Laboratory Services Division
Animal Health Laboratory

AHL Newsletter

AHL Newsletter, Volume 28, Number 2 **June 2024**

In this issue:

AHL Newsletter
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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.*
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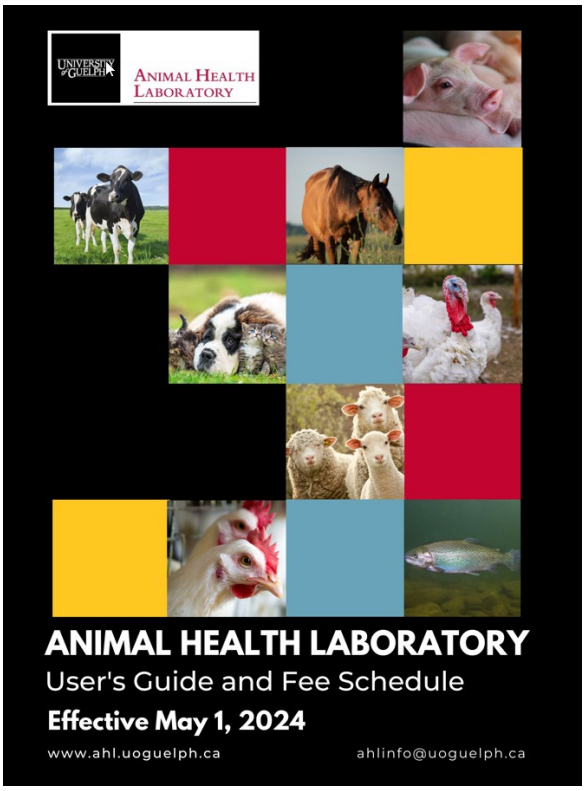
AHL User's Guide and Fee Schedule - May 1, 2024

Includes updated test information, new tests, new test panels, and more!

Mobile friendly! Available on-line at https://www.uoguelph.ca/ahl/

Test information is linked to LabNotes to facilitate test selection and interpretation of results.



New tests since May 2023:

- American Foulbrood - PCR
- *Avibacterium paragallinarum* - PCR
- Bacterial culture, aerobic and anaerobic, minimal inhibitory, companion/other
- Bacterial culture, aerobic, minimal inhibitory concentration, companion/other
- Bacterial culture, fecal, porcine
- Cache Valley virus - PCR
- Chicken rotavirus A/chicken rotavirus D/chicken parvovirus - PCR
- *Clostridium botulinum* (A, B, C, D, E, and F) toxin gene detection - PCR
- Fish *Flavobacterium branchiophilum* - PCR (Bacterial Gill Disease)
- Fish *Flavobacterium columnare* (Columnaris Disease) PCR
- Haemoplasma, Mycoplasma haemobos and Mycoplasma haemolamae - PCR
- Hatchery, environmental culture PCR
- Influenza A virus/Equine rhinovirus A/Equine rhinovirus B - PCR
- Mastitis, environmental culture Enterobacterales
- *Ornithobacterium rhinotracheale* - PCR
- Porcine sapovirus - PCR
- Porcine, bacteriology enteric panel 1
- *S. equi* sbsp *zooepidemicus* SzM - PCR
- Vitamin A and E, serum - HPLC
- Vitamin A, serum - HPLC

Update from the Director

The view from the Director's office

Spring is a period of change as we look forward to planting (gardens and fields), rearing young animals, and transitioning to the all-too-brief summer experienced here in Canada. Every May 1st, AHL publishes a new Fee Schedule and User's Guide (page 2) with helpful information on submitting samples to the laboratory for testing. This information is also available on the AHL website at https://www.uoguelph.ca/ahl/

There are significant changes in several bacteriology tests at AHL, related to upgraded technology that expedites screening (see poultry environmental sample testing update) and provides more relevant and accurate results (see AHL testing update for companion animals and other (exotic) animal species). Additional important updates for the poultry industry include a change in fowl adenovirus reporting, and a report on avian metapneumovirus, a notifiable disease that has been confirmed in multiple premises in Ontario.

The staff highlights section of this newsletter describes some notable staffing changes at AHL. After 26 years, Nick Schrier is retiring from his role as technical supervisor of the Toxicology laboratory. We are grateful for Nick's technical expertise, his willingness to develop new tests for clients, his attention to quality assurance, and his collegiality. We welcome Jeffrey Charters, currently working in the Toxicology lab, as our new Toxicology supervisor. Also starting in the AHL Guelph anatomic pathology unit is Dr. Lisa Gordon. Welcome Jeff and Lisa! Best wishes as you retire Nick!

We wish everyone an enjoyable and relaxing summer.

Maria Spinato, Director

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

Proper packaging and shipping of high risk samples - how to prevent leaks

Tim Pasma, Andrew Brooks

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

AHL Newsletter 2024;28(2):4.

When shipping bodies for postmortem at the AHL, it is important to package the submission carefully to prevent leaks. Leaking packages are a potential zoonotic risk to clinic staff, courier drivers and laboratory staff.

The body should be packaged in accordance with the standards for Transportation of Dangerous Goods type P650 packaging (formerly referred to as a 1B container). This packaging should contain 3 layers: 2 layers of inner packaging and 1 layer of outer packaging.

For inner packaging, wrap the body in leak proof material (e.g., triple wrapped in plastic bags), surrounded by absorbent material. Next, add the layer of secondary leak proof packaging (e.g., a biohazard bag). Ice packs (not ice cubes) can be added as required to keep the sample cool, especially in hot weather.

The outer packaging should be a rigid impervious container such as a Rubbermaid tote or plastic container with latches.

If you have a large or unusual specimen, please contact us first to discuss the plan for shipping.

Please plan your shipping so that samples do not arrive or become delayed in transit on weekends or holidays. *AHL*

OAHN Update – June 2024

Mike Deane, Tanya Rossi

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

This spring, the Ontario Animal Health Network has been very busy, preparing reports, resources, and completing research projects. To view any of our network reports and research projects, go to OAHN.ca and navigate to the species you are interested in.

Raw Food Info Sheet

The OAHN Companion Animals network, along with the Worms and Germs blog, has created an info sheet on raw meat diets for pets. This resource addresses the raw diet controversy, types of raw meat diets, risks associated with these diets, outbreaks associated with raw diets, how to reduce the risk, and what pets should never be fed raw food. View the info sheet here: https://www.oahn.ca/resources/raw-diets-infosheet/

OAHN Wildlife Project: Pilot Surveillance for Avian Influenza Virus (AIV) in Feral Cats

The OAHN Wildlife network is currently collecting samples for its project on avian influenza virus in feral cats. To learn more about this project, and find out the sampling and submission procedure, click here: https://www.oahn.ca/resources/oahn-wildlife-project-pilot-surveillance-for-avian-influenza-virus-aiv-in-feral-cats/

Chronic Wasting Disease Webinars

The OAHN Wildlife network organized two very informative and well attended webinars in April. Dr. Cory Anderson covered: carcass management and disposal, important strategies in maintaining disease-free jurisdictions, and the one health implications of surveillance for cross-species CWD transmission that CIDRAP has launched. View the recording here: https://www.oahn.ca/resources/oahn-cwd-and-one-health-update-with-dr-cory-anderson-recording/

Dr. Sam Allen presented on CWD and brucellosis surveillance in Wyoming cervids, and the challenges and opportunities of CWD management in a highly infected, endemic area. View the recording here: https://www.oahn.ca/resources/oahn-cwd-and-one-health-update-with-dr-cory-anderson-recording/

New Reports

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

Poultry Network – https://www.oahn.ca/reports/oahn-poultry-expert-network-quarterly-veterinary-report-q1-2024/

- Modified results for fowl adenovirus PCR at AHL: Interpretation guidance
- Avian metapneumovirus: Cases on the rise in multiple US states
- Poultry veterinary survey highlights – Q1 2024

Equine Network - https://www.oahn.ca/reports/equine-veterinary-report-q4-2023/

- OAHN Equine Instagram and Facebook page
- Strangles resources
- Bits N Snips
- Network member reports
- Laboratory data Q4 – Highlights
- Equine research
- Disease surveillance summary

Bovine Network - https://www.oahn.ca/reports/oahn-bovine-expert-network-quarterly-veterinary-report-q4-2023/

- Global surveillance: Influenza A (H5N1) detected in U.S livestock
- Q4 Bovine data from AHL
- Salmonella Dublin Update 2023
- OAHN Mastitis report 2023
- Ontario bovine disease surveillance

Swine Network - https://www.oahn.ca/reports/swine-veterinary-report-q4-2023/

- Novel Influenza A- H3N2 cluster 2010.1 update
- Salmonella surveillance
- OAHN Veterinary clinical impression survey veterinary comments
- Porcine Circovirus Type II (PCV2)
- Porcine Epidemic Diarrhea (PEDV)/ Porcine Deltacoronavirus (PDCoV)
- Laboratory diagnostic reports
- Ontario slaughter statistics
- International disease topics of interest summary
- OAHN Project update: Porcine Hemagglutinating Encephalomyelitis virus

Staff highlights

Nick Schrier retired on May 31, 2024 after working in the AHL Toxicology laboratory as a Professional Scientist and Technical Supervisor for the past 26 years. Over that period, Nick has overseen many scientific and technological advances in the toxicology unit, and has trained many of the staff that continue to work in this unit. His positive attitude and collegial approach made him a valuable member of the AHL team and he will be missed. We wish him a long, happy and healthy retirement, full of satisfying hobbies (travelling and rehabbing old vehicles), and many memorable occasions with family and friends. Congratulations Nick!

Jeffrey Charters was the successful candidate for the position of Technical Supervisor, Toxicology laboratory. Jeffrey completed a BSc in Environmental Chemistry at Trent, and a MSc in Oceanography at UBC. He has been working in the AHL Toxicology laboratory since 2012. Jeffrey previously worked as a bike messenger in Toronto and collected seawater samples on an icebreaker. He enjoys biking, reading, dogs and recently completed his first Paris to Ancaster bike race. Welcome Jeffrey!

Dr. Lisa Gordon recently joined the AHL Guelph Anatomic Pathology unit for a 1-year contract position. After completing her DVM at OVC, Dr. Gordon worked as a mixed animal practitioner for 2 years before returning to Guelph for a DVSc program in anatomic pathology. During her program, she researched the effect of age on aquatic bird bornavirus-1 infection in turkeys. Lisa mentioned that she enjoys baking in her spare time, and is happy to be working with the Anatomic Pathology team at AHL. Welcome Lisa!

RUMINANTS

Ruminant lungworms

Jacob Avula, Tim Pasma

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AHL Newsletter 2024;28(2):9.

Sheep and goats are parasitized by 3 species of lungworms; namely, *Dictyocaulus filaria*, *Muellerius capillaris* and *Protostrongylus rufescence*. Of these, only *Muellerius capillaris* has been isolated from cases here in AHL. To date, there have been 60 positive cases, and a highly significant number of them were from goats.

Muellerius capillaris are very thin worms found in alveoli and coiled in pulmonary parenchyma, producing greyish nodules. Eggs laid by adult female worms hatch and the first stage larva (L1) crawl up the bronchi, reach throat and are swallowed and then passed out in the feces. These larvae need a snail or slug (intermediate host) for further development. Once they enter the snail, the larvae develop into the infective third stage. When the sheep/goat ingest these snails, the infective larvae are released in the gastrointestinal tract, and then start migrating to their site of predilection - the lung – where they grow to the adult stage.

The main clinical signs of lungworm infection in sheep or goats are dyspnea and persistent cough. They may also have reduced weight gains. Diagnosis is based on clinical signs and isolation of first stage larvae in feces using the Baermann test. The larvae measure 300-320 um and are easily identified by the presence of a kinked tail with accessory spine towards the end of the tail (**Fig. 1**). A time-series observation was made on *Muellerius capillaris* larval recovery using the Baermann apparatus at different time points after storing a positive sample in the refrigerator. The sample was set up in the Baermann on days 6, 13, 22 and 29 after collection, and larvae were successfully recovered at all time points.

Cattle are parasitized by only one species of lung worm; namely, *Dictyocaulus viviparous*. To date, 6 positive cases have been recorded at AHL. These worms are larger (4-8 cm) than *Muellerius* and live in smaller bronchi. The ovoviviparous eggs laid by the female worms are coughed up, swallowed and hatch as they move through the gastrointestinal tract. The L1 are passed out in feces where they develop to the infective third stage. Cattle eat the larvae while grazing, which then move through the intestinal wall and finally reach lungs, settling in bronchi.

The main clinical signs of infection are dyspnea, coughing with extended neck, nasal discharge and weight loss. The coughing sounds like a dry non-productive exhalation (‘husk’).

Diagnosis is based on clinical signs and harvesting of L1in feces using the Baermann apparatus. The larvae measure 300-360 um and have characteristic dark food granules in the intestinal cells (**Fig. 2**). A time-series observation was made on *Dictyocaulus viviparus* larval recovery using the Baermann apparatus at different time points after storing the positive sample in the refrigerator. The sample was set up in the Baermann on days 3, 11 and 13 after collection, and larvae were successfully recovered at all time points.

A negative fecal result is not a proof of absence of the disease, especially in adult cattle with mild symptoms in which larvae are rarely found. For Baermann testing (AHL test code “ Baer”), at least 15-20 g of fresh feces for sheep, and 40-50 g for cattle are required. *AHL*

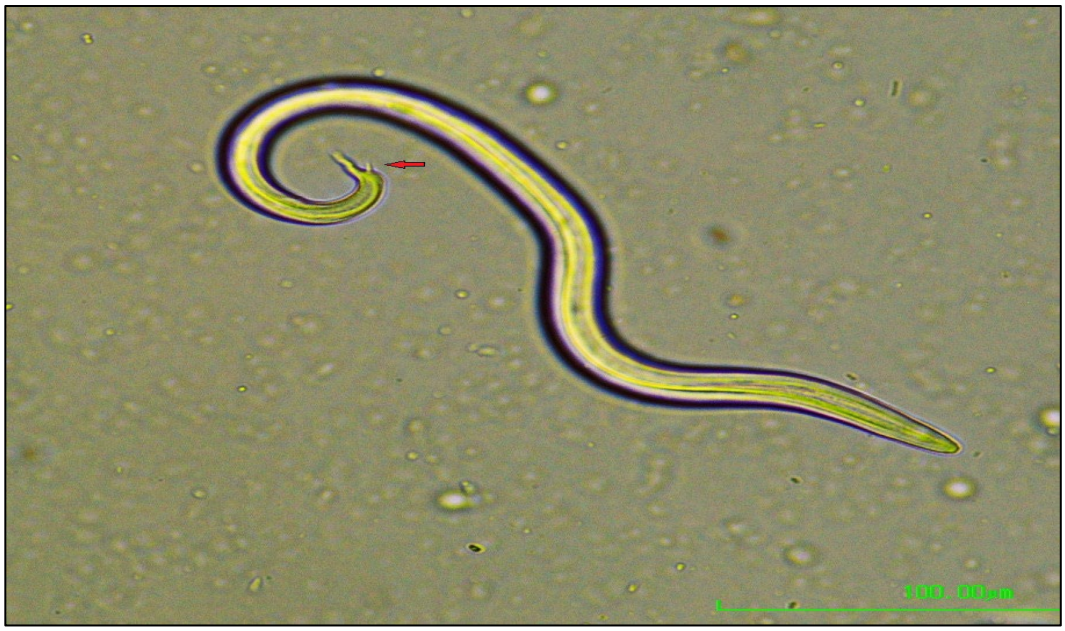

Figure 1: L1 of *Muellerius capillaris* with kinked tail and accessory spine.

Figure 2. L1 of *Dictyocaulus viviparus* shows dark food granules in the intestinal cells.

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Systemic vasculitis and lower urinary tract obstruction in a lamb caused by ovine herpesvirus-2

Emily Brouwer

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AHL Newsletter 2024;28(2):11.

An 8-week-old polled Dorset ram lamb presented to the referring veterinarian for a six-day history of stranguria and dribbling urine. During the physical examination, the veterinarian exteriorized the penis and was unable to identify any obstructions in the vermiform appendage but noted that the prepuce contained a thick fibrin cast. During the physical examination, the veterinarian observed that the animal was also having trouble breathing, and there were thick plaques of white exudate on the roof of the mouth. The veterinarian suspected urolithiasis and pneumonia, and treated the lamb with muscle relaxants, meloxicam, florfenicol, and penicillin. The lamb died two days later and was submitted to the Animal Health Laboratory for postmortem examination.

On gross examination, the hard palate, buccal gingiva, edges of the tongue, and oropharynx were extensively coated in thick mats of yellow-tan friable exudate that was loosely adhered to underlying erosions and deep mucosal ulcers (**Fig. 1**). These ulcers, lined by fibrinous exudate, extended the length of the esophageal mucosa to the level of the rumen. The preputial orifice and an approximately one-centimeter band of surrounding skin was bright red, alopecic and ulcerated.

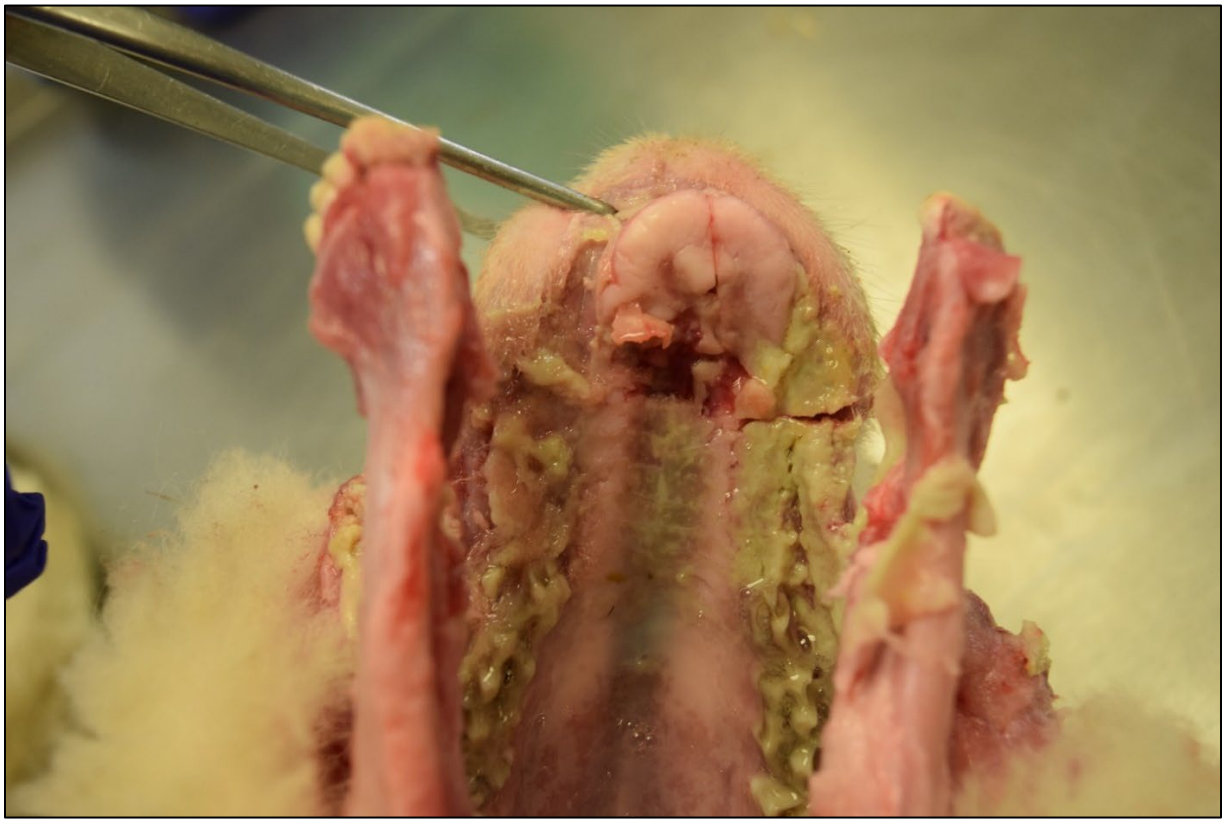

Figure 1. Mucosal ulcers covered by plaques of fibrin in the oral cavity.

Dissection of the urinary tract revealed mild dilation of the renal pelvis (hydronephrosis) with bilateral ureteral dilation (hydroureter), but the urinary bladder was empty. The mucosa of the urinary bladder was mottled pink and red with a dull surface. No obstruction was identified in the urethra, but the preputial mucosa was dark red, and the penis was adhered to the internal aspect of the prepuce by thick mats of fibrin. No vermiform appendage was identified.

The presence of hydronephrosis and hydroureter was compatible with the clinical diagnosis of lower urinary tract obstruction, and it was determined at postmortem examination that this obstruction was due to extrinsic blockage of the urethra due to extensive preputial inflammation. The process of exteriorizing the penis during physical examination likely removed the urethral blockage and allowed for micturition prior to submission.

Histopathologic examination identified widespread lymphocytic and neutrophilic vasculitis in various tissues, including the oral and esophageal mucosa, penis, prepuce, and heart. These areas of vasculitis were often associated with tissue necrosis, mucosal ulceration, and fibrinosuppurative inflammation. These histologic findings are compatible with the syndrome of systemic vasculitis caused by ovine herpesvirus-2, and subsequent PCR testing of the oral and esophageal mucosa was positive for OvHV-2 with cycle thresholds of 22.88 and 25.01, respectively.

Due to extensive oral ulceration, viral vesicular diseases, particularly Foot-and-Mouth disease (FMD), were considered. No coronary band lesions were identified, and thus a foreign animal disease was considered unlikely. Confirmatory negative testing for FMDV was performed as a precaution.

Ovine herpesvirus-2 is one of the ruminant gammaherpesviruses associated with Malignant Catarrhal Fever (MCF) in cattle and other ungulates. Sporadic cases of systemic vasculitis with clinical signs and lesions similar to MCF have been reported in Ontario sheep, and may present as excessive salivation, anorexia, and oculonasal discharge. Since sheep are the host-adapted species for this virus, infection is typically widespread and asymptomatic. The diagnosis is made when there are characteristic histologic lesions in conjunction with positive OvHV-2 PCR results. In situ hybridization has previously been performed on affected animals in Ontario and has demonstrated intralesional virus antigen in PCR-positive animals.

Lower urinary tract obstruction appears to be a novel clinical sign of this syndrome. *AHL*

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Listeriosis in farmed ruminants

Emily Rätsep,

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

AHL Newsletter 2024;28(2):12.

Listeriosis, caused by the bacterium *Listeria monocytogenes*, is an infectious disease that affects a wide range of mammalian species, including humans. This environmental bacterium is present in soil,

vegetable matter, and feces of healthy animals. Although ubiquitous, it can cause serious disease under certain conditions. This is most often seen in farmed ruminants due to feeding poorly fermented silage above a pH of 5.0-5.5. The bacteria optimally grow in temperature ranges between 30 and 37 °C, although they are also capable of reproducing in temperatures as low as -0.4 °C and as high as 45 °C. They also can reproduce in a wide pH range, from 4.5-9.6. While present in the natural environment, *Listeria* usually only results in sporadic disease and is considered non-contagious. However, outbreaks are known to occur when animals are exposed to a single contaminated source such as silage. Disease is generally occurs most often in winter and early spring, though outbreaks have also been reported in animals fed poor quality pastures or rotting vegetable matter. Moist preserved feed such as rotting hay or feed with significant soil contamination may also represent a potential risk.

Clinical disease can vary greatly and may include: neurological disease, sepsis, abortion, mastitis, or gastroenteritis. The most common disease form is generally considered to be listerial encephalitis which is often a chronic disease with approximately 4-6 weeks between exposure and presentation. The clinical course itself is generally rapid in sheep and, with death occurring within 24-48 hours after onset of neurological signs. In cattle the disease course is more chronic, lasting about 1-2 weeks. In these affected animals, neurological signs can vary, but are generally unilateral due to an ascending infection along the trigeminal nerve following penetration of the oral mucosa. Infection results in ataxia, nystagmus, dropped jaws, chewing problems, head tilt and cranial nerve deficits. Other less specific clinical signs such as fever, anorexia, depression, proprioception deficits, circling or head pressing can also be observed. Grossly, changes are minimal and often subjective. These can include meningeal congestion and occasionally reddening of the parenchyma of the brainstem. Histologic lesions are notable, consisting of microabscesses and perivascular cuffing primarily affect the brainstem (**Figs. 1, 2**).

Abortion or early neonatal mortality is the second most common manifestation of *Listeria* infection and is most often reported in small ruminants. In a pregnant ewe, bacteremia with subsequent placental and fetal infection can result in abortion, usually 5-10 days post exposure. If infection occurs later in pregnancy, it can result in stillbirths or early neonatal mortality due to sepsis. There are few gross lesions; however, areas of necrosis can be observed in the placenta, liver, spleen and heart. Histological changes include intercotyledonary placentitis characterized by multifocal areas of necrosis with neutrophils, and frequent, dense colonies of coccobacilli (**Fig. 3**).

Septicemia is most commonly recognized in neonates or pregnant ewes exposed to high numbers of bacteria. Rare presentations include dermatitis and gastrointestinal listeriosis typified by enteritis with diarrhea, possible hemorrhaging, and ulceration of the gastrointestinal mucosa. Listerial mastitis is rarely clinically apparent.

Diagnosis is dependent on characteristic histological lesions combined with results of bacterial culture on enriched media. At the AHL, an immunohistochemical stain is also available for *L. monocytogenes*, allowing for diagnosis in histological sections if no fresh tissue is available for culture.

It is important to recognize that exposed animals may become latent carriers and could potentially shed the bacteria in milk, thereby representing a potential zoonotic risk, particularly for immunocompromised people. Feces, urine, aborted fetuses and uterine discharge may also be potentially infectious; therefore, practicing good hygiene when handling known or suspected cases of listeriosis is of critical importance.
AHL

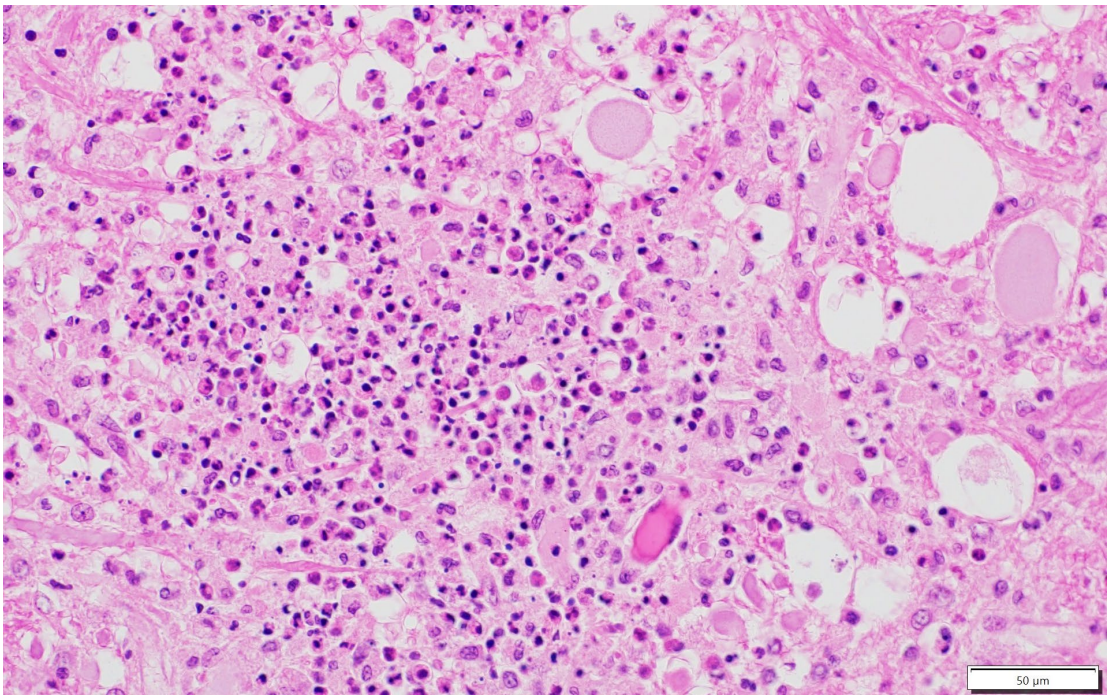

Figure 1. Goat brainstem. Necrosis of the parenchyma with microabscesses composed of neutrophils and macrophages. H&E stain, 40x.

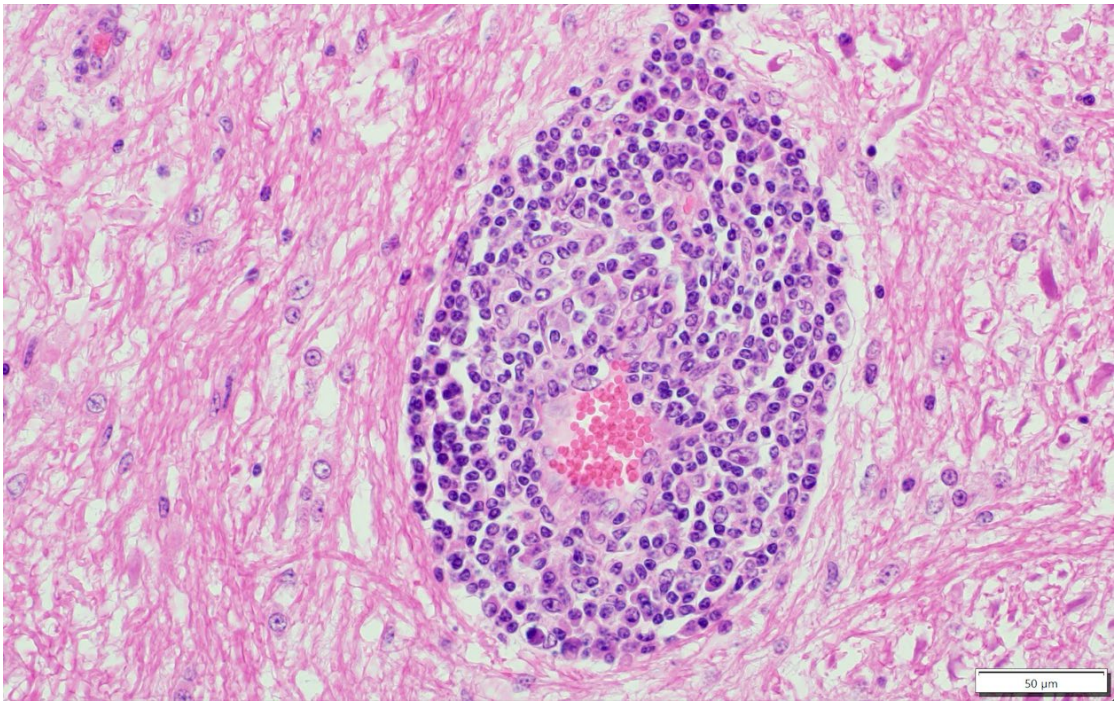

Figure 2. Bovine brain. Perivascular cuffing of cerebral blood vessels with macrophages, lymphocytes and fewer neutrophils. H&E stain, 40x.

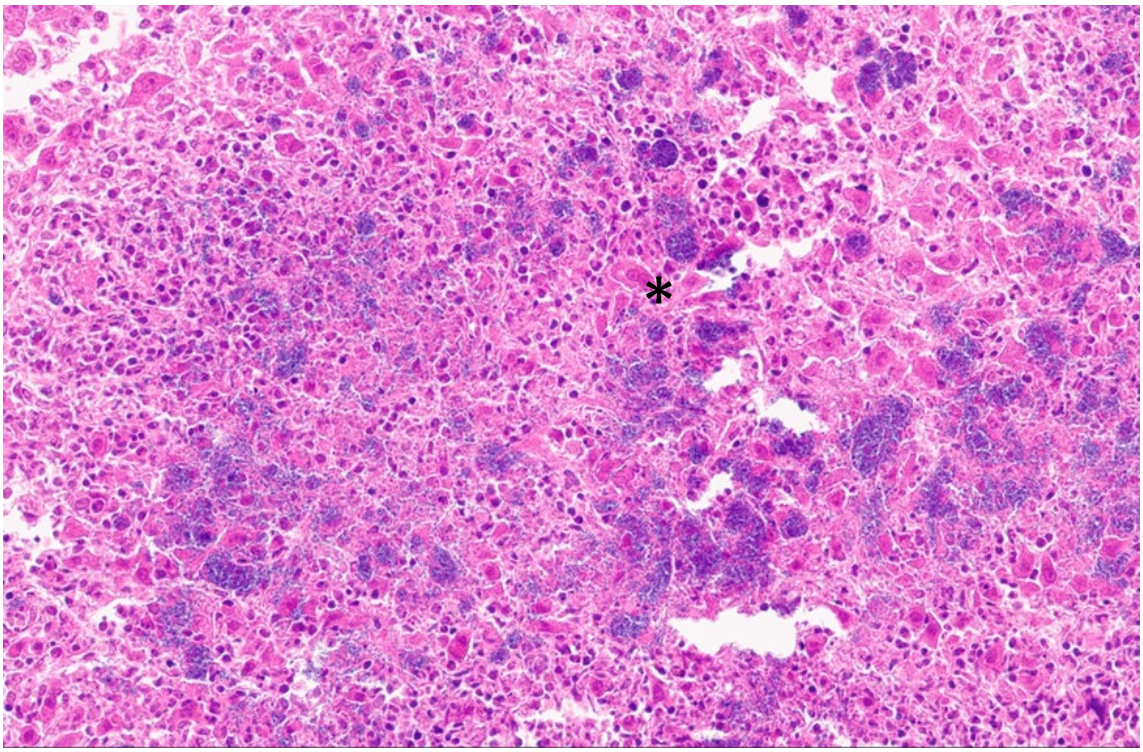

Figure 3. Goat fetal liver. Focus of necrosis with neutrophils and dense colonies of coccobacilli (*) amongst the necrotic debris. H&E stain, 40x.

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SWINE

AHL bacteriology swine testing update

Durda Slavic

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AHL Newsletter 2024;28(2):16.

On May 1, 2024, the AHL bacteriology section added two new tests for our swine clients.

1. Bacterial culture, fecal, porcine, **'cultsfe'**, $32.25 per sample, includes culture of fecal samples for *Escherichia coli*, *Salmonella* spp. and *Clostridium perfringens*. Susceptibility testing is included for *Salmonella* spp. only. The 'bsetup' charge of $25.00 will be added once to each case. This test code can be used for enteric cases in pigs of any age group.

2. Porcine bacteriology enteric panel 1, **'pentpa1'**, $65.25 per sample, includes culture of fecal samples for *E. coli*, *Salmonella* spp., *C. perfringens*, and ETEC (enterotoxigenic *E. coli*) PCR for two *E. coli* isolates per sample. Susceptibility testing will be done for confirmed ETEC and *Salmonella* spp. isolates only. The 'bsetup' charge, $25.00 will be added once to each case. This test is recommended for enteric cases in pigs up to and including 14 weeks of age.

   Note: The F4 (K88) and F5 (K99) slide agglutination test is being replaced by ETEC PCR because the production of F4/F5 antibodies has been discontinued. For more information, please see AHL Newsletter article: https://www.uoguelph.ca/ahl/make-change-ecoli-genotyping

Avian metapneumovirus subtype B: First cases confirmed in Canada

Emily Martin

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2024;28(2):17.

In recent months, multiple states throughout the US have documented increased cases of avian metapneumovirus infection (aMPV) with subtypes A and B. Although subtype C has been identified in wild birds previously, we have recently identified subtype B in commercial poultry flocks in Canada. Therefore, the poultry industry needs to be aware of this disease and the associated clinical presentations.

Avian metapneumovirus is in the family *Pneumoviridae*, genus *Metapneumovirus*. It is an enveloped, single-stranded negative-sense RNA, and has 4 identified antigenic subtypes (A to D). Subtypes A and B are identified in chickens and turkeys, whereas subtype C is identified primarily in turkeys as well as ducks. Other birds at risk include pheasants, game birds, and guinea fowl. Clinically healthy wild birds are considered a reservoir for this organism; e.g., waterfowl, sparrows, swallows, pigeons, and falcons. Wild birds and game birds have been found to be seropositive.

Avian metapneumovirus infection results in respiratory and reproductive disorders. High density poultry populations tend to have a higher incidence of disease. The organism is spread primarily through direct contact or fomites. The incubation period is 3-7 days, and the disease spreads rapidly within and between flocks. An entire flock can become clinically affected within a day. The birds shed virus for only a few days, and there is no latency or carrier state. However, there are species differences in the onset and development of lesions. Unfortunately, clinical signs and lesions are non-specific.

TURKEYS:
Clinical disease caused by aMPV in turkeys has been called turkey rhinotracheitis (TRT) and avian pneumovirus infection of turkeys (APV).
Regardless of age, turkey morbidity ranges from 40 to 100%, and mortality ranges from 0.4% to 50%. Clinical signs include snicking, rales, nasal discharge, foamy conjunctivitis, swollen infraorbital sinuses (**Fig. 1A**), submandibular edema, coughing, open mouth breathing, and head shaking. Severe disease can be identified in 3 to 12-week-old turkeys. In breeders, uterine prolapse can occur secondary to coughing. In layers, there can be up to 70% drop in egg production (range 10-40%), including increased occurrence of poor shell quality and peritonitis. Recovery can take up to 3 weeks.

CHICKENS:
Clinical disease caused by aMPV in chickens, guinea fowl and pheasants has been called swollen head syndrome (SHS).
The disease is not as well defined in chickens, and can be subclinical. Less than 4% of the flock may be affected. Mortality is rarely >2%. Egg production in broiler breeders and egg quality in egg layers are affected. Clinical signs can include swelling of the periorbital and infraorbital sinuses, torticollis, disorientation, and opisthotonus (**Fig. 1B**).

DUCKS:

Clinical signs can include respiratory symptoms (subtype C), decreased egg production (40-85%), and poor shell quality (i.e., soft, thin shelled, cracked).

There is variable pathogenicity between strains. Uncomplicated aMPV infection may have mild clinical signs, and can clear in 10-14 days. However, secondary infection(s) can increase the severity of disease. The virus affects the function of the cilia of the respiratory and reproductive epithelial cells, increasing susceptibility to secondary infections. Secondary infections include bacteria such as *E. coli*, ORT, *Pasteurella* spp., *B. avium*, *R. anatipestifer*, and *Mycoplasma* (MG), in addition to aspergillosis and viral infections (i.e., IBV), resulting in potential development of airsacculitis and pneumonia.

On postmortem examination the follow lesions can be observed:
Turkeys: Mucoid exudates in turbinates and trachea. Catarrhal inflammation of the upper respiratory tract such as rhinitis, laryngitis, and tracheitis. Various reproductive tract abnormalities in mature birds, e.g., egg peritonitis, folded shell membranes, misshapen eggs. Secondary infections can result in lesions of airsacculitis, pericarditis, pneumonia, perihepatitis, and in severe cases, subcutaneous exudate and osteomyelitis in cranial bones (**Fig. 1C**).
Chickens: Severe edema of the subcutaneous tissues of the head, neck and wattles. Variable swelling of the infraorbital sinuses.

When deciding on diagnostic testing, it is important to realize that this virus does not persist within affected birds. The virus is cleared quickly, and may only be detectable for 6-7 days post infection; therefore, by the time clinical signs are recognized, virus may be undetectable by PCR testing. Combining PCR and ELISA testing will aid in diagnosing and tracking disease within and between flocks. Antibody titres may be detectable 7 days post infection. Recommended samples include nasal secretions and sinus or tracheal swabs of mildly affected birds to test for aMPV and secondary infections. Histopathology requires collection of fresh tissues to attempt to detect intracytoplasmic inclusion bodies in upper respiratory ciliated epithelial cells, otherwise lesions are not considered pathognomonic.

There is no treatment for uncomplicated aMPV infection. Suggested interventions include general recommendations for disease management including biosecurity (preventing exposure to wild birds or other infected poultry), disinfection, and dedicated barn clothing. Good barn management involves providing optimal ventilation, controlling temperature, preventing overcrowding, maintaining litter quality, and avoiding multiage facilities.

Since this is an enveloped virus, it is sensitive to multiple disinfectants, including quaternary ammonia products and bleach. It is stable at pH 3.0-9.0, and is inactivated at 56 °C for 30 minutes. However, it has longer survival times (i.e., weeks) at lower temperatures, and that could explain some seasonal occurrences.

AHL has recently validated PCR testing that includes detection of multiple subtypes of aMPV, including A, B, and C. The ELISA available at the AHL can also detect antibodies against multiple subtypes of aMPV. If you have questions about this disease or diagnostic testing, we recommend veterinarians contact the AHL. *AHL*

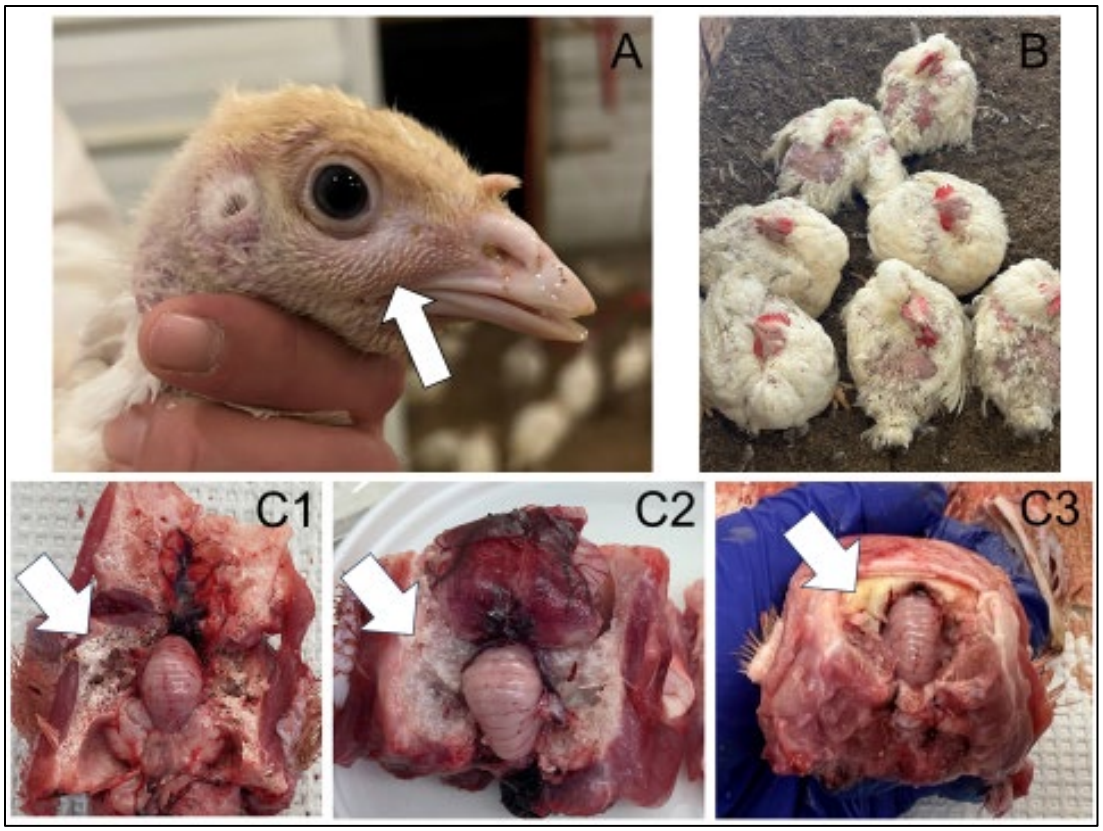

Figure 1. Lesions caused by aMPV infection. **A:** Swollen infraorbital sinuses in a turkey (Photo: Select Genetics). **B:** Chickens displaying torticollis and opisthotonus (Photo: US poultry integrator). **C1:** Normal turkey cranial bones. **C2.** Cranial bones with opaque fluid. **C3.** Cranial bones with osteomyelitis (Photos: Dr. Jenny Nicholds and Dr. Jason Sousa, PDRC, Georgia).

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Poultry environmental sample testing update

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AHL Newsletter 2024;28(2):19.

In order to provide faster turn-around-time (TAT) for screening of poultry environmental samples for the presence of *Salmonella* spp., the AHL bacteriology section switched to a new test on May 1, 2024. This new test involves real-time PCR (qPCR) targeting two *Salmonella* spp. specific genes. An internal control (IC) is also included to detect any potential PCR inhibitors. This screening method is used for all samples previously submitted for testing under 'hsfe' and 'hsfeb' testing codes, as well as fluff samples

for which counts and culture are required. These 2 test codes are replaced with one **'hsfepcr'** test code.

Once received, samples are incubated in buffered peptone water (BPW) overnight, and the screening qPCR test is done the following day (excluding weekends and statutory holidays), providing 24 hr TAT. Results are reported as either '**negative'** or '**culture to follow'**. All '**culture to follow'** samples undergo routine bacterial culture followed by *Salmonella* grouping and serotyping, if applicable.

This screening test was validated using 1630 environmental samples and the summary of test performance is shown in Table 1. There is almost perfect (0.886 to 0.959) to excellent agreement (0.846) between culture and PCR results.

Table 1. Diagnostic sensitivity, specificity, agreement between tests, Kappa value, positive predictive value (PPV) and negative predictive value (NPV) shown for the overall PCR results as well as for individual sample types.

	Overall	**Booties**	**Fluff**	**Environmental swabs**	**Peptone**
Sensitivity	95.0 %	95.0 %	96.2 %	100 %	87.5 %
Specificity	99.5 %	99.6 %	99.1 %	99.4 %	98.7 %
Agreement	98.9 %	98.7 %	98.8 %	99.4 %	98.0 %
Kappa	0.949	0.959	0.937	0.886	0.846
PPV	98.5 %	98.5 %	96.2 %	80.0 %	84.0 %
NPV	99.2 %	98.7 %	99.6 %	100 %	99.1 %

Modified results for fowl adenovirus PCR at AHL: Guidance for interpretation

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AHL Newsletter 2024;28(2):20.

Recently, the reporting of fowl adenovirus (FAdV) PCR at the AHL has changed. Previously, the reporting included results for FAdVE, FAdVD and FAdVAC. However, now the results are reported as FAdVE, FAdVD and FAdVABCDE. The reason for this change is that FAdVE and FAdVD are the most common species identified in Ontario. By changing the reporting, the results clearly indicate if there is any other group of fowl adenovirus detected other than FAdVE and FAdVD.

Interpretation is as follows:

- If FAdVE and/or FAdVD are positive, FAdVABCDE should also be positive.
- If there are high Ct (Cycle threshold) values, then there will be the odd circumstance where FAdVABCDE will be negative when FAdVE and/or FAdVD is positive.
- If FAdVE and FAdVD are negative and FAdVABCDE is positive, then a different "species" of FAdV is detected.

If you have any questions on interpretation of results, please do not hesitate to call the lab and discuss.
AHL

HORSES

Alcohol and Gaming Commission of Ontario (AGCO) Death Registry / Equine Incidences in Ontario Racing program: 2023 Postmortem summary

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AHL Newsletter 2024;28(2):21.

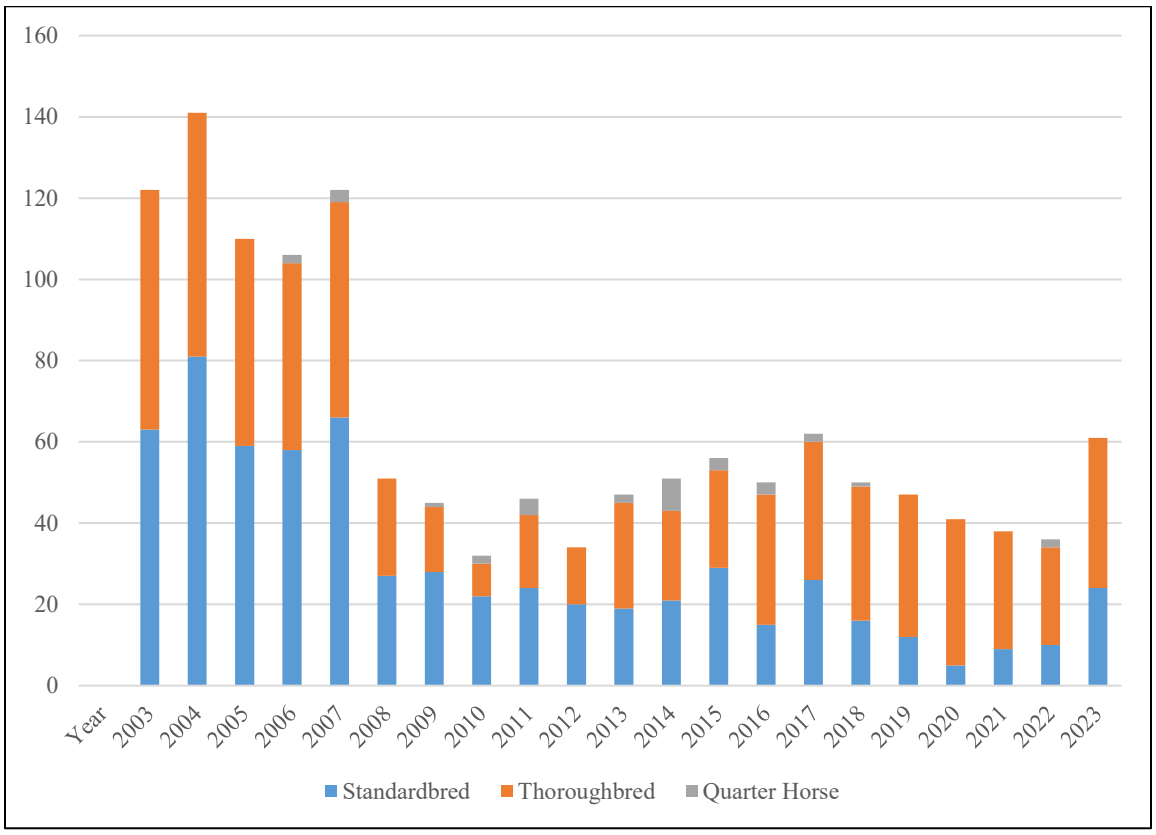
The Alcohol and Gaming Commission of Ontario (AGCO; formerly the Ontario Racing Commission, ORC) continues in its **proactive approach to advance racehorse welfare and safety of human and animal participants**. In 2003, Ontario became one of the first North American racing jurisdictions to require mandatory reporting of racehorse deaths, in order to monitor, research and improve knowledge of why these events occur. Postmortem (PM) exams conducted at the Animal Health Laboratory (AHL) through the AGCO Death Registry (DR, 2003-2016) and Equine Incidences in Ontario Racing (EIOR, 2016-current) programs continue to provide comprehensive data regarding the causes of morbidity and mortality in racehorses in this province. The 2023 racing season marks the 21st year of the PM program. To date, PMs have been carried out on 1,348 horses, including 634 (47%) Standardbreds, 681 (51%) Thoroughbreds, and 33 (2%) Quarter Horses (**Table 1**). Annual variation in the number of PM cases reflects the discretionary requirement for PM of reported deaths on the part of the Registrar of AGCO.

A summary of diagnoses by body system for 2023 AGCO PM cases is provided (**Fig. 1**).

Since 2015, **computed tomography (CT) of fractured and contralateral limbs** has been carried out on select AGCO postmortem cases through collaboration with the Diagnostic Imaging section of the Ontario Veterinary College Health Sciences Center. The goal of this in-depth examination is to identify pre-existent lesions, primarily in bone, that contribute to catastrophic fractures. In 2023, CT imaging was performed on 28 limb fracture cases submitted for PM. **Pre-existent lesions in bone were identified by CT and considered potentially predisposing to fracture in 14/28 (50%) cases.**

Exercise-associated sudden death continues to be of concern to the racing industry. At the AHL, an in-depth PM protocol is used in the evaluation of these cases, with special emphasis on cardiovascular and respiratory systems. In 2023, the cause of death (COD) was investigated in 16/61 (26%) horses that died during or shortly after exercising. Death was attributed to multiple causes including significant pulmonary hemorrhage compatible with the syndrome of equine exercise-associated fatal pulmonary hemorrhage (EAFPH - 7 horses); acute hemorrhage due to various causes (thoracic aortic rupture -1 horse; rib fracture with hemothorax -1 horse; severe hemothorax with source unidentified – 1 horse); and skull fracture (1 horse) (**Fig. 2**). The COD was undetermined in 5/16 (31%) exercise-associated sudden death cases in 2023. Over the duration of the postmortem program (2003-2023), the COD was undetermined in 53/218 (24%) sudden death cases. It has been speculated that **exercise-associated cardiac dysrhythmia**, leading to acute heart failure and pulmonary hypertension, may be the underlying cause of death among many of these horses, and may also contribute to pulmonary hemorrhage. Typically, no morphologic lesions are detected in the heart as a cause or result of fatal ventricular dysrhythmia, and the diagnosis cannot be confirmed based on PM findings. *AHL*

Table 1. Breed distribution of AGCO postmortem submissions to the AHL, 2003-2023.



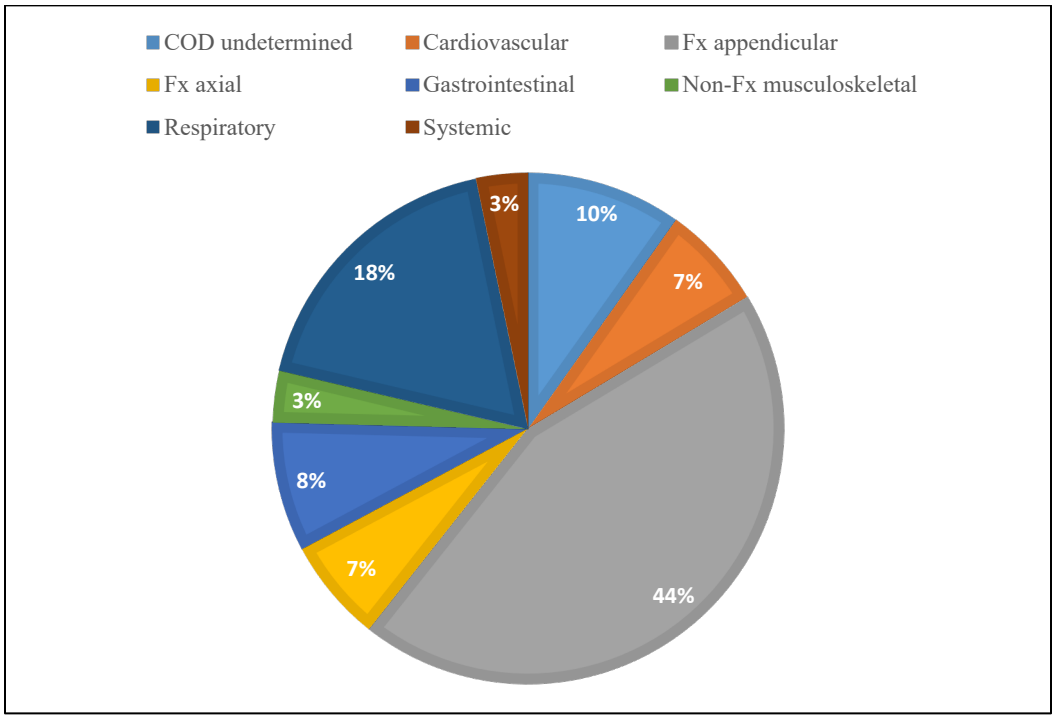


Figure 1. Diagnoses by body system for AGCO postmortem submissions to the AHL, 2023.

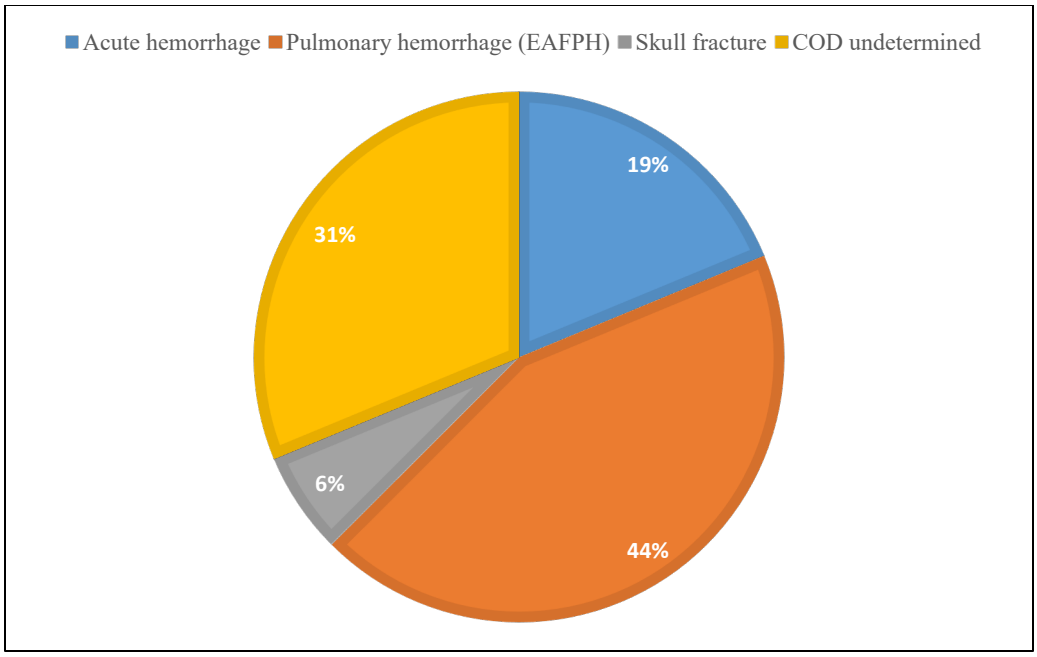


Figure 2. Diagnoses by body system for AGCO exercise-associated sudden death cases submitted to the AHL, 2023.

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Testing for strangles (*Streptococcus equi)*

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Strangles, caused by *Streptococcus equi* subspecies *equi*, can be detected by PCR or bacterial culture at the AHL.

The PCR test is highly sensitive, can detect small numbers of bacteria, and is useful for detecting carrier horses. However, the PCR cannot differentiate between bacteria that are alive or are dead/fragmented, and therefore, a positive PCR result may not indicate an active infection.

A bacterial culture is less sensitive and has a longer turnaround time. It can miss horses that are in the early stage of infection, or those carrying a low bacterial load. A culture is also required for antimicrobial sensitivity testing.

It is important to understand that no single test will detect all positive animals. Sensitivity of testing for strangles can be increased by:

- sampling by guttural pouch lavage and/or nasopharyngeal lavage;
- sampling 48 hours or more after the onset of fever;
- testing at regular intervals if you suspect a horse has been exposed or infected;
- submitting samples from as many animals as possible in an outbreak situation;
- using an endoscope to assist with collection of the sample and visualization of the affected passages.

Bacterial culture detects viable *S. equi*, while PCR will detect DNA from viable or non-viable bacteria. A recent study by Weese et al found that a qPCR Ct of 34.2 was a reasonable breakpoint for the likelihood of the presence of culturable *S. equi*.

PCR sequence typing will differentiate between vaccine and field strains, and this test is available at the AHL for a strong positive qPCR result (Ct < 30) and pure bacterial culture results.

If you obtain a *Strep. equi* test result that is not expected based upon clinical findings and history, please contact the AHL to review the result and discuss follow-up options. *AHL*

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Guttural pouch mycosis in a Quarter Horse filly

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AHL Newsletter 2024;28(2):24.

Early in April, a Quarter Horse filly was presented for pyrexia, fever, purulent nasal discharge, right-sided swelling of the head, and a cough of 1 week duration. At that time, the right ear and upper eyelid had a notable droop. Testing of the nasal discharge was both culture and PCR positive for *Streptococcus equi* subsp. *zooepidemicus*. Treatment was initiated, and the pyrexia and lethargy resolved within 5 days. As part of follow up, an endoscopic exam of the guttural pouches was performed, during which large plaques of purulent debris were visualized in the right guttural pouch near the opening. As there was minimal improvement with treatment, referral was initiated. However, prior to transfer, there was acute severe hemorrhage from the nostrils and mouth, resulting in sudden death of the filly.

At necropsy, there was marked hemorrhage present within the nasal passages and guttural pouches bilaterally, as well as within the tracheal lumen and the stomach. The guttural pouches were dissected, and an extensive tan-yellow plaque was present, effacing approximately 80-85% of the mucosal surface of the right guttural pouch (**Fig. 1**). The plaque both effaced and obscured the right internal carotid artery, right external carotid artery and the cranial nerves in the right guttural pouch (hypoglossal, glossopharyngeal, vagus and accessory nerves). Necrosis was extensive, particularly within the right guttural pouch, but also extended through the septum with a plaque visible within the left guttural pouch on its medial aspect. On sectioning into tissue beneath the plaques, areas of dark red to black

discoloration extended into the underlying muscle, connective tissue and the right stylohyoid bone subdividing the lateral and medial portions of the right guttural pouch.

Histologically, hemorrhage frequently dissected necrotic areas. In a section taken from the septum between the right and left guttural pouches, there was full thickness necrosis of the tissues underlying the plaques. The plaques were composed of necrotic cellular debris, a mixed inflammatory cell infiltrate, and dense colonies of coccobacilli (**Fig. 2A**). These areas were overlaid by thick mats of fungal hyphae that appeared histologically consistent with *Aspergillus* spp. (**Fig. 2B**).

The postmortem findings confirmed a diagnosis of guttural pouch necrosis with severe hemorrhage as the cause of death in this horse. Based on the presence of the large necrotic areas of mucosa combined with dense plaques of friable, dark yellow-green material, a fungal cause was suspected grossly and was further confirmed histologically. Guttural pouch mycosis is a relatively rare disease of the upper respiratory tract in horses. Although there is no apparent predisposition to development of guttural pouch mycosis, it is most commonly reported during the warmer months in stabled horses in temperate climates.

While guttural pouch mycosis is usually unilateral, extension from one guttural pouch to another is possible, as occurred in this case. The underlying cause is uncertain, although it is suspected that the fungal infection occurs opportunistically following damage that occurs due to trauma, local inflammation and/or a primary bacterial infection. Following injury, damage to the mucosal barrier results in an environment hospitable to colonization by a secondary fungal pathogen, resulting in invasion into deeper tissues, including the underlying vasculature and nerves. A predilection towards forming on the roof of the medial compartment of the guttural pouch in close anatomic association with the carotid arteries and various cranial nerves explains the various reported clinical signs that include: facial paralysis, Horner's syndrome, sweating, shivering, nasal discharge, dysphagia, abnormal swelling of the head and epistaxis. Invasion of the carotid arteries by fungal hyphae can result in fatal hemorrhage.

In this case, it is suspected that an initial bacterial infection (i.e., *S. equi* subsp. *zooepidemicus*) may have resulted in localized trauma and secondary fungal colonization. Unfortunately, this pathogenesis is challenging to confirm as chronic changes will often obscure the initial inciting causes. As there are multiple potential causes of nasal discharge in horses, including *Streptococcus equi* subsp. *equi* (which is now an immediately notifiable disease in Ontario), this case highlights the importance of using multiple cooperative methods of investigation such as endoscopy, bacterial culture, PCR, and histology when working up a case. *AHL*

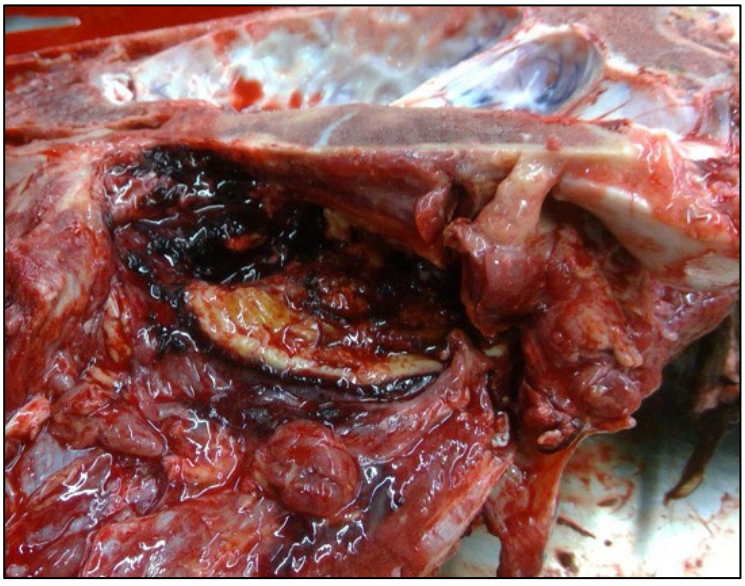

Figure 1. Opened guttural pouch, equine. The guttural pouch contains clotted blood and a friable tan-yellow plaque overlies the mucosa.

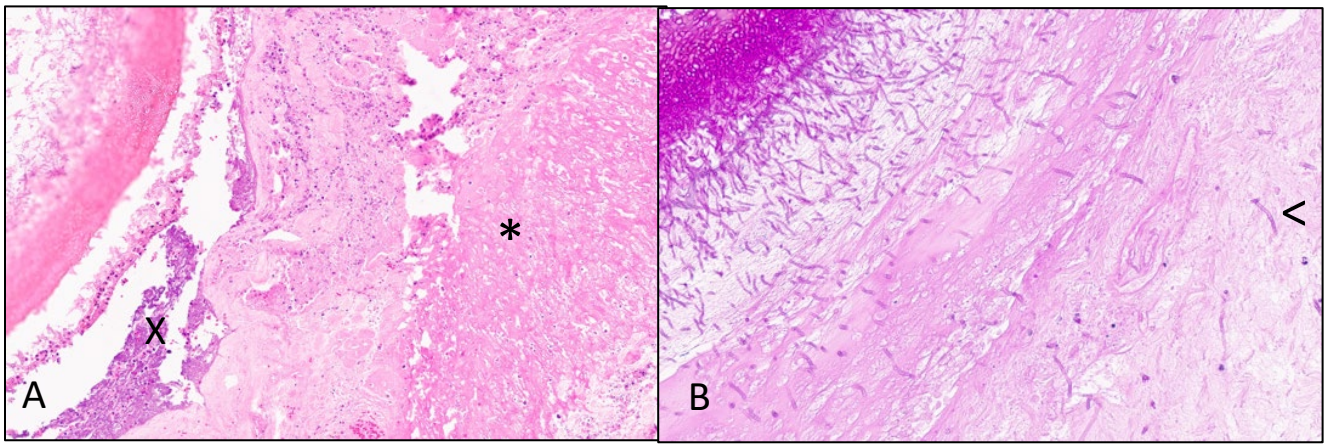

Figure 2. Histologic sections of affected guttural pouch. A. Area of necrosis underlying fungal mat (*), with colonies of bacteria (X). (H&E stain, 20X). B. Area of necrosis overlaid by PAS-staining fungal mat with histologic features suggestive of *Aspergillus* spp. There is some invasion of the underlying necrotic tissue by fungi (<). (PAS stain, 40x).

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COMPANION ANIMALS

AHL testing update for companion animals and other (exotic) animal species

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AHL Newsletter 2024;28(2):27.

Starting May 1, 2024, AHL changed how antimicrobial susceptibility testing is charged for tests involving companion animals (dogs, cats, horses) and other (exotic) animal species. Only a single susceptibility test will be included in the price of culture. Any additional susceptibility tests requested by the client will be performed at an extra charge. **It is recommended that additional susceptibilities be requested by specifying number on the submission form (e.g., "report up to 3 significant bacterial sensitivities") when submitting a sample for testing, as AHL does not store bacterial isolates for more than 3 days after results are reported to clients.** There will be two options to select for susceptibility testing: disk diffusion (DD) and minimal inhibitory concentration (MIC). Please refer to the following tests when ordering culture and susceptibility testing:

1. Bacterial culture, aerobic, **disk diffusion**, 'cultn', **$56.00** per sample and **$25.00** set-up charge per case, includes culture for aerobic bacteria. Disk diffusion susceptibility testing for only ONE significant pathogen per case is included. Additional susceptibilities must be requested and extra charges, **$15.00** per isolate, will apply.

2. Bacterial culture, aerobic and anaerobic, **disk diffusion**, 'ancultn', **$152.00** per sample, includes culture for aerobic and anaerobic bacteria. Disk diffusion testing for only ONE significant aerobic pathogen per case is included. Additional susceptibilities must be requested and extra charges, **$15.00** per isolate, will apply. Susceptibility testing is not routinely performed on anaerobes. If susceptibility testing for anaerobes is required, please email the lab at ahlbact@uoguelph.ca for more information.

3. Bacterial culture, aerobic, **minimal inhibitory concentration**, 'cultnm', **$65.00** per sample and **$25.00** set-up charge per case, includes culture for aerobic bacteria. Minimal inhibition concentration susceptibility testing for only ONE significant pathogen per case is included. Additional susceptibilities must be requested and extra charges, **$42.50** per isolate, will apply.

4. Bacterial culture, aerobic and anaerobic, **minimal inhibitory concentration**, 'anculnm', **$163.80** per sample, includes culture for aerobic and anaerobic bacteria. Minimal inhibition concentration susceptibility testing for only ONE significant aerobic pathogen per case is included. Additional susceptibilities for aerobic bacteria must be requested and extra charges, **$42.50** per isolate, will apply. Susceptibility testing is not routinely performed on anaerobes. If susceptibility testing for anaerobes is required, please email the lab at ahlbact@uoguelph.ca for more information.

AHL is strongly encouraging use of MIC susceptibility testing, as more interpretative Clinical Laboratory Standard Institute (CLSI) guidelines for animal species/bacteria/drug combinations are available in comparison to disk diffusion, and this trend will likely continue in the future. *AHL*

Systemic herpesvirus infection in a puppy

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AHL Newsletter 2024;28(2):28.

An English Bulldog breeder sought the services of a veterinarian after eight puppies from three different litters had died suddenly over an unspecified period of time. A four-week-old male puppy had stopped eating several days prior to death and was treated with acyclovir and clavamox due to a history of herpesvirus infection in the kennel. The puppy died despite treatment and was submitted to the Animal Health Laboratory for postmortem examination.

On gross examination, the puppy was in good body condition and had no significant external findings. On internal examination, innumerable 1-2 mm diameter flat red foci were identified in the renal cortices, which corresponded to segmental streaks of hemorrhage extending from cortex to medulla. Similar pinpoint to1 mm diameter foci were randomly distributed throughout the hepatic parenchyma and the spleen (**Fig. 1**). The lungs were diffusely dark pink and firm.

Histologically, these foci in the kidneys, liver and spleen corresponded to foci of acute necrosis and hemorrhage (**Figs. 2, 3**). In the lung, there were similar foci of parenchymal necrosis with more widespread interstitial pneumonia. Very rarely, intranuclear eosinophilic inclusion bodies typical of herpesvirus were identified in areas of necrosis. Kidney was submitted for canid herpesvirus-1 PCR and was positive with a cycle threshold of 20.29.

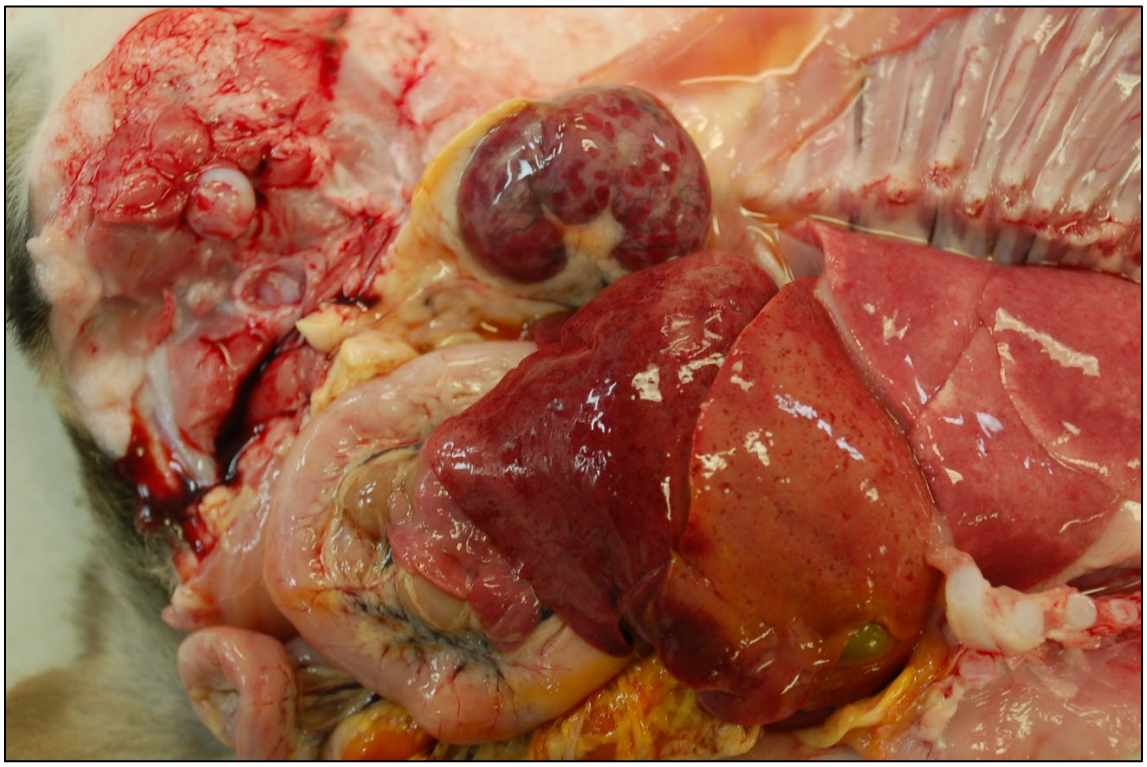

Figure 1. Opened abdominal and thoracic cavities. The kidney and liver demonstrate multifocal to coalescing areas of necrosis and hemorrhage, characteristic of systemic canid herpesvirus-1 infection.

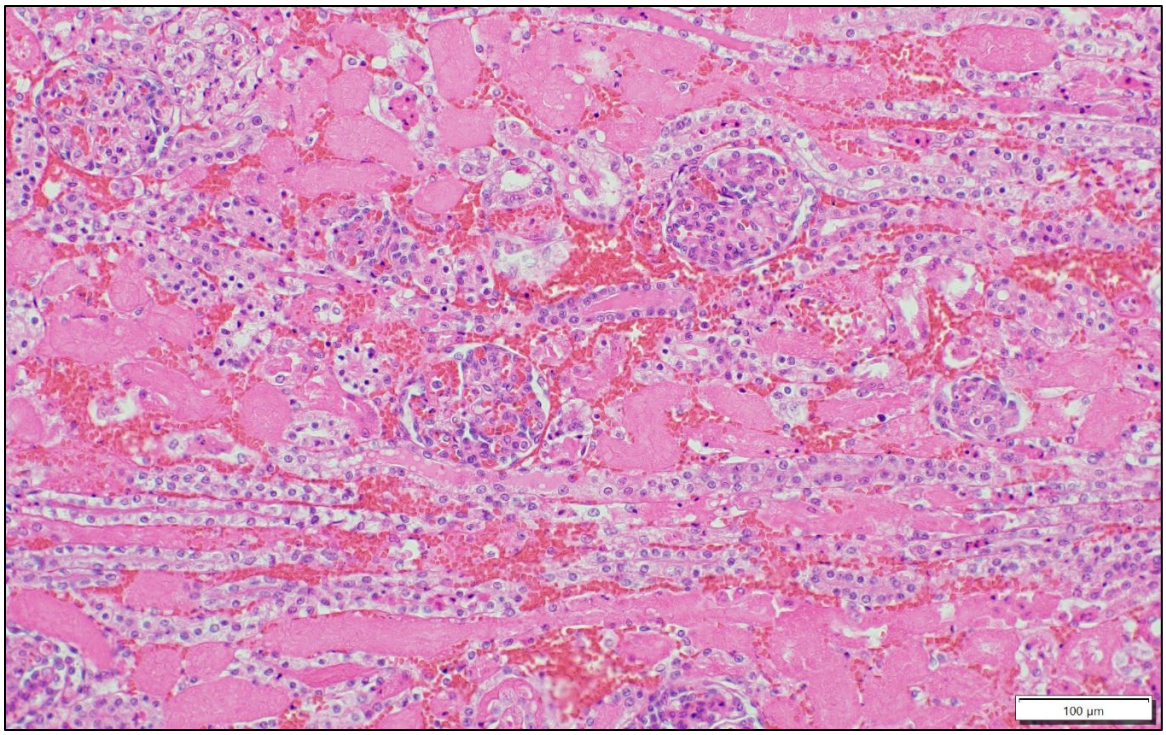

Figure 2. Kidney. Acute tubular necrosis with interstitial hemorrhage. H&E, 20X.

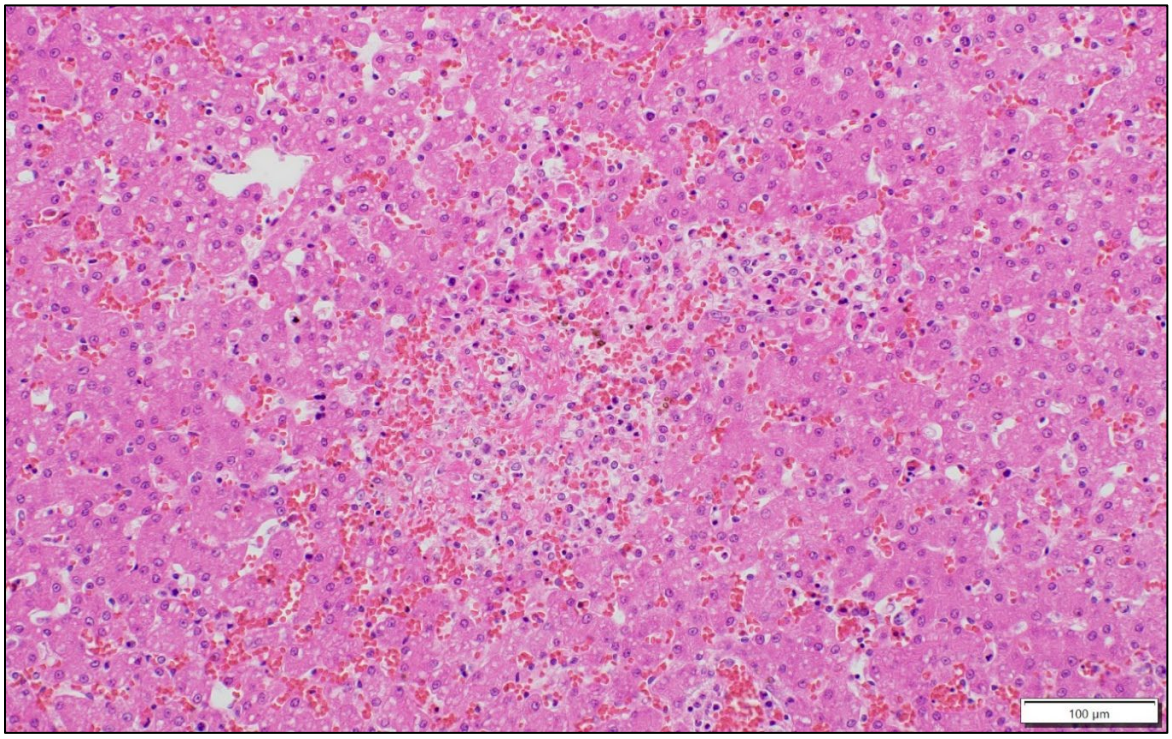

Figure 3. Liver. Acute hepatocellular necrosis and hemorrhage. H&E, 20X.

The combination of gross and histologic lesions is considered essentially pathognomonic for systemic canine herpesvirus infection in puppies. This fatal condition tends to occur in puppies less than four weeks of age, as younger animals have difficulty with thermoregulation and herpesvirus requires a cooler body temperature in order to replicate. Litter mortality rates are high, and clinical signs in affected

puppies can include respiratory distress, nasal discharge, anorexia, vomiting, increased vocalization, seizures, and sudden death. Dams of infected litters may not show clinical signs or may have non-specific upper respiratory signs. Neonates can be infected in utero, during birth from maternal genital lesions, or from oronasal secretions of the dam. Similar to all herpesviral infections, the infection is life long, and periods of recrudescence in infected animals often coincide with periods of stress, such as pregnancy, or immunosuppression. *AHL*

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Gallbladder hematoma presenting as a mucocele in a dog

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AHL Newsletter 2024;28(2):30.

The gallbladder and a liver biopsy from a 6-year-old male neutered Brussels Griffin dog were submitted to the Animal Health Laboratory following a history of vomiting. On ultrasound examination, abundant hyperdense material had been noted in the gallbladder, which raised concern for gallbladder mucocele. A cholecystectomy was performed. The submitted gallbladder was enlarged and the capsule over the surface appeared intact. On the cut section, the tissue was divided into two structures with a large, round structure filled with dark red blood compressing the gallbladder into a thin crescent-shaped rim around one side (**Fig. 1**). This appearance was confirmed histologically where the tissue formed two "lumina" divided by a discontinuous fibrous septa but surrounded by a continuous fibrous capsule. The first lumen was lined by gallbladder mucosa with occasional ectatic glandular structures, and was filled with abundant bile mixed with a small amount of hemorrhage. Along one side, the gallbladder wall underwent transmural necrosis with marked attenuation of the epithelium which gave way to complete loss of the wall (**Fig. 2**). This focal area of disruption was surrounded by hemorrhage and abundant fibrin. The rupture communicated with an enclosed space contained within the same fibrous capsule which was unlined by any epithelium, and contained abundant hemorrhage and clumps of fibrin with rare neutrophils. There was a small amount of liver parenchymal tissue along the far border of the fibrous wall, at the edge of the section opposite to the viable gallbladder tissue.

This lesion was diagnosed as a mural hematoma of the gallbladder. There were also changes consistent with early mucocele formation in the compressed gallbladder. The granulation tissue surrounding the hematoma indicated a degree of chronicity, and may explain why the hematoma was not evident externally during surgery or trimming, as it was well-contained by fibrous tissue which blended with the gallbladder capsule.

Gallbladder hematomas are rarely reported in humans, and a single case report of spontaneous gallbladder mural hematoma in a dog was found in the literature. In that case as well as in ours, the clinical presentation was indistinguishable from a gallbladder mucocele. Possible causes of gallbladder hematomas identified in human cases include blunt force trauma to the abdomen, underlying coagulopathy, cholelithiasis, and gallbladder neoplasia. In this case and the other reported canine case, no cause was identified. A swab from the tissue was submitted for bacterial culture and no bacteria were isolated. Surgical removal was uncomplicated, and the dog recovered well from surgery. *AHL*

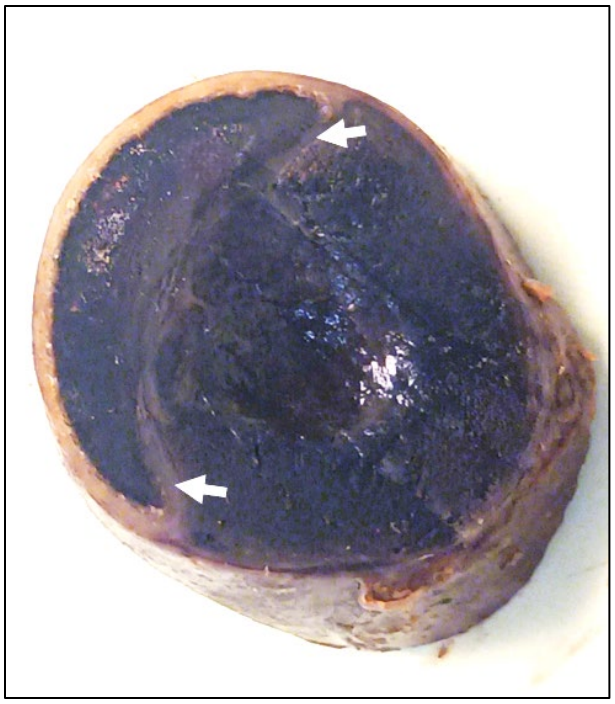

Figure 1. Cross section of the gallbladder at the time of trimming. The compressed gallbladder lumen is forming a crescent on the left side of the image, and the hematoma forms the majority of the tissue on the right. The white arrows indicate the fibrous septa separating these structures.

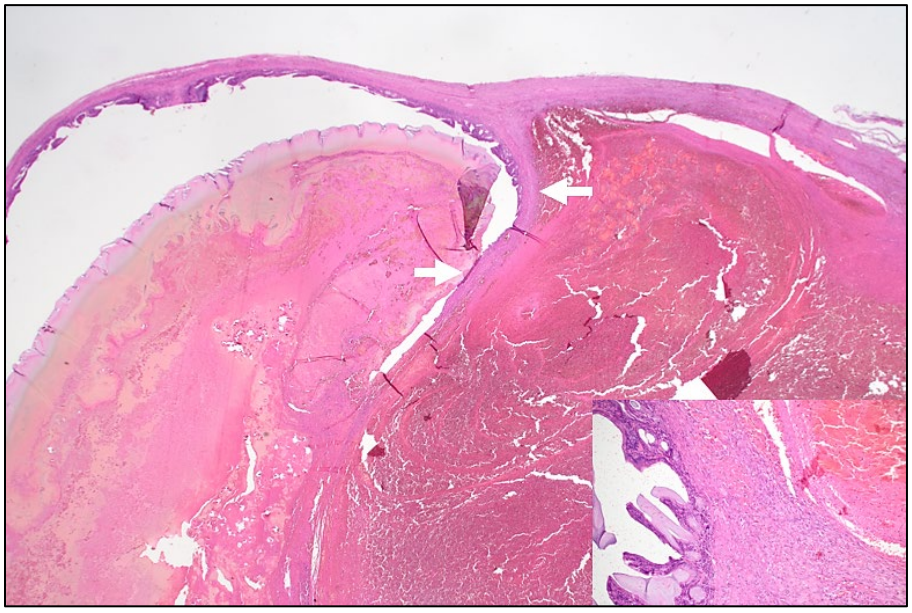

Figure 2. Histologic cross section of the gallbladder with the same orientation as figure 1. The hematoma is on the right with abundant blood in the lumen. The gallbladder forms a compressed, crescent structure on the left, with an incomplete fibrous septa between them (indicated by the arrows). The inset shows the gallbladder mucosa on the left and the unlined fibrous wall of the hematoma on the right. H&E

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Presumptive monensin toxicosis in a 6-month-old dog

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A 6-month-old intact male bloodhound presented to the Ontario Veterinary College (OVC) with a 2-3 day history of progressive ataxia, hind limb weakness, lethargy and red-brown discolored urine.

The CBC showed an inflammatory leukogram, as indicated by mild neutrophilic leukocytosis with a regenerative left shift and evidence of neutrophil toxicity, consisting of cytoplasmic basophilia and identification of Döhle bodies (**Table 1**). The biochemistry profile showed a markedly elevated activity of the enzymes creatine kinase (CK) and alanine-amino transferase (ALT), as well as a moderate hyperphosphatemia (**Table 1**). These findings were consistent with severe myopathy. Although ALT is relatively "liver specific" in dogs, and elevated activity in this case might have resulted from concomitant liver damage, the enzyme is also present in striated skeletal and cardiac muscle, and the elevated serum activity could have also resulted from severe muscle damage.

Table 1. CBC and biochemistry significant abnormalities.

	Neutrophils $x10^9$/L (2.9 - 10.6)*	Bands $x10^9$/L (0.0 - 0.3)*	CK U/L (40 - 255)*	ALT U/L (19 - 107)*	Phosphorous mmol/L (0.90 - 1.85)*
Day 01	13.5	0.31	736,520	2,103	3.03
Day 03			603,900	3,284	2.26

* reference interval

Urine was collected via cystocentesis. The urinalysis revealed a concentrated red urine that was highly positive for "protein" and "blood" on the reactive strip, with only rare erythrocytes noted on the sediment (**Table 2**). These findings ruled out hematuria. Since the animal was not anemic and there were no signs of hemolysis, the red discoloration of the urine and positive result for "blood" were interpreted as myoglobinuria, consistent with other laboratory findings supporting myopathy (rhabdomyolysis).

Table 2. Urinalysis

	Colour	SpG	Multistix protein	Blood	RBCs/hpf
Day 01	Red	1.040	3(+)	3(+)	0-2

Supportive care was provided, including sedation, anxiolytics and IV fluids. The patient improved clinically, did not develop signs of cardiac disease and was discharged after a few days in the hospital. This dog lived on a farm and was seen consuming calf feed medicated with monensin. Based on this observation, clinical presentation and laboratory findings, a clinical diagnosis of monensin toxicosis was made.

Monensin is an ionophore antibiotic used as a feed additive in approved livestock species (poultry and cattle) due to anti-coccidial properties and enhancement of feed efficiency (Canada); and to reduce the incidence of ketosis in peri-parturient dairy cattle (Europe). However, incorrect dosing or exposure of non-target species (e.g. dogs and horses) may result in toxicosis and mortality. Although cases of monensin toxicosis in dogs are relatively uncommon, it is important to be aware of its potential toxicity and to take the necessary precautions to minimize the risk of exposure. A few reports in the UK raised concerns for increased frequency of cases in dogs (1, 2), leading to updated warnings on the safety of these products and the suggestion of "guidelines for treatment of monensin toxicosis in dogs:" (3). *AHL*

References

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