Laboratory Services Division
Animal Health Laboratory

# AHL Newsletter

**AHL Newsletter, Volume 27, Number 2** **June 2023**

***In this issue:***



**AHL Newsletter**
June, 2023 - Volume 27, Number 2 ISSN 1481-7179

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

To receive an **electronic copy of this Newsletter**, please send your email address to us at holiver@uoguelph.ca

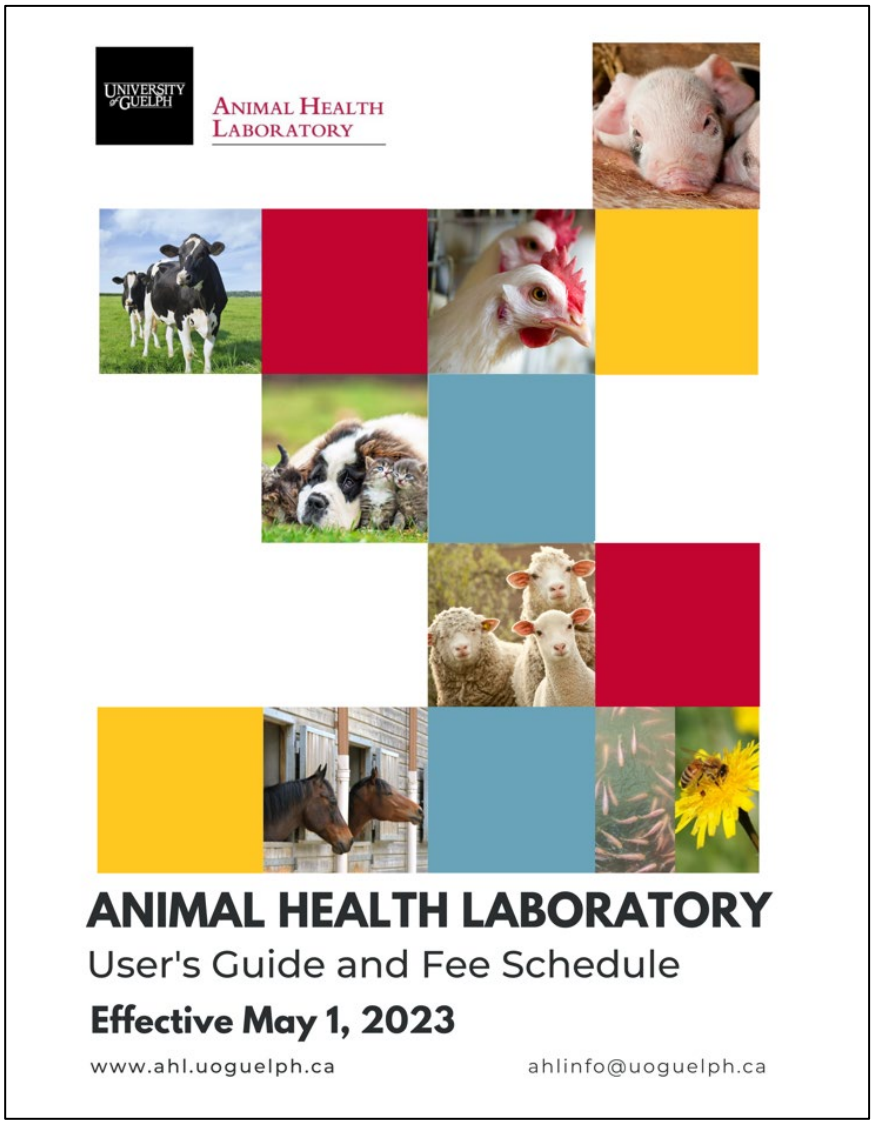
# AHL User's Guide and Fee Schedule - May 1, 2023

Includes 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 2022**:

*Chlamydia* spp. - PCR, non-food animals
Copper determination in biopsies - ICP-MS
IAPD (Ontario Interactive Animal Pathogen Dashboards) membership (Tableau viewer license charge)
Iodine in serum and urine - ICP-MS
*Mycoplasma bovis* in semen samples - PCR
*Parelaphostrongylus tenuis* (meningeal worm) - PCR and sequencing identification
Rabbit hemorrhagic disease virus-2 - PCR

# Update from the Director

*The view from the Director's office*

Our June newsletter typically notifies clients of our updated annual AHL User's Guide and Fee Schedule which is revised every year on May 1. This year, you may notice significant fee increases which are a result of economic pressures that all businesses are experiencing in this post-pandemic period. Supply and staffing costs have increased, requiring compensatory increases in some test fees. If you have any specific concerns or challenges related to test pricing, please reach out to us - we would be happy to review test options that might better suit your needs.

This May, we said farewell to our long-serving AHL Client Services Veterinarian, Dr. Jim Fairles, who has retired. You can view a photograph of Jim holding a commemorative cartoon with all of his favourite hobbies in our Staff highlights section. Included in this section is a photograph and biography of AHL's new Client Services Veterinarian, Dr. Tim Pasma. Congratulations Jim and welcome Tim!

As in many previous years, we seem to have progressed rapidly through the spring season into full-blown summer. I'm sure many of you are ready to finally experience a full complement of summer-time activities as we begin to view the pandemic in the rear-view mirror after three long years of restrictions. I hope that you are able to relax and enjoy the summer of 2023 with your families and friends. We all deserve it.

*Maria Spinato, Director*

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

# Specimen Reception update

*Tim Pasma*

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

**Laboratory Submissions and Premises Identification**

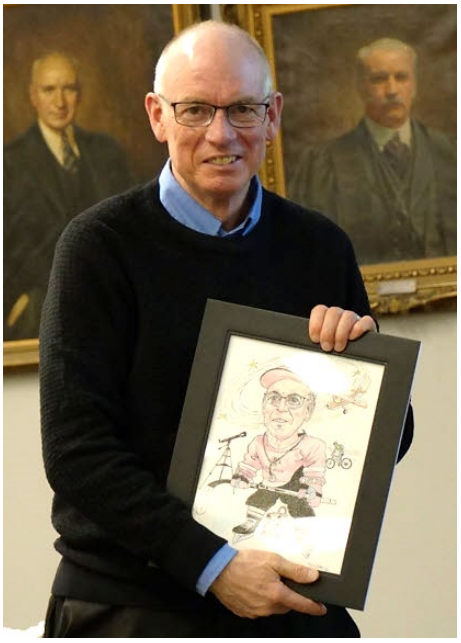
Premises identification (PID) is an important component of traceability. Along with animal identification and movement reporting, PIDs facilitate the traceability of animal and food products throughout the agri-food system. The Provincial Premises Registry Program, which runs the premises identification database in Ontario, began in July of 2011. The program is voluntary, but may be required in situations such as participating in a supply managed commodity or for OAHN projects.

PID is also a critical tool for emergency preparedness by helping personnel responding to a natural disaster or animal disease outbreak. PIDs assist with surveillance of disease as it facilitates the tracking of disease across Ontario, as done by OMAFRA for monitoring of swine influenza at the county level. It is also used in disease control programs such as the Swine Health Area Regional Control (SHARC) program.

The AHL strongly encourages including PIDs on all submissions. Over 80% of submissions from the swine sector contain PIDs, through the work of swine veterinarians and AHL to increase the number of swine submissions including PIDs. We encourage others to get involved in improved emergency management and disease surveillance by obtaining and using PIDs. Please contact the AHL if you wish to discuss PIDs and electronic submissions to the lab.

# Staff Highlights

## Congratulations to Dr. Jim Fairles on his retirement



Dr. Jim Fairles retired on April 30, 2023 after serving as AHL Client Services Veterinarian for 20 years. We owe him many thanks for his dedication to the veterinary community, and his exceptional service to AHL staff and clients. We wish him a long, happy and healthy retirement, full of satisfying hobbies (raising money for charitable organizations, training and participating in triathlons and flying his plane over the Ontario countryside), and many special occasions with family and friends. Congratulations Jim!

## Welcome Dr. Tim Pasma, AHL's new Client Services Veterinarian

Dr. Tim Pasma has joined AHL as our new Client Services Veterinarian. Tim graduated with his DVM from Guelph, and worked as an Associate Veterinarian at a mixed animal practice in Ontario before moving to Manitoba where he worked in a large swine practice. He then worked for the Manitoba CVO's office as a Disease Control and Animal Welfare Veterinarian. During this time, he obtained an MSc in the Department of Community Health Sciences, Faculty of Medicine at the University of Manitoba. Subsequently, he returned to Ontario to work as an epidemiologist at OMAFRA, and he recently completed an MBA at the University of Guelph. Welcome Tim!

# OAHN Update – June 2023

*Mike Deane, Tanya Rossi*

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

This spring, the OAHN networks held multiple meetings, released reports, and completed many research projects. 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.

**New Resources and Completed Research Projects:**

- OAHN Companion Animal Network Project: Rabies titres in imported dogs
- OAHN Swine Network Project: CanSpot ASF submissions and communications
- OAHN Equine Network Project: Investigation into the seasonal variations in ACTH levels in Ontario's senior horses
- Infographics: Treat Me Right – Acute diarrhea and giardia in cats and dogs

**Continuing Education Videos:**

Lactococcosis in rainbow trout
Trace mineral status in beef herds

The OAHN Aquaculture Network organized a talk with Dr. Veronique LePage who shared the Ontario rainbow trout aquaculture perspective on lactococcosis. The video can be viewed here: https://www.oahn.ca/resources/video-lactococcosis-the-ontario-rainbow-trout-aquaculture-perspective-with-dr-veronique-lepage/

The OAHN Bovine Network organized a webinar to discuss the findings from its Trace mineral status in Ontario beef herds research project. Dr. Cheryl Waldner from the University of Saskatchewan gave a talk on 'Micronutrients deficiencies and their contribution to a variety of herd problems'. The recording of this webinar and Dr. Waldner's talk can be found here (must be registered with OAHN and logged in to view): https://www.oahn.ca/resources/video-trace-status-in-ontario-beef-herds/

**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.

.

- Chicken anemia virus (CAV) in Ontario: Understanding breeder vaccination and broiler flock management
- Poultry veterinary survey highlights – Q1 2023
- Events and news

- Bits ‘N Snips
- Bladder stones, equine coronavirus, melanomas
- Network member reports
- Syndromic and lab surveillance dashboard
- ResearchONequine

- Webinar: Trace mineral monitoring in beef cattle
- OAHN-OABP In-clinic milk culture survey
- *Salmonella* Dublin update
- OAHN Mastitis report
- Surveillance: Q3 and Q4 Animal Health Laboratory data
- Articles of interest

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

# Update on bacterial taxonomy

*Đurđa Slavić, Sarah Lippert, Charles Doekes*

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

AHL Newsletter 2023;27(2):8.

Since advanced molecular methods, whole generation sequencing (WGS) in particular, are being used for microbial characterization, there have been many changes in bacterial taxonomy (**Table 1**). Two of the most frequent changes are description of new species (e.g., *Actinobacillus vicugnae, Glaesserella australis*) and reclassification of species within the existing groups (e.g., *Actinomyces* into *Schaalia*). Here is the summary of the most recent changes that are being reported on AHL cases with bacterial culture results.

**Table 1.** Selected bacterial taxonomy changes.

| **Bacterial species** | **Taxonomy update** |
|---|---|
| *Actinobacillus spp.* | *Actinobacillus vicugnae (isolated from alpacas)* |
| *Actinomyces canis* | *Schaalia canis* |
| *Actinomyces hyovaginalis* | *Schaalia hyovaginalis* |
| *Enterobacter cowanii* | *Kosakonia cowanii* |
| *Glaesserella spp.* | *Glaesserella australis* |
| *Moraxella urethralis* | *Oligella urethralis* |
| *Pasteurella pneumotropica* | *Rodentibacter pneumotropicus* |

# RUMINANTS

## Congenital cardiac vascular malformation in a calf

*Dominique Comeau, Amanda Mansz*

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

AHL Newsletter 2023;27(2):9.

Following the identification of a large heart mass in a young veal calf at slaughter, a partial section of the heart was submitted to the AHL for histologic evaluation. On gross examination, the mass measured 10 cm x 7 cm x 5 cm, had a smooth pink to tan mottled surface, and protruded into what appeared to be the right ventricular chamber. On cut section, there were multiple empty tubular channels and blind-ended pockets lined by fibrous tissue that resembled large blood vessels (**Fig. 1**). On histologic examination, the mass was composed of multiple large vascular channels with thick fibrous tissue walls (**Fig. 2**). These vessels were surrounded by abundant adipose tissue and irregular islands of atrophic heart muscle. Areas of the heart distant from the mass were histologically unremarkable.

Vascular hamartomas have been uncommonly reported in the hearts of cattle at slaughter, and represent benign proliferations of structures endogenous to a specific tissue. Abundant mature adipose tissue has been reported associated with these masses, as was seen in this case. These malformations are not generally associated with clinical disease. *AHL*

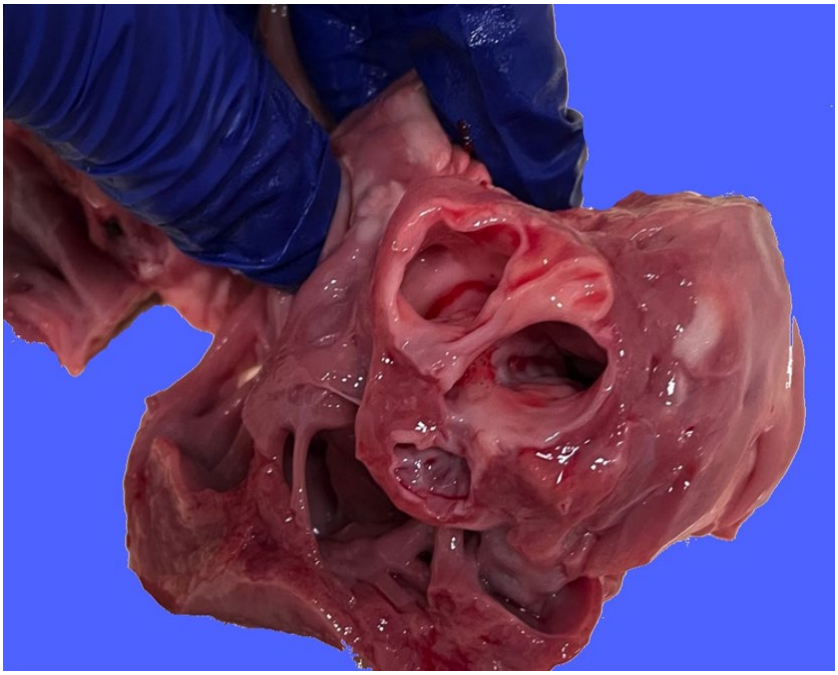


**Figure 1.** Mass composed of multiple large vascular channels within the wall of the presumed right ventricular wall.

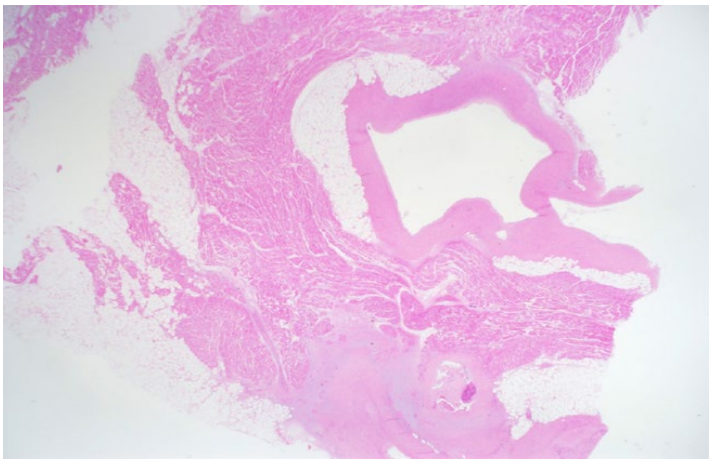


**Figure 2.** Overview of the histologic section of the mass. It is composed of abundant adipose tissue and cardiac muscle containing irregular, large vascular channels. H&E stain.

**Reference**
1. Sugiyama A, et al. Cardiac vascular hamartoma in two slaughtered cattle. J Comp Path 2007;136 (2-3):202-205.

---

# *Yersinia pseudotuberculosis abortions in small ruminants*

*Amanda Mansz*

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

AHL Newsletter 2023;27(2):10.

A postmortem submission of two 2-3 week pre-term Saanen (meat) caprine fetuses from a flock with several recent abortions was received at the AHL in January 2023. At postmortem examination, both fetuses had slightly enlarged livers with subcapsular congestion and hemorrhage with random multifocal pin-point foci of necrosis (**Fig. 1A**). One of two fetuses had a moderate amount of clear abdominal fluid, edema of the abdominal mesentery, diaphragm and subcutaneous tissues of the ventral abdomen/thorax. Renal and pulmonary hemorrhages were noted, and the distal small intestine was dark red to purple on both the serosal and mucosal surfaces (**Fig. 1A**). Microscopic lesions of embolic hepatic and splenic necrosis, suppurative gastroenteritis and suppurative pneumonia were compatible with fetal sepsis (**Fig. 1B**). Subsequently, in March 2023 a Rideau-cross ovine fetus and placenta was received for postmortem. Although visible gross lesions of the fetus were minimal, placental lesions were appreciable and included mild intercotyledonary thickening and opacity with subtle necrosis of cotyledons. Microscopic lesions were also compatible with fetal sepsis, consisting of suppurative pneumonia, epicarditis and abomasitis. There was necrosuppurative placentitis with vascular necrosis and visible rod-shaped bacteria. Bacterial culture of the lung and abomasal content from the caprine fetuses, and of the placenta, lung and abomasal content from the ovine fetus, all grew large numbers of *Yersinia pseudotuberculosis*.

*Yersinia pseudotuberculosis* is a Gram-negative aerobic or facultative anaerobic rod-shaped bacterium implicated in causing various diseases in a wide range of species, and is also a zoonotic food-borne pathogen. *Y. pseudotuberculosis* is found worldwide and can be an inhabitant of the intestinal tract of asymptomatic birds, rodents, livestock, primates, and laboratory animals. Transmission most commonly

occurs by the fecal-oral route following ingestion of contaminated food or water. The bacteria can survive long periods of time in the environment, even at temperature as low as 4 degrees Celsius.

In a 23-year retrospective study conducted in North America, *Y. pseudotuberculosis*–associated disease was diagnosed in 42 goats from 21 counties, with a strong seasonality in winter and spring. The most frequently diagnosed syndromes in ruminants include: enterotyphlocolitis, lymphadenitis, abortion and neonatal death, mastitis, orchitis, septicemia, and ocular disease. The pathogenesis of abortion is thought to follow invasion of the intestinal epithelium of the dam, transient bacteremia, and localization in the maternal caruncle, followed by passage to the chorioallantois and fetus. Most published cases of *Y. pseudotuberculosis* in small ruminants are of individual animals or small groups rather than large outbreaks. This organism is an important documented cause of food-borne illness in humans, and outbreaks have occurred following ingestion of contaminated milk, meat, fresh vegetables, and water. *AHL*

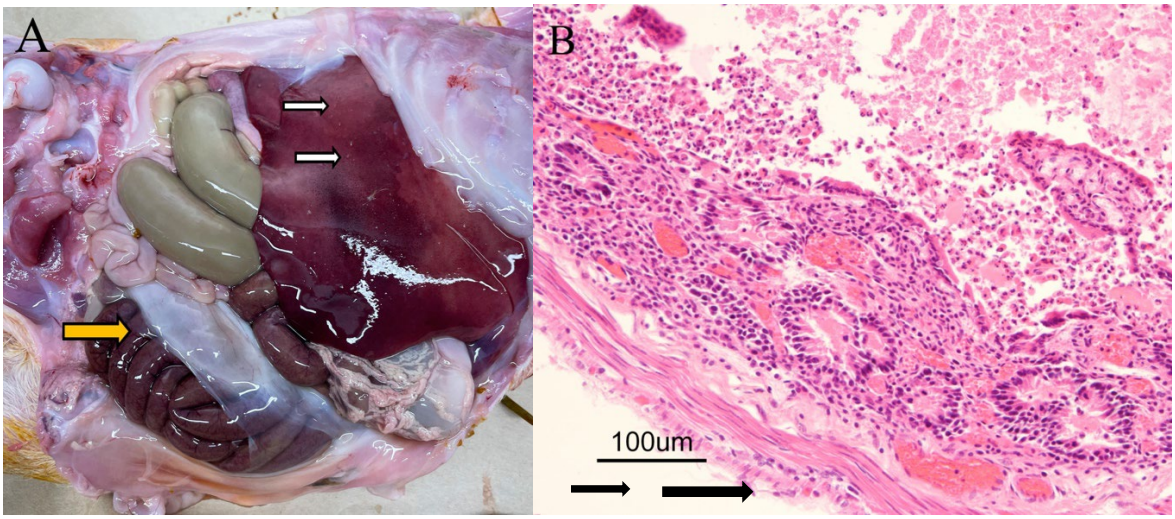



**Figure 1.** Aborted goat fetus with *Y. pseudotuberculosis* sepsis **A.** Blotchy hepatic subcapsular congestion and hemorrhage with multiple pin-point white foci of necrosis (white arrows). Dark red to purple distal small intestine covered with edematous mesentery (yellow arrow) **B.** Rafts of neutrophils and necrotic cellular debris line the flattened and eroded jejunal lumen and fill crypts. H&E stain.

**References**

1. Giannitti F, et al. *Yersinia pseudotuberculosis* infections in goats and other animals diagnosed at the California Animal Health and Food Safety Laboratory System: 1990-2012. J Vet Diagn Invest 2014;26:88-95.
2. Schlafer DH, Foster RA. Female Genital System. In: Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, 6th ed. Maxie MG, ed. Elsevier, 2016;vol 3:413.

# Congenital vitamin A and mineral deficiencies in a beef calf

*Josepha DeLay, Mackenzie Littlejohn*

*Animal Health Laboratory, University of Guelph, Guelph, ON (DeLay), Kirkton Veterinary Clinical, Kirkton, ON (Littlejohn)*

AHL Newsletter 2023;27(2):12.

A near-term beef calf was born prematurely and died a few hours after birth. This was the third premature delivery or late-gestation abortion in this herd of 55 cows over a span of 2 months. One premature calf survived until 3 days of age. Cows were routinely vaccinated against BVDV, viral respiratory pathogens, *Leptospira* spp, and neonatal enteric pathogens.

On postmortem examination, the calf was a normally formed male with features compatible with full gestational age. Multifocally over the entire body, hair was coated with clumps of soft, thick grey putty-like debris (**Fig. 1**). Patchy alopecia and hyperemia were present in skin in inguinal and axillary regions. Internal lesions were limited to blotchy red discoloration in lung. No other significant gross lesions were identified. Placenta was not available for evaluation. The calf had received colostrum by esophageal tube.

Skin lesions correlated microscopically with abundant orthokeratotic hyperkeratotic debris at the epidermal surface and concurrent follicular keratosis (**Fig.2**). Additional histologic lesions in the calf included fibrinosuppurative bronchopneumonia, funisitis (inflammation of the umbilical cord), and unilateral corneal ulcer with marginal granulation tissue. Inflammatory lesions in lung, umbilical cord, and cornea were compatible with an infectious, likely bacterial cause. Given the calf's very young age (hours old), prenatal infection was considered likely, and association with placentitis was suspected. In comparison, cutaneous hyperkeratosis was a more long-standing lesion suggestive of a separate etiology, and raised concern of vitamin A deficiency as the cause. No fungi were detected in association with skin lesions in PAS-stained tissue sections.

Bacterial, viral, or protozoal pathogens were not detected in tissues by bacterial culture or PCR tests for BHV-1, BVDV, bovine adenovirus, *Leptospira* spp, and *Neospora caninum*. Suspected bacterial bronchopneumonia and funisitis were considered likely significant as the cause of the calf's death, but a definitive etiology could not be confirmed.

Liver vitamin A level in this calf was extremely low (0.32 ppm; reference intervals 1.4-4.3 ppm / bovine fetus and 14.0-35.7 ppm / bovine neonate), supporting vitamin A deficiency as the cause for hyperkeratotic skin lesions. Fetuses and neonatal calves have low levels of vitamin A stored in liver compared with older animals, as transfer across the placenta is marginal. Hepatic stores of vitamin A are increased in neonates through ingestion of colostrum. This calf was also deficient in selenium and manganese, based on liver analyses.

Vitamin A is an essential nutrient in mammals. Provitamin compounds (carotenoids) are ingested from plant-based dietary sources, and conversion to an active form (retinol) occurs in intestinal epithelium, with subsequent storage in liver. Poor quality feed and forages produced under drought conditions have lower levels of vitamin A precursors, promoting deficiency. Deficiency of vitamin A adversely affects retinal function and vision, growth and maintenance of epithelial tissues (including skin), bone development, immune function, and reproductive function. In bovine fetuses and neonates, inadequate vitamin A levels most commonly result in neurologic signs including vision loss and ataxia, resulting

from constriction of cranial nerves by defective remodeling of bones of the skull, and abnormal CSF production and flow. Cutaneous hyperkeratosis, as seen in this calf, is infrequently described in vitamin A-deficient neonates but has been documented in juvenile cattle. Vitamin A deficiency rarely occurs in isolation, and testing for other vitamins and minerals can reveal multiple micronutrient deficiencies, as in this case. Review of feeding management in this herd identified inconsistencies in vitamin-mineral supplementation of pregnant cows. The supplement was changed to one specific for late-gestation cows, and feeder space was doubled to improve cow access. The herd will continue to be monitored over the next gestational period. *AHL*


**Figure 1.** Abundant clumped putty-like debris (keratin) at skin surface over head of bovine neonate (arrows).

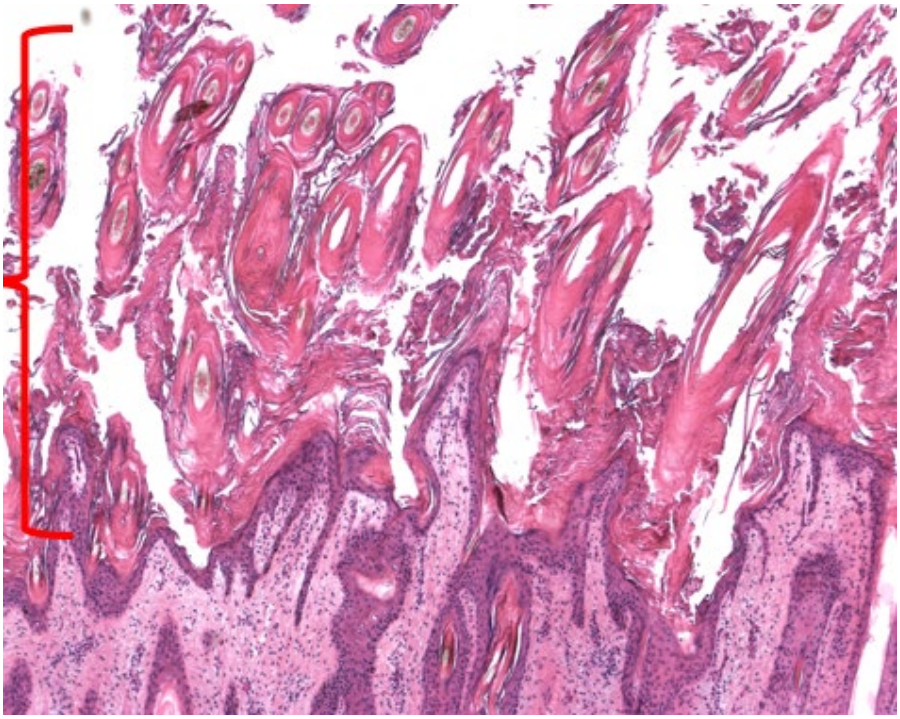


**Figure 2.** Massively thick layer of hyperkeratotic debris at epidermal surface of haired skin in a bovine neonate with vitamin A deficiency (red bracket). H&E stain.

**References**

1.Baldwin TJ, et al. Dermatopathy in juvenile Angus cattle due to vitamin A deficiency. J Vet Diagn Inv 2012;24(4):763-766.
2.Dewell G. Vitamin A deficiency in beef calves. Iowa State University Extension and Outreach publication, 2014. https://store.extension.iastate.edu/product/Vitamin-A-Deficiency-in-Beef-Calves
3.Hill BD, Holroyd, RG, Sullivan M. Clinical and pathological findings associated with congential hypovitaminosis A in extensively grazed beef cattle. Aust Vet J 2009;87(3):94-98.
4.McDowell LR. Vitamin A. In: Vitamins in Animal and Human Nutrition. Iowa State University Press, 2000:15-90.

# First detection of *Glaesserella australis* in swine herds in Ontario

*Đurđa Slavić, Sarah Lippert and Madison McGrogan*

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

AHL Newsletter 2023;27(2):15.

For the first time, *Glaesserella australis* was confirmed in two different swine herds in Ontario. Samples from multiple pigs were submitted to the AHL from both herds, and *G. australis* was isolated from pericardium and lung of one pig in each herd. In both cases, a variety of other pathogens were also detected including *Streptococcus suis*, *Pasteurella multocida*, *Actinobacillus porcitonsillarum*, influenza A, and PRRSV.

A novel *Glaesserella* sp. causing disease in Australian pigs was first time reported in 2018, followed by a new species description of *Glaesserella australis* in 2020. This novel bacterial species closely resembles *Glaesserella parasuis,* and it has been isolated from clinically affected pigs with signs of bronchopneumonia and from lung lesions and abscesses detected in abattoirs from clinically healthy pigs. Distribution of the lung lesions did not have any specific pattern, with abscesses scattered throughout dorsal as well as cranioventral lobes. However, clinical and histopathological findings highly resemble the ones caused by *Actinobacillus pleuropneumoniae* which may be explained by the fact that some *G. australis* isolates carry genes with 84% similarity to ApxIII cytotoxin of *A. pleuropneumoniae*. While the full virulence potential of these isolates remains to be established, their prevalence in Australian pigs appears to be low, with only 5.6% (3/54) farms being positive for this bacterium.

Given the very recent isolation of *G. australis* in Ontario, information about its prevalence and clinical significance also needs to be established. At AHL, all swine submissions are routinely checked for the presence of *G. australis* since November of 2022, and based on preliminary findings (i.e., 2 positive farms) the prevalence in clinical cases appears to be low. AHL will continue to monitor the prevalence of *G. australis*, and will provide updates as they become available. *AHL*

**References**

1. Sun X, et al. An improved multiplex PCR for *Actinobacillus pleuropneumoniae*, *Glaesserella australis* and *Pasteurella multocida*. J Microbiol Methods 2021;191:106360.
2. Turni C, et al. *Glaesserella australis* sp. nov. isolated from the lungs of pigs. Int J Syst Evol Microbiol 2020;70:3686-3692.
3. Watt AE, et al. A novel *Glaesserella* sp. isolated from pigs with severe respiratory infections has a mosaic genome with virulence factors putatively acquired by horizontal transfer. Appl Environ Microbiol 2012;84:e00092-18.

# First detection of porcine astrovirus-4 in suckling pigs in Ontario

*Rebecca Egan, Kevin Vilaca*

*Animal Health Laboratory, University of Guelph, Guelph, ON (Egan); South West Veterinary Services, Stratford ON (Vilaca)*

AHL Newsletter 2023;27(2):16.

In November of 2022, a diagnostic work-up was initiated to investigate the cause of increased pneumonia and loss of condition in nursery pigs within three barns receiving piglets sourced from a single 3000 pig farrow-to-wean sow herd. Overall, pigs were in good condition upon arrival to the nursery, with occasional coughing that seemed to subside within the first week. In the second week post-placement, several pigs in these nurseries began to lose condition and developed signs of pneumonia, such as coughing and increased respiratory effort. Sick pigs were treated with antibiotics, and the mortality rate peaked at approximately 7%, with most of the losses occurring in the second and third weeks post-placement. On-farm postmortems were performed on nursery pigs from each barn, revealing lungs that failed to collapse. A small amount of fibrin was noted on the surface of the lung in one animal.

Tissues from piglets obtained from each nursery were submitted to the AHL for histopathology and ancillary testing. Microscopic examination identified a variety of pulmonary lesions, including bronchopneumonia (nursery A, two pigs), interstitial pneumonia with chronic pleuritis (nursery B, three pigs), and pulmonary edema (nursery C, one pig). Bacterial cultures grew *Streptococcus dysgalactiae* from a pooled sample of heart and lung collected from one of the pigs with bronchopneumonia. Involvement of *Mycoplasma hyopneumoniae* was suspected based on the histologic appearance, but further testing was not performed. *Streptococcus suis* was isolated from thoracic and brain swabs from nurseries B and C, and this pathogen is suspected to have been playing a role in the development of disease in these groups. A definitive cause for the interstitial pneumonia in nursery B was not determined. Porcine reproductive and respiratory syndrome virus (PRRSV) and porcine circovirus-2 (PCV-2) were not detected in various samples, including oral fluids, serum and lung tissue. There was no immunohistochemical detection of influenza A, PCV-2, or PRRSV antigens in microscopic sections of lung. PCR testing for porcine parainfluenza virus-1 (PPIV-1) was performed on three lung samples (2 from nursery B and one from nursery C), and one sample from nursery B was positive with a high Ct value (34.5). In light of these findings, it is unclear whether PPIV-1 was directly involved in the development of pneumonia in these animals.

Upon further investigation of the sow barn, a pronounced cough had been noted in the weeks prior. Postmortem examination was performed on three piglets, and the sole gross finding was patchy lobular pulmonary atelectasis in cranioventral lung, similar to influenza virus infection (**Fig. 1**). Samples were submitted to the AHL for histopathology and ancillary testing, and all three pigs were found to have microscopic lesions of suppurative pneumonia with epithelial attenuation and/or hyperplasia affecting multiple airways (**Fig. 2**), suggestive of cellular injury caused by epitheliotropic viral infection. Pig 1 also had suppurative lymphadenitis and systemic lesions compatible with septicemia; however, bacterial culture was not performed to identify a causal pathogen. PCR testing did not detect influenza A in oral fluids, and PRRSV, PCV-2, and PPIV-1 were not detected in lung samples. In addition, there was no immunohistochemical detection of influenza A, PCV-2 or PRRSV antigens in microscopic sections of lung. PCR testing was also negative for *Mycoplasma hyopneumoniae*. Lung samples were then submitted for PCR testing to assess for the presence of porcine astrovirus type 4 (PoAstV4) which has recently been described in association with respiratory disease in young pigs. This virus was detected in

all three lung samples (Ct values of 31.8, 29.8, and 30.4), and RNA *in situ* hybridization confirmed the presence of viral antigen in affected respiratory epithelium in 2 of the 3 pigs (**Fig. 3B**). Not long after this diagnostic investigation, the cough in the sow barn resolved and nursery performance improved. It was hypothesized that introduction of large numbers of gilts following PRRSV eradication earlier that year may have led to reduced immunity in the gilt litters, which combined with viral respiratory infection in suckling pigs, may have predisposed animals to various infections in the nursery.

At present, there are five known lineages of porcine astrovirus, causing neurologic disease, enteric disease, or asymptomatic infection. More recently, detection and characterization of a novel genotype of PoAstV4 from nasal swabs obtained from suckling pigs with acute clinical respiratory disease has been reported, and while a causal link between PoAstV4 and respiratory disease in pigs has not been fully established, there is research to suggest that infection may contribute to the development of clinical disease. These results highlight the importance of assessment for PoAstV4, as well as other viruses such as PPIV-1 and porcine hemagglutinating encephalomyelitis virus (PHEV), in cases where microscopic lesions suggestive of epitheliotropic viral infection are present in the trachea and/or upper airway and influenza virus is not identified. Also, given the distribution of these lesions, it is crucial to routinely collect trachea for microscopic examination (formalin-fixed) and ancillary testing (fresh/frozen) when performing a diagnostic investigation of porcine respiratory disease. *AHL*

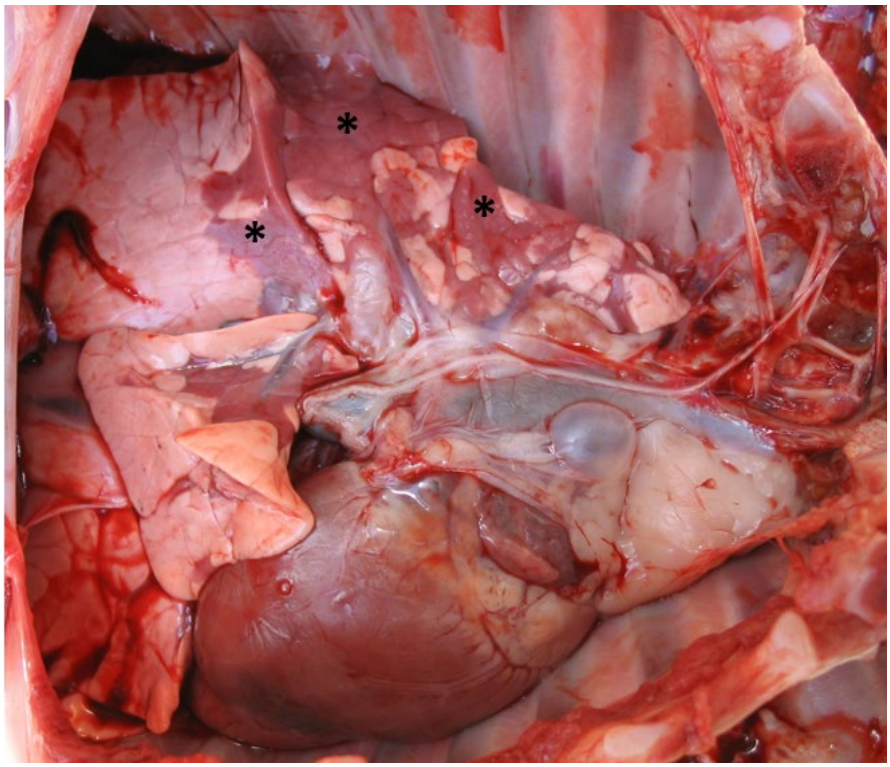



**Figure 1.** Nursing pig. Cranioventral lung exhibits multifocal to coalescing regions of lobular atelectasis (*).

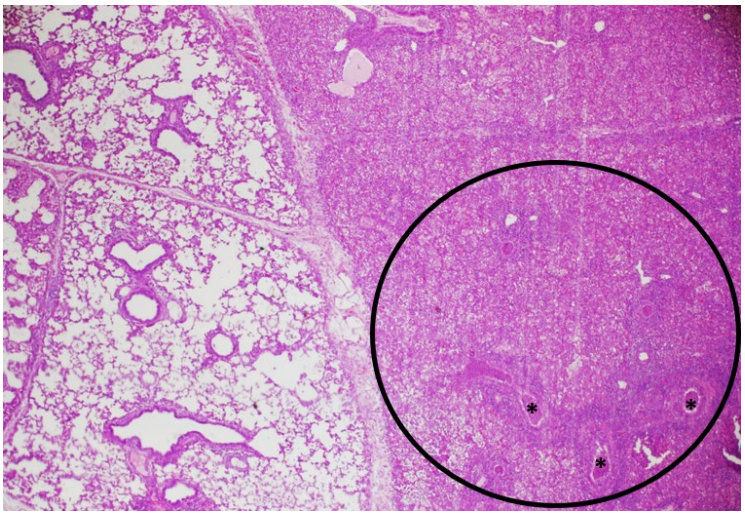


**Figure 2.** Nursing pig. Microscopic section of lung (H&E stain, 4x). Affected lobules on the right half of the image correspond to the grossly noted areas of lobular atelectasis, and multiple small airways contain luminal accumulation of neutrophils (*), accompanied by mild alveolar edema with variable accumulation of macrophages and neutrophils.

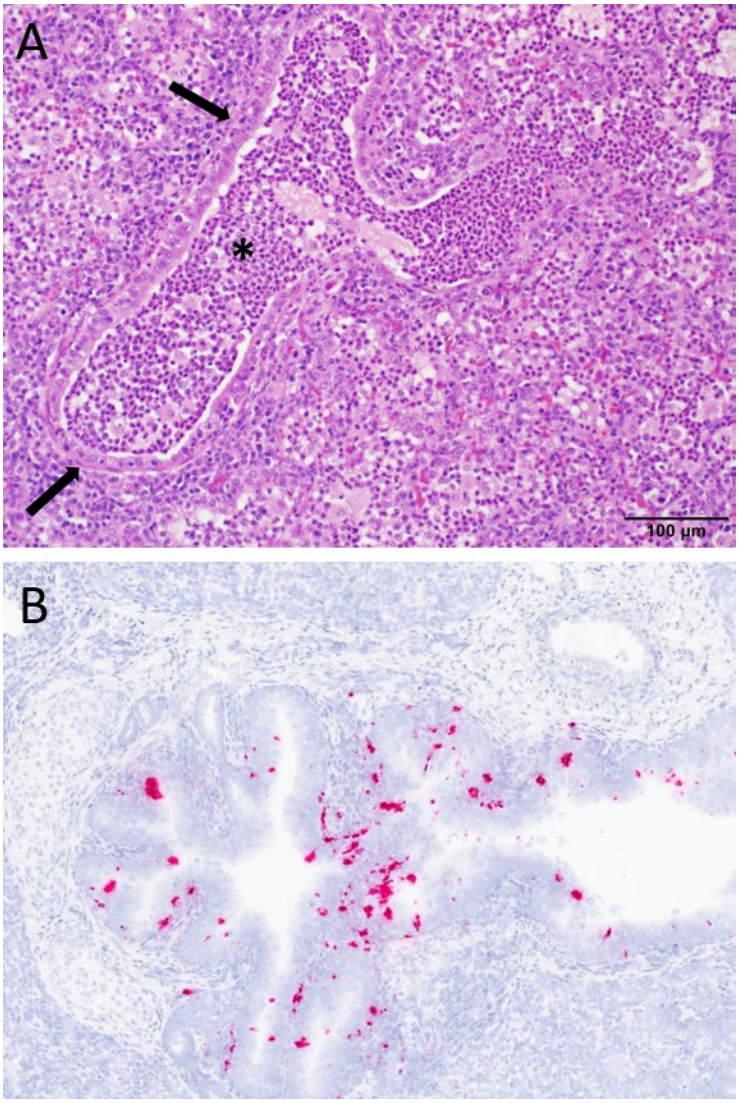



**Figure 3.** Nursing pig. Microscopic sections of lung (H&E stain, 20x). **A.** Bronchiolar epithelial attenuation (arrows) with luminal accumulation of suppurative exudate (*), as well as accumulation of neutrophils and macrophages in adjacent alveoli. **B.** RNA *in situ* hybridization assay demonstrating moderate staining for PoASTV4 in bronchial epithelium. (Photo courtesy of Dr. Derscheid, Iowa State Veterinary Diagnostic Laboratory).

**References**

1. Rahe MC, et al. Porcine Astrovirus-4 detection in lesions of epitheliotropic viral infection in the porcine respiratory tract. Transbound Emerg Dis 2023, Article ID 9113355.
2. Padmanabhan A, Hause BM. Detection and characterization of a novel genotype of porcine astrovirus-4 from nasal swabs from pigs with acute respiratory disease. Arch Virol 2016;161(9):2575–2579.

# *Enterococcus cecorum*: Changing clinical presentation after 2 decades in an emerging poultry pathogen

*Emily Martin*

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

AHL Newsletter 2023;27(2):19.

Since 2002, pathogenic strains of *E. cecorum* have been identified in chickens, starting with outbreaks in Scotland and the Netherlands, then progressing to other countries including Canada, USA, Iran, and South Africa. Prior to 2002, *E. cecorum* was identified as a commensal in avian species (e.g., turkeys, Muscovy ducks, pigeons, canaries) and mammals such as horses, cattle, pigs, dogs, and cats; however, but identification of pathogenic strains has had a significant impact on broiler and broiler breeder health and production. **While *E. cecorum* emerged as primarily causing bone lesions and lameness, recent outbreaks have manifested as septicemia at an earlier age.**

*E. cecorum* infection causes two stages of disease. Initially, 2 to 3-week-old broilers can become infected but remain asymptomatic, or have clinical signs of depression (i.e., ruffled feathers, closed eyes). Bacterial sepsis can cause lesions of hepatitis, pericarditis and splenomegaly resulting in increased mortality. Subsequently, 5 to 6-week-old broilers / broiler breeders (or slightly older broiler breeders ~13 weeks old) develop lameness or bilateral paralysis where birds can be found sitting with their legs extended forward. Mortality is usually higher at this stage due to culling, dehydration and starvation.

On postmortem examination, joint lesions typified by synovitis and arthritis of the coxofemoral, stifle, and hock joints have accumulation of creamy yellow fluid and sometimes caseous material resulting in joint swelling. Spondylitis and osteomyelitis can be identified in the free thoracic vertebra (FTV), proximal femur, or proximal tibia. Lesions in the free thoracic vertebra and adjacent vertebral bodies develop into a dorsally-expansile inflammatory mass that causes spinal cord compression resulting in bilateral paralysis. In severe cases, this vertebral mass expands ventrally into the celomic cavity and creates a localized enlargement of the spinal column that can be seen in the area of the caudal lungs / cranial kidneys.

*E. cecorum* lesions are similar to those caused by other infectious agents; therefore, bacterial culture is required to confirm the causative agent. It is possible to culture *E. cecorum* from the FTV and femoral heads without observing gross lesions. Commensal and pathogenic strains co-exist, and virulence varies among pathogenic strains. In multiple studies, it was found that commensal isolates have higher diversity whereas pathogenic isolates originating from different countries group phylogenetically. These belong predominantly to one clade, and are distinct from the commensal poultry isolates. There are few virulence factors identified in both the commensal and pathogenic strains of *E. cecorum*.

Even after 2 decades, the pathogenesis and transmission of *E. cecorum* is still under investigation, not only in chickens, but also in other avian species. It is known that pathogenic strains colonize the intestines and spread rapidly through a flock. It is also known that *Enterococcus* spp. (including *E. cecorum*) survive well in the environment, and this could be the source of repeated infections within a barn. Research has shown that *E. cecorum* survives longer in litter than in dust or on PVC material, and that there is longer survival at lower temperatures and at lower relative humidity. Pathogenic strains

showed a longer survival time than commensals under similar conditions.

Currently, our understanding of *E. cecorum* is still evolving in avian species. Further research into transmission and sources of infection will aid in developing prevention and management strategies. *AHL*


**References**

1. 2023 ACPV Workshop. Enterococcus: An old dog with new tricks. March 12, 2023 Sacramento, CA. https://aaap.memberclicks.net/assets/ACPV_Workshops/2023_Workshop/2023_ACPV%20Workshop%20Program.pdf
2. Wijetunge DS, et al. Fingerprinting of poultry isolates of *Enterococcus cecorum* using three molecular typing methods. JVDI 2012;24(6):1166-1171. https://doi.org/10.1177/1040638712463563
3.Schreier J, Rautenschlein S, Jung A. Different virulence levels of *Enterococcus cecorum* strains in experimentally infected meattype chickens. PLoS ONE 2021;16(11):e0259904. https://doi.org/10.1371/journal.pone.0259904
4.Jung A, et al. A review of *Enterococcus cecorum* infection in poultry. Avian Diseases. 2018;62(3), 261–271. https://www.jstor.org/stable/26537546
5.Laurentie J, et al. Comparative genome analysis of *Enterococcus cecorum* reveals intercontinental spread of a lineage of clinical poultry isolates. mSphere 2023;8(2):1-21. https://doi.org/10.1128/msphere.00495-22
6.Grund A, Rautenschlein S, Jung A. Tenacity of *Enterococcus cecorum* at different environmental conditions. J Appl Microbiol 2021;130:1494-1507. https://doi.org/10.1111/jam.14899


---

# Selection of environmental tests for detection of *Enterococcus cecorum*


*Đurđa Slavić, Sarah Lippert*

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



AHL Newsletter 2023;27(2):20.


There are 3 different environmental culture tests offered by the AHL bacteriology section (**Table 1**). Two of the methods target specific bacterial pathogens such as *E. coli*, *Pseudomonas aeruginosa*, *Salmonella* spp., and *Clostridium perfringens,* whereas the third test is used for detection of aerobic but not anaerobic growth (i.e., not *C. perfringens*) in the environment. When interested in the presence of any aerobic bacterial pathogens, including *Enterococcus cecorum,* the 'acule3' test needs to be requested. If applicable, it is recommended to specify which bacterial species one is looking for on the submission form. *AHL*

**Table 1.** Environmental tests selection in AHL bacteriology section

| Test name | Test code(s) | Description |
|---|---|---|
| Bacterial culture, environmental 1 | *acule* | Testing for the presence of *E. coli*, *Pseudomonas aeruginosa*, *Salmonella* spp., and *Clostridium perfringens*. |
| Bacterial culture, environmental 2 | *acule2* | Testing for the presence of *E. coli*, *Pseudomonas aeruginosa* and *Salmonella* spp. |
| Bacterial culture, environmental 3 | *acule3* | Testing for the presence of aerobic bacterial growth. |

# Investigation into the circannual variation in endogenous plasma ACTH concentrations in Ontario senior horses

*Kristiina Ruotsalo, Alison Moore*

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

AHL Newsletter 2023;27(2):21.

PPID (pars intermedia pituitary dysfunction) is an endocrine disorder that occurs in over 20% of aged horses, ponies and donkeys. Clinical signs and measurement of plasma endogenous ACTH (eACTH) concentrations currently form the basis of diagnosis, as dynamic testing, such as the thyrotropin-releasing hormone (TRH) stimulation test, is not easily accessible due to the lack of a readily available source of thyrotropin. A number of factors can affect eACTH concentrations, including individual variation, timing of sampling with respect to the course of disease, and increases related to illness, pain, or stress, thus possibly confounding clinical interpretation. Additionally, the equine pituitary gland exhibits a circannual rhythm in the release of eACTH. This circannual increase has previously been referred to as a 'seasonal or fall' increase, however it is now recognized that rather than exhibiting a sharp autumnal peak in eACTH concentrations, gradually increasing secretory activity begins during the summer, peaks, and then declines. The timing of this circannual increase has been shown to vary with respect to geographic location, and is speculated to be linked to photoperiods. The circannual increase occurs both in clinically healthy horses and those affected with PPID, although the increase in affected horses is typically of greater magnitude.

A preliminary study was undertaken with the support of the OAHN Equine Network to characterize the onset, duration, and magnitude of the circannual increase in eACTH concentrations in a group of clinically healthy senior Ontario horses of various breeds and both sexes. Participating veterinarians recruited horses aged 15 years or older for monthly eACTH sampling with the owner's consent. Horses were considered healthy based on physical examination, health status questionnaire, and unremarkable study intake CBC and biochemistry profile results. No horses receiving medication for PPID, EMS, thyroid hormone supplementation, or corticosteroids were entered into the study. Horses remained in Ontario for the duration of the sampling period. No ponies, donkeys, mules or miniature horses were included, due to the potential breed effect on eACTH concentrations. Timing of sample collection was not standardized, however, a minimum of one hour post exercise or training was recommended.

EDTA plasma samples were received frozen and analyzed using an ACTH chemiluminescent immunoassay and Immulite 1000 instrumentation (Siemens, Canada) at the Animal Health Laboratory.

Twenty-three horses, ranging in age from 16 to 23 years entered the study. Seven horses completed all 12 months of consecutive sampling. Remaining horses were sampled 5 to 11 times. The reasons for the loss of horses from the study were not investigated.

Due to limited eACTH results at some time points, descriptive analysis of data was deemed most appropriate. Average eACTH concentrations approached 10 pmol/L in June and July, reached a peak value of 17.3 pmol/L in September, remained mildly increased in October, and then resumed concentrations of <10 pmol/L, thus supporting a circannual increase in eACTH from June to October (**Fig. 1**).

A large degree of individual variation with respect to the magnitude and onset of increase in plasma eACTH concentrations was evident.

Data from two horses was excluded as eACTH concentrations significantly exceeded 10 pmol/L at most measured time points, reaching values greater than 100 pmol/L during the months of June to October. These two horses were reportedly clinically normal, but the possibility of occult PPID needs to be considered. Horses with PPID do exhibit evidence of circannual increases in eACTH, although these increases are exaggerated in comparison to clinically healthy individuals.

Additional investigations involving a larger cohort of clinically well senior horses, targeting the June to October sampling interval, will be required to establish statistically significant reference intervals.

In the interim, the following guidelines for interpretation of eACTH concentrations may be clinically helpful, recognizing that the circannual change in eACTH is gradual and subject to individual variation:

- December to mid-June: eACTH concentrations > 10 pmol/L may support a diagnosis of PPID in horses with the appropriate signalment and clinical signs;
- July to mid-November: eACTH concentrations >20 pmol/L may support a diagnosis of PPID in horses with appropriate signalment and clinical signs;
- Individual horses may have higher circannual increases in eACTH concentrations, with a return to baseline in late winter / early spring;
- As with all laboratory tests, eACTH results must be interpreted in the context of age, clinical history, and physical examination findings.

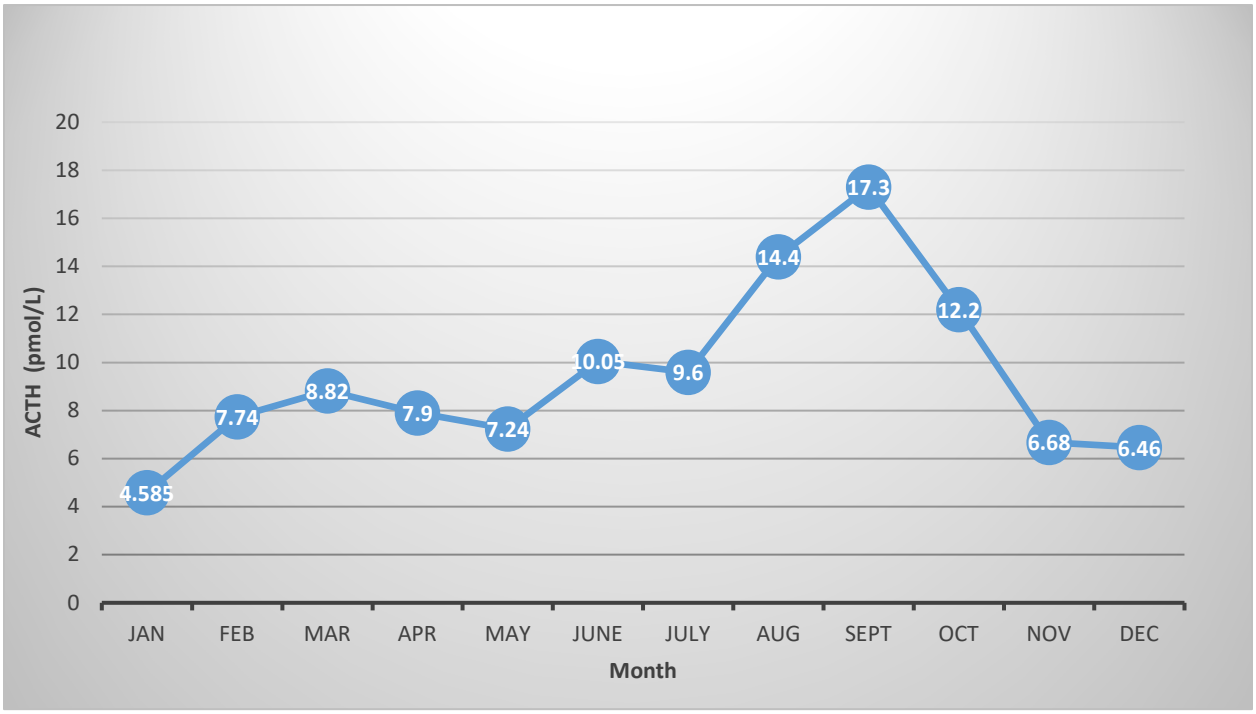



**Figure 1.** Average monthly plasma eACTH concentrations in a group of Ontario senior horses.

We would like to thank all of the veterinarians participating in this study, the horses, and their owners.
*AHL*

**References**
1.Durham A, et al. Clinically and temporally specific diagnostic thresholds for plasma ACTH in the horse. Equine Vet J 2021:53:250-260.
2. Durham AE, Potier JF, Huber L. The effect of month and breed on plasma adrenocorticotropic hormone concentration in equids. The Veterinary Journal 2022;286:105857.
3. Equine Endocrinology Group: Recommendations for the Diagnosis and Treatment of Pituitary Pars Intermedia Dysfunction (PPID). http://sites.tufts.edu/equineendogroup.

This study is available on the OAHN.ca and ResearchONequine.ca websites:

OAHN Equine Network Project: Investigation into the seasonal variations in ACTH levels in Ontario's senior horses - Ontario Animal Health Network
OAHN Projects - ResearchONequine

---

# *Interstitial pneumonia in a donkey*

*Amanda Mansz*

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

AHL Newsletter 2023;27(2):23.

Following the death of a 12-year-old middle-aged donkey with acute signs of respiratory distress and fever, the animal was submitted to the AHL for postmortem. On gross examination, the lungs failed to collapse and there were discrete rib impressions along the dorsal pleural surfaces. Approximately 80% of the right lung lobes and 40% of the left had multifocal nodular to regionally diffuse areas of pleural and parenchymal fibrosis represented by regions of firm, non-aerated pallor. At this stage, two different respiratory syndromes that affect donkeys were being considered.

Donkey pulmonary fibrosis (DPF) is a spontaneous syndrome documented in the lungs of 35% of aged donkeys (mean age 30 years). DPF can be an incidental finding at autopsy or may result in chronic respiratory disease and debilitation. Grossly, DPF is expected to appear as multifocal and coalescing large foci of pulmonary pleural fibrosis over the dorsal lungs which can occasionally extend ventrally. The most predominant microscopic lesion described in DPF is pleural and subpleural fibrosis; however, the severity and extent of the fibrosis can vary. The cause of DPF remains unknown and there is no evidence that the development of DPF is related to lung infection with asinine herpesviruses (AsHV).

The alternative diagnosis is a different syndrome termed interstitial "fibrosing" pneumonia in donkeys which has been associated infection with AsHV-4 and AsHV-5. The classic gross lesions are lungs that fail to collapse with patchy or multifocal firm lesions that more commonly affect the cranioventral areas. Histologically, lesions are significantly more inflammatory than DPF, and consist of a mix of bronchiolitis and interstitial pneumonia with the presence of multinucleated syncytial cells. In some cases, dramatic interstitial fibrosis is a feature. Diagnosis is based on PCR testing to detect AHV-4 or AHV-5 in association with the characteristic histologic lesions.

Microscopic lesions noted in this case consisted of severe generalized pulmonary interstitial fibrosis with histiocytic, lymphocytic and neutrophilic bronchointerstitial pneumonia and syncytial cell formation (**Fig. 1)**, warranting a diagnosis of interstitial pneumonia likely associated with AsHV-4 and AsHV-5 infection. AHL does not currently offer in-house testing for AsHV-4 and 5; however, send-out PCR testing for AsHVs is available through UC Davis in California. *AHL*

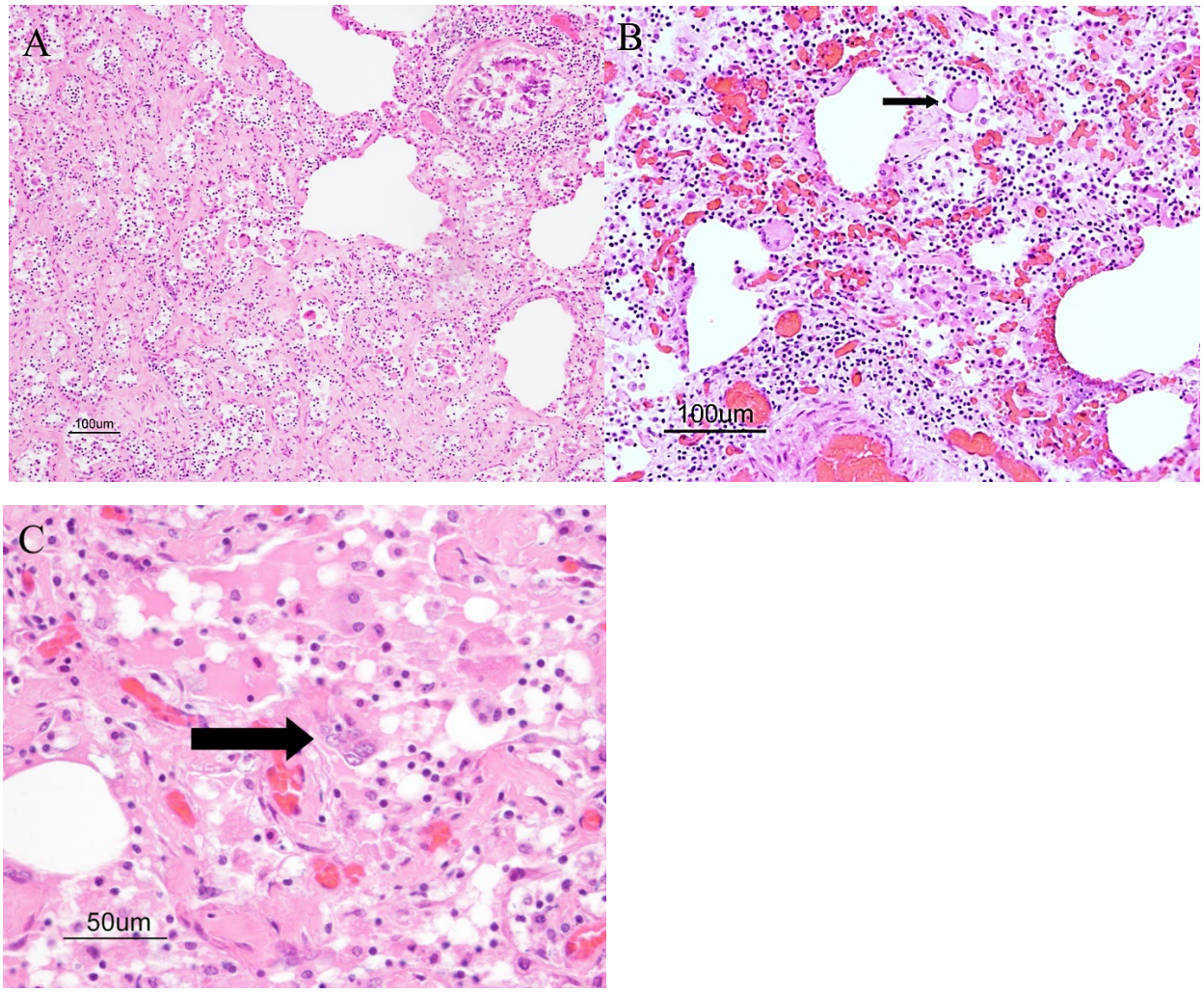



**Figure 1.** Histologic lesions in donkey lung compatible with interstitial fibrosing pneumonia**.** H&E stains. **A.** Massive expansion of the alveolar interstitium by dense collagen bundles (fibrosis) that compress adjacent alveoli. 10x. **B.** Bronchiolar epithelium is denuded, and airspaces contain numerous macrophages, lymphocytes, fewer neutrophils and large multinucleate cells (arrow). 20x. **C.** Syncytial cell (arrow) within a bronchiole. 40x.

**References**

1. Maboni G, et al. Detection of asinine gammaherpesviruses in association with pulmonary fibrosis in free-ranging donkeys. J Vet Diagn Invest 2022;34(1):167-171.
2. Caswell JL, Williams KJ. Respiratory System. In: Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, 6th ed. Maxie MG, ed. Elsevier, 2016;vol 2:514, 568.

# Parvoviral myocarditis in littermate puppies and a review of AHL cases 2013-2023

*Emily Brouwer, Dominique Comeau, Patricia Lechten*

*Animal Health Laboratory, University of Guelph, Guelph, ON (Brouwer, Comeau); Allandale Veterinary Hospital, Barrie, ON (Lechten)*

AHL Newsletter 2023;27(2):25.

Two littermate golden retriever puppies were submitted to the Animal Health Laboratory for postmortem examination approximately three weeks apart. The puppies originated from an unregistered breeder, and were subsequently adopted by a rescue organization. Of the six puppies in the litter, three had died suddenly and one was euthanized due to acute respiratory distress.

The first puppy that was submitted for postmortem examination was nine weeks old at the time of death. He had a four day history of slightly laboured breathing followed by sudden collapse and cardiopulmonary arrest. The puppy received routine vaccinations approximately one week prior to death. There were no clinical abnormalities noted at the time of vaccination.

The second puppy was eleven weeks old at the time of death. He was euthanized following an episode of labored breathing starting two days prior. At the time of his hospitalization, the postmortem examination had been performed on his littermate. Based on his clinical signs and the diagnosis made in his sibling, it was presumed that he was in congestive heart failure.

Postmortem examination of the first puppy revealed relatively mild gross lesions of pulmonary edema, meningeal congestion and petechial to ecchymotic hemorrhages within the pancreas. Lesions were more dramatic in the second puppy which had diffuse pulmonary edema and mild pleural effusion. Gross cardiac lesions were limited to the second puppy and were characterized by diffuse myocardial pallor and mild biventricular dilation. On cut section, the myocardial pallor was most pronounced in the interventricular septum (**Fig. 1**). The heart weight ratios were within published reference ranges for this species.

On histologic examination, the first puppy had extensive multifocal loss of cardiomyocytes with replacement by thin bands of edematous collagen, lymphocytes and plasma cells. Cardiomyocytes in affected areas were often shrunken with angular cell outlines. Rarely, the cardiomyocyte nuclei contained irregular, poorly-defined basophilic material suspicious for viral inclusion bodies. The lesions in the second puppy were more chronic in nature, with extensive replacement of the myocardium by fibrous connective tissue interspersed with clusters of lymphocytes and plasma cells (**Fig. 2**). Pulmonary lesions in both dogs were compatible with acute congestive heart failure.

Both puppies were positive for canine parvovirus using real-time PCR with cycle thresholds of 25.52 and 32.07, performed on scrolls of heart and fresh tissue in puppies one and two, respectively. The antigen was also demonstrated within the myocardial lesions using immunohistochemistry. Positive immunoreactivity was detected in both animals, both within the inflammatory population as well as the cardiomyocytes. Testing of the first puppy for canine herpesvirus, another common cause of myocarditis in puppies, was negative. Following these ancillary test results, a diagnosis of parvoviral myocarditis was made in both puppies.

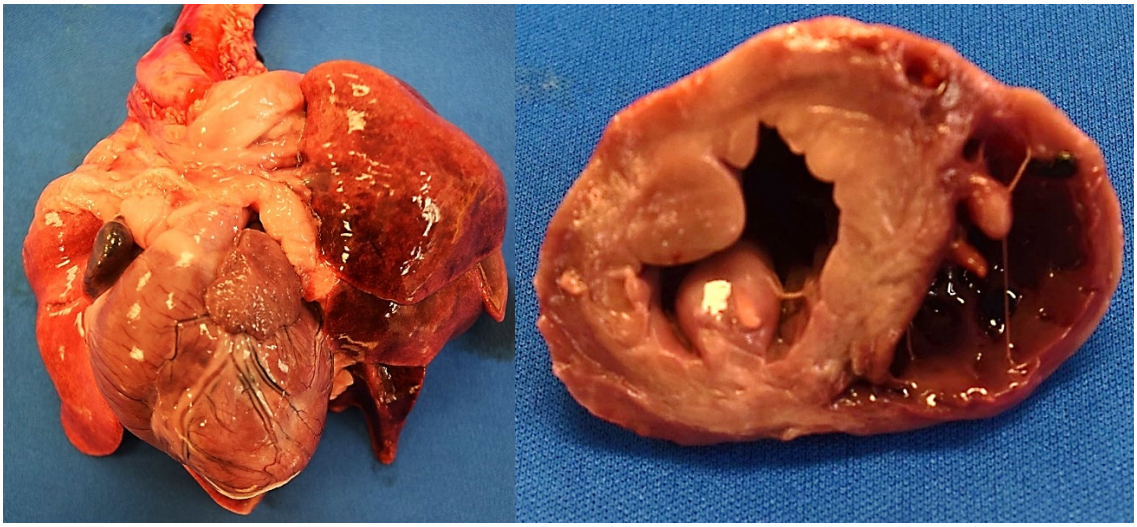


**Figure 1.** Heart of puppy number two with ventricular myocardial pallor (left). Cross section of ventricular myocardium (right).

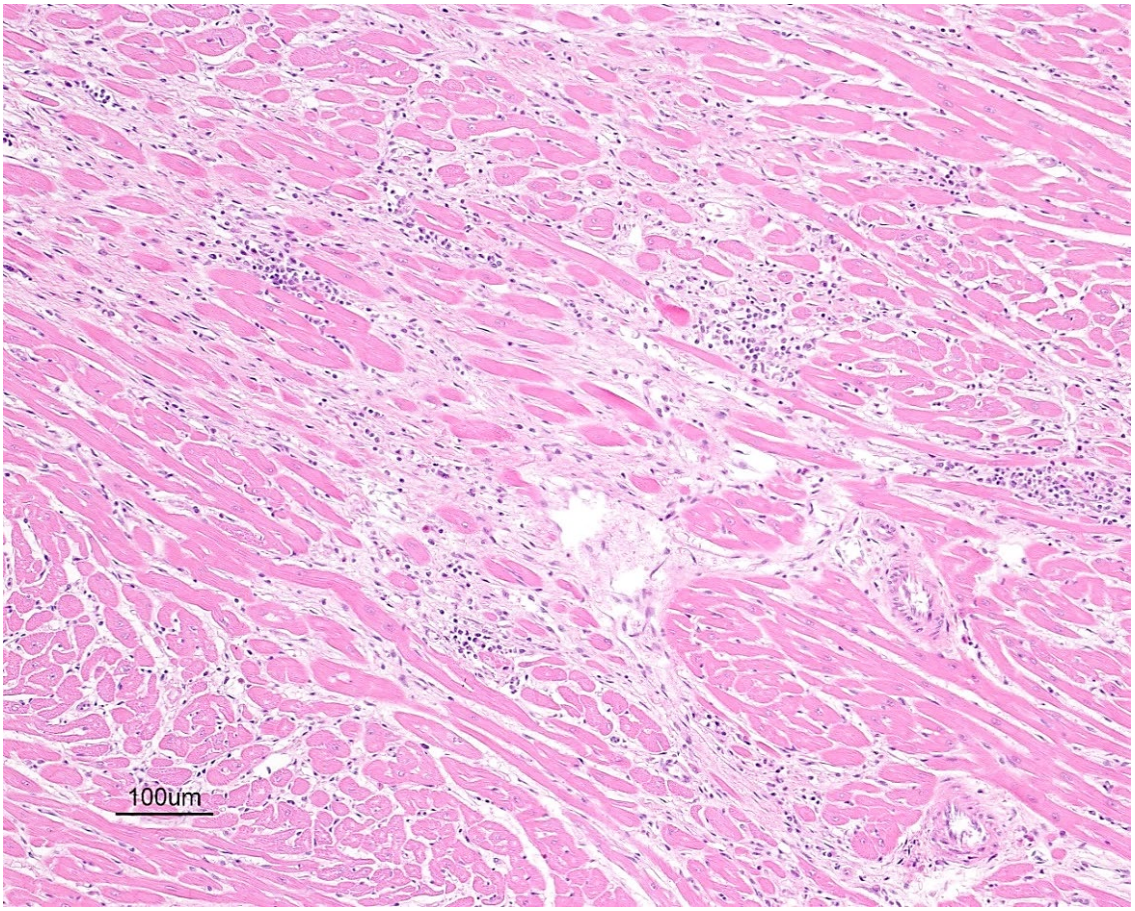



**Figure 2.** Myocardium of puppy number two, H & E stain, 10X. Note extensive fibrosis, nonsuppurative inflammatory infiltrates, and loss of cardiomyocytes.

In the last ten years, only five cases of parvoviral myocarditis have been diagnosed at the Animal Health Laboratory (**Table 1.**). These puppies ranged in age from 5.5 weeks to 11 weeks of age. Two were mixed breeds, one was a French bulldog, and two were golden retrievers. All had similar lesions of cardiomyocyte necrosis and lymphoplasmacytic inflammation with varying degrees of fibrosis. The etiology in all cases was confirmed using real-time PCR, immunohistochemistry, or both ancillary test procedures.

**Table 1.** Parvoviral myocarditis cases submitted to the AHL between 2013 and 2023.

| **Breed** | **Age** | **Myocardial fibrosis** | **PCR** | **IHC** |
|---|---|---|---|---|
| Mixed breed | 5.5 weeks | + | + (Ct 22.01) | + |
| Mixed breed | 8 weeks | +++ | + (Ct 25.56) | n/a |
| French bulldog | 9 weeks | + | + (Ct 30.87) | - |
| Golden retriever | 9 weeks | + | + (Ct 25.52) | + |
| Golden retriever | 11 weeks | +++ | + (Ct 32.07) | + |

While most are familiar with the common diarrheal illness attributed to canine parvovirus-2 infection, widespread vaccination efforts have greatly diminished the incidence of myocarditis in young puppies. Development of myocarditis occurs when there is perinatal infection of the puppies either within the first two weeks of life, or if the naïve dam is infected late in gestation and *in utero* transmission occurs. Infection of proliferating cardiomyocytes results in necrotizing myocarditis, and those that survive the initial infection typically go on to develop nonsuppurative inflammation and myocardial fibrosis which impacts myocardial function. The amount of virus detectable within the heart will decrease as the infected myocardiocytes are replaced by fibrosis, and may not be detectable in a long-surviving animal. In the subset of AHL cases from the last ten years, five animals with lesions of nonsuppurative myocarditis were positive for parvovirus on PCR, but of these five, one case was PCR positive and the virus was not detectable using immunohistochemistry, and one case was not confirmed with immunohistochemistry.

The two surviving littermates of the submitted golden retrievers were assessed by a cardiologist following the diagnosis of their siblings. Both surviving puppies had echocardiograms performed which identified mild left ventricular dysfunction with subtle left-sided dilation in one puppy, and mild systolic dysfunction in the other puppy. Neither required medical intervention, though continued monitoring, including baseline troponin, was recommended. *AHL*

**References**

1. Ford J, et al. Parvovirus infection is associated with myocarditis and myocardial fibrosis in young dogs. Vet Pathol 2017;54(6):964-971.
2. Uzal FA, Plattner BL, Hostetter JM. Chapter 1:Alimentary System. In: Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, 6th ed. Maxie MG, ed. Elsevier, 2016;vol 2:154.
3. Robinson WF, Robinson NA. Chapter 1:Cardiovascular System. In: Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, 6th ed. Maxie MG, ed. Elsevier, 2016;vol 3:42-43.

# Ulcerative dermatitis and vasculitis due to cutaneous *Pseudomonas aeruginosa* infection following grooming

*Dominique Comeau, Hailey Bertrand-Sluman*

*Animal Health Laboratory, University of Guelph, Guelph, ON (Comeau); Parkside Animal Hospital, North Bay, ON (Bertrand-Sluman)*

AHL Newsletter 2023;27(2):28.

Skin biopsies from an 8-year-old female spayed Labrador retriever crossbred dog were submitted to the Animal Health Laboratory to investigate extensive ulcerative and erosive lesions in the skin over the dorsum, sides, and lateral hindlimbs (**Fig. 1**). The dog had initially presented with a fever of unknown origin two days prior to the acute development of the skin ulcers. All other workup performed, including bloodwork, radiographs, urinalysis, and ultrasound were within normal limits. The dog was shaved to allow the skin lesions to be fully cleaned, injectable antibiotics and steroids were administered, and she was started on prednisone.

On histologic examination of skin biopsies, there was extensive ulceration of the skin surface which was often covered in a thick crust of fibrin, neutrophils, and cellular debris (**Fig. 2**). There was marked inflammation of the underlying dermis, and occasionally, there was separation of the remaining epidermis from the dermis. Within the dermal inflammation, there was targeting of the small superficial blood vessels by neutrophils, with destruction of some of the vessel walls (**Fig. 3**). The vessel walls contained neutrophils which were often degenerating, consistent with leukocytoclastic vasculitis. The intact epidermis distant from the ulcers was histologically unremarkable, making immune-mediated conditions such as toxic epidermal necrolysis / erythema multiforme or bullous pemphigoid less likely. Primary cutaneous vasculitis was considered, however, this would typically involve the deeper larger blood vessels rather than the small superficial vessels noted in this case.

Overall, the skin lesions histologically resembled a burn with secondary hypersensitivity or infection-induced vasculitis. This was suggestive of a severe bacterial infection with a staphylococcal-type bacterium, resulting in a condition known as "staphylococcal scalded skin syndrome". This syndrome occurs due to the production and secretion of toxins by the bacteria, leading to sloughing of the superficial layers of skin.

A swab of the lesions was submitted for bacterial culture and had abundant growth of *Pseudomonas aeruginosa in* pure culture. This bacterium is common in wet or aquatic environments, and is a common cause of skin infections following swimming or bathing. It can produce toxins similar to those produced by *Staphylococcus* spp., leading to a lesion which is indistinguishable from scalded skin syndrome. *Pseudomonas aeruginosa* can be found in water and is also a contaminant in opened shampoos and bath products. Additional history revealed this dog had been bathed with a deshedding shampoo a few days prior to development of the lesions.

This dog was continued on a combination of immunosuppressive and antimicrobial therapy. The lesions resolved over several weeks (**Fig. 4**). *AHL*

*The assistance of Dr. Robert Foster (Ontario Veterinary College) in the examination and workup of this case is gratefully acknowledged.*

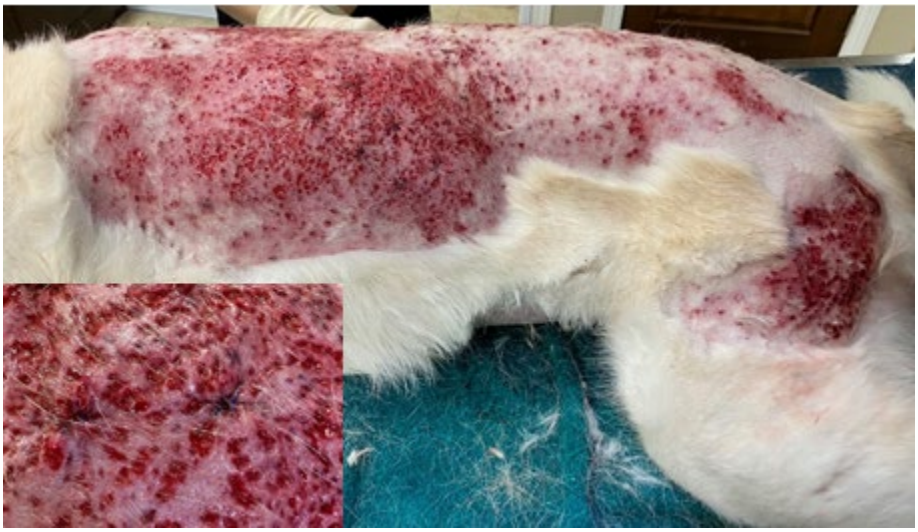


**Figure 1.** Extensive ulcerative skin lesions at the time of initial presentation. Inset: closer view of the skin ulcers.

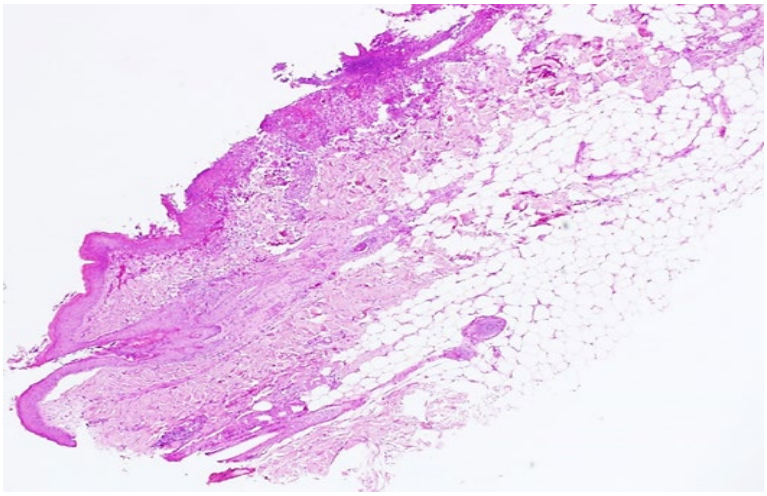


**Figure 2.** Overview of a sample of the skin with extensive ulceration of the surface, covered by a thick crust.

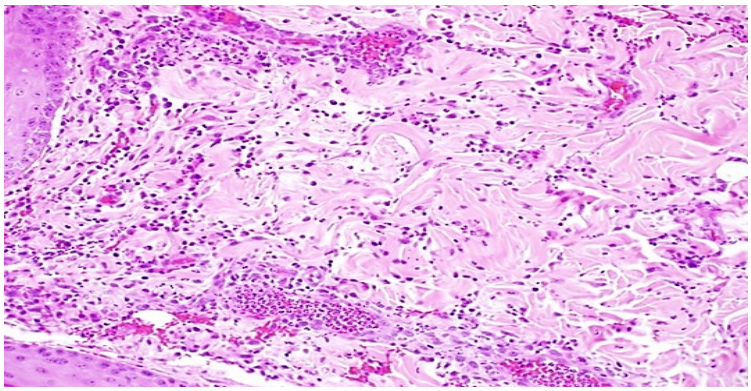


**Figure 3.** Neutrophilic inflammation of the dermis, centered on small blood vessels.

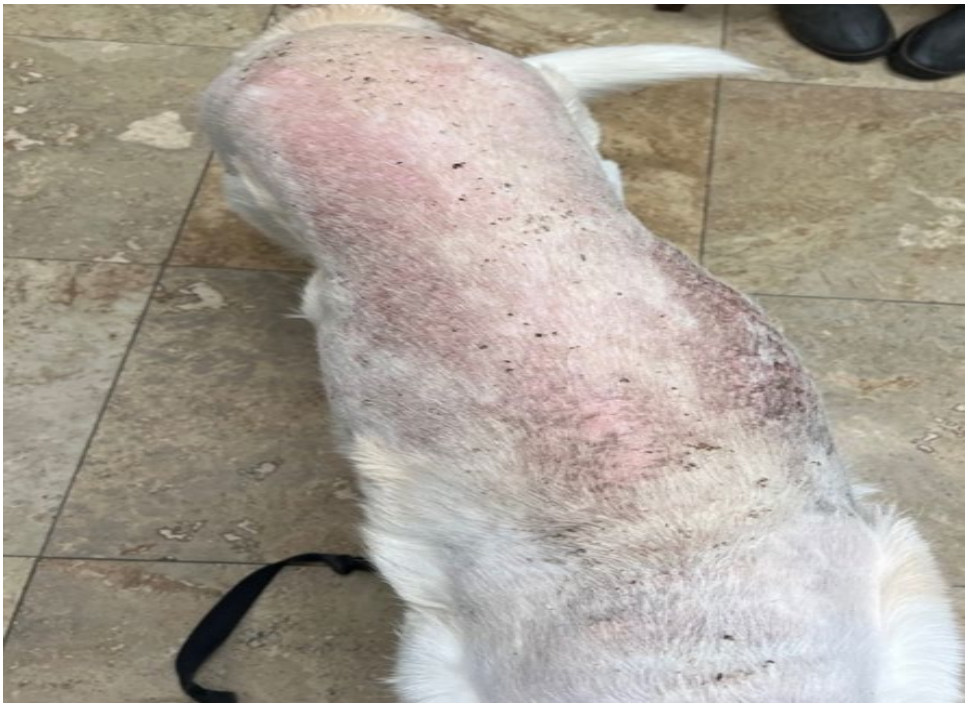


**Figure 4.** Skin lesions resolving approximately 20 days after submission of biopsy samples.