal Disease, Canadian Food Inspection Agency, Winnipeg, MB (Berhane, Embury-Hyatt); Ritson Veterinary Clinic, Oshawa, ON (Boeve)

AHL Newsletter 2023;27(3):26.

In March 2023, a 9-year-old castrated male golden retriever dog was lethargic and hyporexic 48 hours after scavenging on a Canada goose carcass. The dog presented to a veterinarian 72 hours after exposure, at which time it was pyrexic (T 40.2 C / 104.4 F) and was treated with antibiotics. The dog developed

diarrhea and acute respiratory distress later that same day, and died before further intervention was possible.

The dog was submitted to the AHL for postmortem examination. Due to an ongoing highly pathogenic H5N1 avian influenza (HPAI) outbreak in Ontario, and a history of exposure to a wild bird carcass, the dog's body was tested for influenza A virus prior to examination. An H5 subtype influenza A virus was detected by PCR in oropharyngeal swabs. Infection of the goose carcass with H5 influenza A virus was also confirmed. Based on the results of screening tests in both the dog and the scavenged goose, infection of the dog with HPAI was considered likely. As a result, enhanced biosecurity measures and personal protective equipment (PPE) were used for the postmortem exam of the dog.

Gross lesions in the dog targeted lung and adrenal glands. Red discoloration and rubbery consistency involved 85% of right lung and 15% of left lung. The left adrenal gland was small and difficult to locate; the right adrenal gland could not be definitively identified. Histologically, there was acute suppurative pleuritis with minimal alveolar hemorrhage, necrosuppurative tracheobronchial lymphadenitis, and severe bilateral adrenocortical atrophy. In lung, a thin segmental to continuous band of neutrophils and macrophages was present in the pleura. In some sites, few similar inflammatory cells extended into subpleural alveoli. Focal necrosis and fibrin exudation was present in one tracheobronchial lymph node. Bilaterally in adrenal glands, cortices were thin and consisted only of zona glomerulosa. Zona fasciculata and zona reticularis were replaced by a narrow band of plump macrophages.

Influenza A virus was detected in lung and brain by PCR. Similar to initial oropharyngeal swab PCR results, the PCR product was typed as H5. Further testing for HPAI was carried out by the Canadian Food Inspection Agency (CFIA) due to the reportable disease requirements for this pathogen. CFIA confirmed H5N1 avian influenza virus in lung and spleen by virus isolation.

Histologic lesions in lung and lymph node of this dog, consisting of suppurative pleuritis and fibrinonecrotizing lymphadenitis, were not typical of influenza and were more suggestive of a bacterial etiology. Bacterial culture from lung yielded moderate growths of *E.coli* and *Klebsiella pneumoniae*, and minimal growth of *Staphylococcus hominus*. However, immunohistochemical (IHC) procedures carried out both at the AHL and by CFIA laboratories, and using 2 different influenza A virus-specific antibodies, confirmed the presence of influenza virus antigen in association with lesions in both lung and tracheobronchial lymph node. Viral antigen was also detected in brain, although there was no microscopic evidence of inflammation or necrosis in this site. In addition, influenza virus nucleic acid was detected in lung, tracheobronchial lymph node, and tonsil by *in situ* hybridization (ISH).

Sporadic infection with HPAI has been reported in humans, but human-to-human transmission has not been documented. Human cases are most often considered sporadic spill-over events in workers involved in culling activities. In wild and domestic carnivores, including dogs and cats, exposure to the virus is assumed or confirmed to be associated with ingestion of infected birds. In the dog in this report, infection with HPAI was confirmed and is considered significant with regards to the cause of the dog's death, especially in the context of clinical evidence of pyrexia. Lung lesions associated with influenza virus infection were atypical for this pathogen in mammals but were nonetheless significant. Neurotropism and significant lesions in the central nervous system (CNS) are described in many cases of HPAI infection in other species. Although CNS infection in this dog was confirmed by IHC, no associated microscopic lesions were evident.

Concurrent bilateral adrenocortical atrophy in this dog likely resulted in some degree of adrenocortical insufficiency. The relative contribution to death from both acute infection with HPAI and chronic adrenocortical atrophy / insufficiency is difficult to determine without benefit of antemortem adrenocortical function testing. The cause of adrenocortical atrophy in dogs is often undetermined (idiopathic), although an immune-mediated pathogenesis is suggested. *AHL*

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Mycobacterium bovis pneumonia in an imported cat

Josepha DeLay, Mirjana Savic, Olga Andrievskaia, Felix Gwak

Animal Health Laboratory, University of Guelph, Guelph, ON (DeLay); Canadian Food Inspection Agency, Ottawa, ON (Savic, Andrievskaia); Ashcott Veterinary Clinic, Scarborough, ON (Gwak)

AHL Newsletter 2023;27(3):28.

Mycobacterium bovis was confirmed as the cause of granulomatous pneumonia in a 1-year-old Oriental Longhair cat submitted to the AHL for postmortem examination. The cat had died unexpectedly following a short period of weight loss and lethargy. Antemortem clinical evaluation and treatment had not been pursued. The cat was imported to Canada from China 4 months prior to death.

Significant gross postmortem lesions were limited to the thoracic cavity. Lungs had a diffuse mottled tan-gray color and multiple small foci of hemorrhage (**Fig. 1**). Lung parenchyma was firm, and lungs failed to collapse. Tracheobronchial lymph nodes were enlarged to approximately 2-3x normal size, and had a diffuse light red color on the capsular surface and upon sectioning.

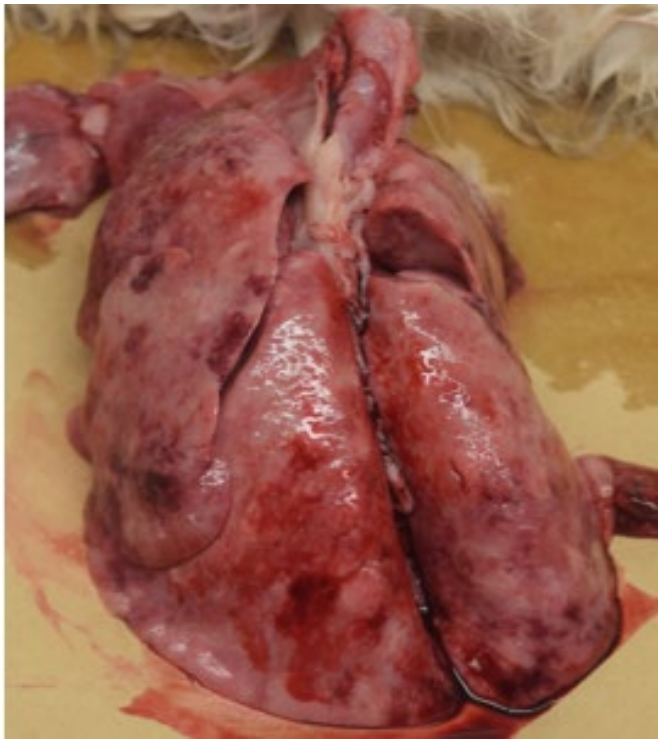

Figure 1. Gross lesions of *M. bovis* granulomatous pneumonia in a cat. Lungs are firm, mottled tan-gray with multiple hemorrhagic foci, and failed to collapse.

Histologically, lung lesions were severe and diffuse. Aggregates of macrophages infiltrated and effaced most alveoli, obscuring normal pulmonary architecture (**Fig. 2**). In some sites, inflammatory cell aggregates were centered on small foci of coagulation necrosis. Small loose clusters of lymphocytes, few neutrophils, and scant fibrin were occasionally intermingled with epithelioid macrophage. Hemorrhage flooded low numbers of alveoli. In tracheobronchial lymph nodes, multiple small clusters of plump epithelioid macrophages in cortex and medulla were occasionally centered on small necrotic foci.

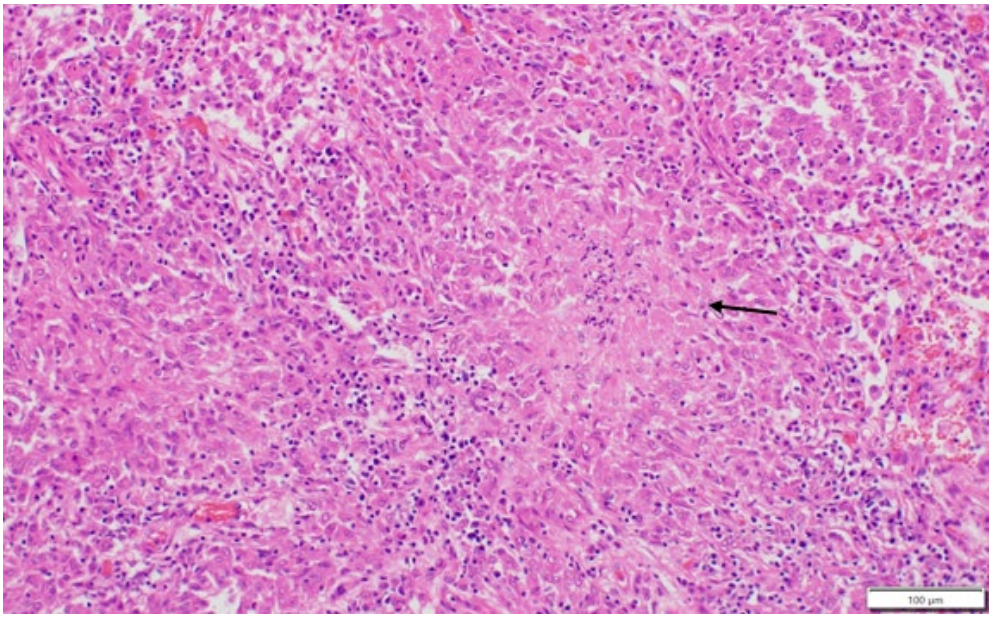

Figure 2. Histologic lesions of *M. bovis* granulomatous pneumonia in a cat. Alveoli are infiltrated and normal pulmonary architecture is obscured by large numbers of densely packed macrophages intermingled with fewer lymphocytes and neutrophils, and small necrotic foci (arrow) (H&E stain)

In lung sections stained with Ziehl-Nielsen and Fite's stains, rare faintly-staining, short acid-fast intracellular bacilli were detected in macrophages. The morphology and staining features of the bacilli were compatible with mycobacteria. No fungi were detected in lung sections stained with periodic acid-Schiff (PAS) stain.

Mycobacterial testing in lung samples from the cat was carried out by the Canadian Food Inspection Agency (CFIA). Nucleic acid of the *Mycobacterium tuberculosis* complex was detected in lung by PCR. The *M. tuberculosis* complex of organisms includes *M. tuberculosis*, *M. bovis*, and several other highly pathogenic, genetically-related mycobacteria causing similar disease conditions in animals and humans. The isolate from lung in this cat was confirmed as *M. bovis* by culture and subsequent speciation by a combination of biochemical tests and whole genome sequencing.

M. bovis infection in cats and other species is typically associated with ingestion of unpasteurized milk or inadequately cooked or raw meat from infected cattle. Lesions in cats most frequently involve the gastrointestinal tract and associated lymph nodes. Localization of *M. bovis* organisms to the respiratory tract, as in this cat, has been described in some cases. Gross lesions in this cat were unusual in the diffuse distribution of lung lesions without distinct nodule (tubercle) formation.

Increased importation of pets to Canada necessitates that veterinarians adopt a broader consideration of differential etiologic diagnoses and zoonotic risks when examining and treating these animals. Imported animals may have been exposed to pathogens that are not present in Canada. Information about an animal's country of origin or travel history should be included in the clinical history for new patients.

Veterinarians and staff should use appropriate personal protective equipment (PPE) when handling animals potentially infected with zoonotic pathogens.

M. bovis is a federally (Canada) and provincially (Ontario) reportable pathogen, and both CFIA and the Ontario Ministry of Agriculture, Food, and Rural Affairs (OMAFRA) must be notified of detection of this pathogen. Federal and provincial public health agencies must also be notified due to the significant zoonotic risk posed by *M. bovis*. *AHL*

References

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