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Animal Health Laboratory

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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Getting the most out of your diagnostic laboratory submissions

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AHL Newsletter 2020;24(1):2.

It is understood that any diagnostic workup needs to have results that are accurate, meaningful and interpretable as the endpoint. This workup should provide a cost-effective approach (best "bang for the buck") to solving the problems that are encountered.

More than once, I have heard in our AHL anatomic pathology rounds that the diagnostic outcome would have been better if only "X" had been done. It behooves us all to make sure that we adhere to the best "diagnostic standard of care" (1) and minimize diagnostic errors when possible.

Listed below are some common questions and issues that we encounter.

- To test or not to test: will the test results be used to direct a change or take an action? This thought process can become complex as it may take more than a single test to establish a pattern from "normal".
- Samples numbers: this is especially important in population medicine to provide confidence with a diagnosis or confirm freedom from disease.
- Avoiding common diagnostic errors:
    - Point of care and in-clinic testing - while these can provide valid, rapid answers, please keep in mind all of the issues around sample integrity, calibration, maintenance and quality control.
    - Insufficient information - no history provided on the submission form. This precludes our laboratory diagnosticians from getting involved and helping with the interpretation of diagnostic results. The complete medical history is not necessary, just a brief summary of your thought process around the case.
    - Wrong sample submitted - we are always available to consult if you have questions!
    - Sample degradation - the cold chain (hot vs cold weather), spinning serum samples, including EDTA blood smears, using virus transport media. These are just some examples to help mitigate deterioration of samples that interfere with laboratory testing.
    - Selecting tests that are not "fit for purpose" - antigen vs antibody testing and biology-related issues that affect sensitivity and specificity. Please consult us if unsure which test is best suited for the diagnostic outcome that you are seeking.
    - Sample selection - consult the AHL Users' Guide and diagnostic plans. A full set of formalin-fixed and fresh tissues increases diagnostic success. Fresh tissues can be held at the laboratory until preliminary histopathology results direct appropriate microbiologic analysis, thereby decreasing overall testing cost.

We encourage you to contact us and discuss your disease investigation so that we may assist you in getting the most out of your diagnostic laboratory submissions. *AHL*

Reference

1. Thompson BS. On the journey for great diagnostics – Tips and tricks to let the magic happen. AABP, St. Louis, Sept. 2019.

AHL new tests developed in 2019

TEST - METHOD	CODE	SPECIES
Aeromonas salmonicida - PCR	asalpcr	Fish
Aves polyomavirus (AvPyV)-PCR; Beak and feather disease virus (BFDV)-PCR; Psittacid herpesvirus (PsHV)-PCR	psitpcr	Avian
Chlamydia suis - real-time PCR	csuipcr	Porcine
Fish processing charge, non-food (up to 4 fish)	kfpmmf	Fish
Fish viral hemorrhagic septicemia virus (VHSV) - PCR	vhsvc	Fish
Flavobacterium psychrophilum - real-time PCR (Bacterial cold water disease)	fpsypcr	Fish
Mycoplasma synoviae - vlhA gene sequencing typing	mstype	Avian
Nucleic acid (RNA/DNA) extraction from paraffin embedded tissues	parafex	All species
Ornithobacterium rhinotracheale (ORT) - ELISA	orte	Avian
Pasteurella multocida – antibody ELISA - Turkey	pmturke	Avian
Salmonella Pullorum-typhoid - microplate agglutination test	salmp	Avian
Scrapie resistance PrP genotyping in goats (codons 146, 211, and 222) - sequencing	prpgoat	Caprine
Toxoplasma gondii - PCR	toxopcr	Bovine, Caprine, Feline, Ovine

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Selected zoonotic pathogens and diseases, 2019

Murray Hazlett, Đurđa Slavić, Davor Ojkic, Hugh Cai

Animal Health Laboratory, University of Guelph, Guelph, ON

AHL Newsletter 2020;24(1):3-4.

Many new, emerging, and re-emerging diseases of people are caused by pathogens originating from animals, or are shared between people and animals. The AHL plays an important role in public health by identifying zoonotic pathogens in > 1,000 cases annually **(Table 1)**.

The percentage of cases identified as positive for leptospirosis decreased in 2019 in all species, and the total number of submissions tested also decreased slightly, except for dogs **(Table 2)**. These are numerator data reliant upon submission biases to the diagnostic laboratory and cannot be regarded as population prevalence estimates. These do not take into account vaccination status, as all species except horses may be routinely vaccinated for leptospirosis. Monitoring programs are not included in this data.

There was a large increase in tests for *Brucella canis*, and the results shown are positive by 2ME-RSAT. In samples sent to the Public Health Ontario Mycobacterium Laboratory, *Mycobacterium tuberculosis* was confirmed in a dog. *AHL*

Table 1. Number of cases with selected zoonotic pathogens isolated and/or identified at the AHL in 2019.

Agent	Bovine	Swine	Equine	Ovine	Caprine	Chicken	Turkey	Canine	Feline	Other	2019	2018
Ascarids (incl *T. canis, T. cati, T. leonina, Baylisascaris* sp.)	1	5	12			29	1	19	2	6	75	69
Blastomyces dermatitidis								5			5	14
Bordetella bronchiseptica		33	8				1	6	3	11	62	50
Borrelia burgdorferi (Lyme disease), serology			21					6			27	26*
Brucella sp. *(non-abortus)*								79			79	2
Campylobacter coli/ jejuni/ fetus subsp. *fetus*	1	2		7	1			3	1	2	17	15
Chlamydia sp.				4	5						9	17
Clostridium difficile		1	3		1						5	6
Coxiella burnetii (Q fever)	8			27	27						62	67
Cryptococcus sp.										1	1	3
Cryptosporidium sp.	232		1	3	9					7	252	257
Eastern equine encephalitis virus			7								7	7
Echinococcus multilocularis								2			2	5
Giardia sp.	7							31			38	9
Listeria monocytogenes	10		1	11	4					3	29	31
Methicillin-resistant *Staph aureus* (MRSA)		1	2					1			4	3
Methicillin-resistant *S. pseudintermedius* (MRSP)								43	4	1	48	64
Rabies virus										9	9	3
Salmonella enterica	75	65	5	9		35	64	3	1	7	264	338
Streptococcus suis	4	130				1			1	4	140	121
Streptococcus equisimilis	3	35	23	1	1			5		3	71	68
Streptococcus zooepidemicus	6		162	4				1	3	1	177	149
Toxoplasma sp.				5	3				2		10	13
Verotoxigenic *E.coli* (VTEC)	7	3			1						11	18
West Nile virus			1							24	25	47
Yersinia enterocolitica	3	1						2			6	5
Total	357	276	246	71	52	65	66	206	17	79	1435	1385
*correction												
For *Brucella canis* number represents positive 2ME-RSAT tests												

Table 2. *Leptospira* spp. seropositive, IHC-positive, or PCR-positive cases identified at the AHL, 2019.

Leptospira spp. serovar	Bovine	Swine	Equine	Canine	Other
L. autumnalis	24	12	18	65	0
L. bratislava	83	24	29	44	4
L .canicola	29	19	6	45	2
L. grippotyphosa	16	4	7	47	0
L. hardjo	82	11	8	16	0
L. icterohaemorrhagiae	54	21	11	59	1
L. pomona	59	9	14	57	0
IHC or PCR-positive	0	0	0	4	0
Positive/tested cases	**59/205**	**4/26**	**32/51**	**114/214**	**4/17**
%positive,2019/2018	29/41%	67/27%	63/78%	53/65%	57/20%

OAHN update – March 2020

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AHL Newsletter 2020;24(1):5.

The Ontario Animal Health Network has been busy throughout the winter, releasing new podcasts, infographics, as well as small flock poultry veterinary videos. Read on to find links and descriptions of what we've been working on.

EVH-1 Infographic and Canine Staphylococcal Pyoderma Podcast

The OAHN Equine network created a new infographic on equine herpesvirus-1 abortion. The infographic was prepared to help horse owners learn about EHV-1 and how to minimize risk for their horses.

The OAHN Companion Animal network created a new podcast: Management and Treatment of Canine Staphylococcal Pyoderma. In this episode, OAHN Companion Animal network co-lead Dr. Maureen Anderson is joined by Dr. Doug DeBoer, Professor of Dermatology from the University of Wisconsin-Madison and Diplomate of the American College of Veterinary Dermatology, to discuss management and treatment of antimicrobial resistant infections, and how to help avoid increasing resistance issues from canine staphylococcal pyoderma.

OAHN Small Flock Poultry Video Series

OAHN has been working with Dr. Victoria Bowes, a diagnostic avian pathologist with a special interest in small flock medicine, to produce a series of videos to assist veterinarians treating small flock poultry. We have recently released the videos on the topic of **acute mortality in small flock poultry**; these can be found here: https://oahn.ca/small-flock/

Completed Research Projects

Each OAHN network has embarked on one or more research initiatives related to disease surveillance for their specific species.

- OAHN Companion Animal Research Project: Update of Infection Prevention and Control - Best Practices for Small Animal Veterinary Clinics - https://oahn.ca/resources/oahn-companion-animal-research-project-infection-prevention-and-control-best-practices-for-small-animal-veterinary-clinics/
- OAHN Companion Animal Research Project: *Brucella canis* in Commercial Dog Breeding Kennels in Southern Ontario - https://oahn.ca/resources/oahn-companion-animal-research-project-brucella-canis-in-commercial-dog-breeding-kennels-in-southern-ontario/
- OAHN Bovine Network Project: OAHN Dairy Veterinary Antimicrobial Sales Benchmarking 2018 - https://oahn.ca/resources/oahn-bovine-network-project-oahn-dairy-veterinary-antimicrobial-sales-benchmarking/

Aquatic Animal Health Report

The semi-annual report from the OAHN Fish network on Aquatic Animal Health is now available online at:
https://oahn.ca/reports/aquatic-animal-health-report-december-2019/

We have lots of other **new reports, lab data, and resources**. Be sure to check out **OAHN.ca**

Increased detection of BRSV associated with respiratory disease in cattle

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AHL Newsletter 2020;24(1):6-7.

This winter, the AHL and the Ontario Animal Health Network (OAHN) received multiple reports of increased rates of respiratory disease in cattle, including outbreaks of pneumonia in lactating dairy cattle. The OAHN Bovine Network will summarize a recent survey of bovine practitioners about the problem in its next quarterly report in March 2020. This article summarizes the frequency of detection of viruses associated with bovine respiratory disease (BRD) at the AHL from Jan. 2017 to Jan. 2020.

Bovine respiratory syncytial virus (BRSV) was the most commonly detected virus. Bovine coronavirus (BCoV), bovine herpesvirus-1 (BoHV-1) and bovine parainfluenza virus-3 (BPIV-3) were detected sporadically. The frequencies of BRSV, BCoV, BoHV-1, and BPIV-3 detected by PCR are summarized in **Fig 1**.

Peak detection of BRSV occurs in the winter months. From Oct. 2019 to Jan. 2020, the proportion of positive BRSV PCR results **increased to 33%, compared to 24% positive in 2018/19 and 15% positive in 2017/18.** Approximately the same number of BRSV PCR tests were performed during this same 4 month period in all 3 winters.

BRSV is an important cause of pneumonia in calves and adult cattle. Acute herd outbreaks are often associated with high morbidity. Mortality rates may vary depending on the severity of co-infections with other pathogens such as *Mannheimia haemolytica*. In uncomplicated BRSV pneumonia, the lesions typically have a cranioventral distribution and the affected lung tissue is red, depressed, and firm. Histologically there is bronchointerstitial pneumonia with syncytial cells, and viral inclusion bodies may be visible depending on the stage of infection (**Fig. 2**). Concurrent bacterial infection may result in severe fibrinous bronchopneumonia.

Although clinical signs and lesions may suggest BRSV infection, further laboratory testing is often required to confirm the diagnosis. BRSV can be detected by PCR performed on nasopharyngeal swabs (in virus transport medium) or fresh lung tissue. Note that BRSV is included in the bovine respiratory virus panel PCR test along with BoHV-1 and BPIV-3. BRSV antigen can be detected in formalin-fixed tissues by immunohistochemistry (**Fig. 3**). To detect seroconversion to BRSV, submit paired sera for the virus neutralization test. Please refer to the AHL User's Guide and AHL website https://www.uoguelph.ca/ahl/diagnostic-plans/diagnostic-plans-bovine for more information pertaining to sample and test selection. *AHL*

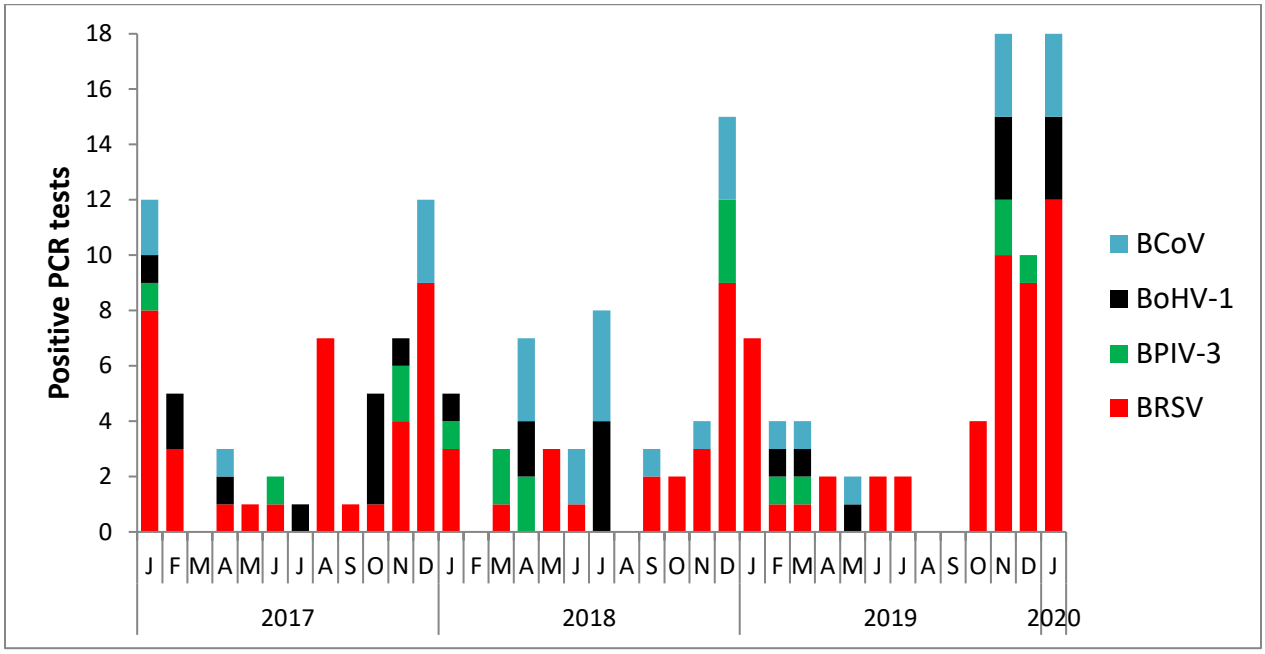

Figure 1. Frequency of bovine respiratory syncytial virus (BRSV), bovine coronavirus (BCoV), bovine herpesvirus-1 (BoHV-1), and bovine parainfluenza virus-3 (BPIV-3), detected by PCR at the AHL from Jan. 2017 to Jan. 2020.

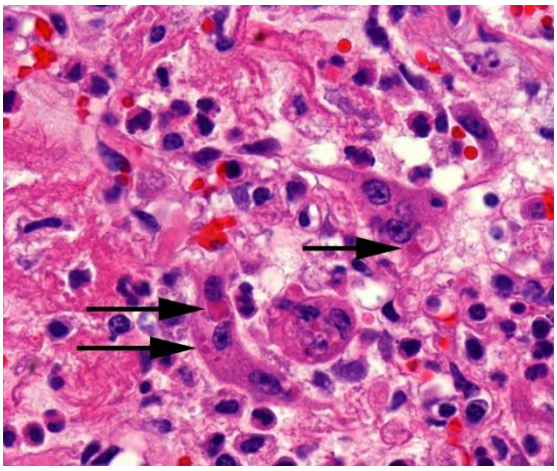

Figure 2. Bronchointerstitial pneumonia in a calf due to BRSV. Note syncytial cells with intracytoplasmic inclusion bodies (arrows).

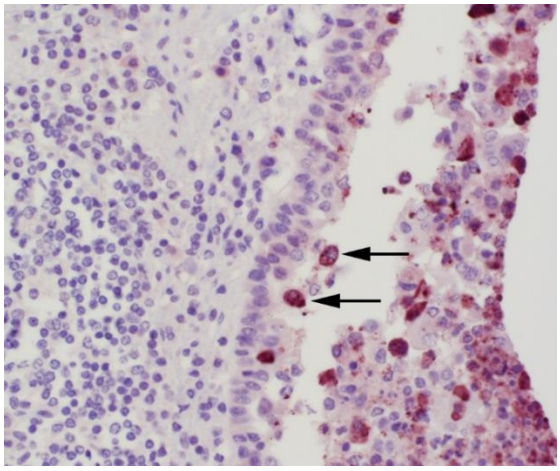

Figure 3. Detection of BRSV in formalin-fixed tissue by immunohistochemistry. Note BRSV (brown chromogen, arrows) within the inflamed bronchiole.

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Mycotic tonsillitis and cervical lymphadenitis in finisher pigs

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AHL Newsletter 2020;24(1):8-9.

A farrow-to-finish swine operation of approximately 100-150 sows and 800 grower/finishers developed a problem affecting approximately 10% of animals in the finishing barn. Pigs presented with variably-sized serous-filled cutaneous swellings that were restricted to the cervical region, measuring up to 15 cm in diameter. No bedding was used in the finishing barn, and the feed was mixed on site. One of the affected pigs was euthanatized and samples were collected from internal viscera (no gross abnormalities noted), as well as the cervical cutaneous nodules. These were submitted to the Animal Health Laboratory for histopathologic examination and bacterial culture.

Significant histologic lesions were restricted to the sections of tonsil and cervical cutaneous lesions. Tonsillar architecture was partially effaced and parenchyma expanded by focally extensive accumulations of infiltrating leukocytes that included eosinophils, lymphocytes, neutrophils and numerous multinucleated giant cells that often contained cross-sections of bulbous fungal hyphae (**Fig. 1**). Sections of the cutaneous nodules were indeterminate regarding site; however, the presence of a distinct capsular boundary was most consistent with lymph node origin. These sections contained extensive foci of necrosis and numerous clusters of karyorrhectic leukocytes. A few scattered multinucleated giant cells containing fungal hyphae were also present. Fungal hyphae were accentuated in a PAS-stained section (**Fig. 2**). A Ziehl Neelsen-stained section was negative for the presence of acid fast bacteria. A diagnosis of granulomatous tonsillitis and cervical lymphadenitis with intralesional fungal hyphae was made.

Bacterial culture of the cervical nodules isolated mixed and variable growths of *Staphylococcus aureus, E. coli, Streptococcus suis* and *S. dysgalactiae* subsp. *equisimilis*. Since numerous fungal hyphae were seen on routine wet mount of the affected tissues, mycotic cultures were also set up. *Geotrichum* spp. was isolated from 3 out of 3 samples; *Mucor* spp. was also isolated from 1 of the 3 tissues. A search of the veterinary literature revealed a single case report of mycotic tonsillitis in a free-ranging weaner pig from Australia due to *Geotrichum candidum* . *Geotrichum* spp. is a saprophytic fungus found on fruits, vegetables, and dairy products. The authors of this report speculated that the feeding of macadamia nut trash may have inoculated the tonsils of this pig with this uncommon organism. Sporadic cases of geotrichosis in other species, including humans, are often linked to immunosuppression.

Additional history was obtained from the herd veterinarian in this case. **The presumed source of infection was poor quality, moldy ensiled high moisture corn. Tonsillar inoculation by fungi following oral exposure with subsequent dissemination to cervical lymph nodes was the probable mechanism for development of the cervical cutaneous nodules**. *AHL*

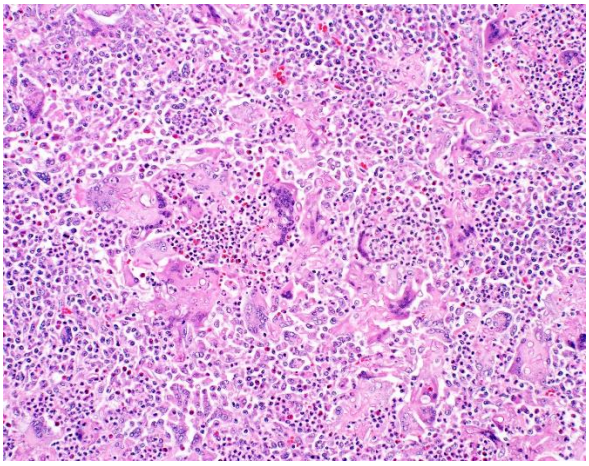

Figure 1. Granulomatous tonsillitis associated with numerous infiltrating multinucleated giant cells (H&E).

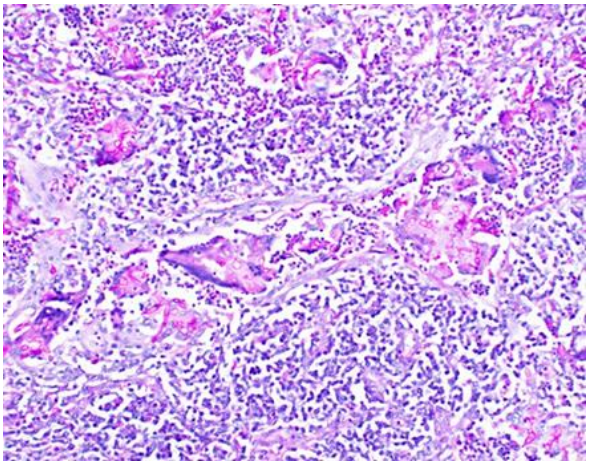

Figure 2. PAS-stained section revealing positively-staining intracellular fungal hyphae.

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Streptococcus equi subsp. *zooepidemicus* update

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AHL Newsletter 2020;24(1):9.

Streptococcus equi subsp. *zooepidemicus* is a Gram-positive commensal bacterium frequently present on mucosal surfaces. Although primarily considered an equine pathogen, *S. zooepidemicus* can cause infections in a variety of animal species, including humans. Most recently, *S. zooepidemicus* was reported as causing abortions, conception failures, and sudden death in gilts and mature sows in Manitoba on four epidemiologically-linked premises. Following this initial report, sudden death of pigs caused by *S. zooepidemicus* at two different Manitoba assembly yards and at slaughterhouses in Canada and the USA was observed. These cases were also epidemiologically linked to the index premises. *Streptococcus zooepidemicus* was consistently isolated from all cases and isolates were submitted for whole genome sequencing (WGS). WGS revealed that these isolates are closely related to the *S. zooepidemicus* ATCC 35246 strain that caused significant losses in the swine industry in China in 1976.

A search of the AHL database since May 2007 revealed isolation of *S. zooepidemicus* from various animal species. As expected, this bacterium was predominantly cultured from equine samples followed by bovine, feline, canine, small ruminants and porcine. Occasionally, it was also isolated from alpacas, camels, llamas, grey squirrels, ferrets, chinchillas, deer, and birds. A closer examination of porcine cases (12 in total including 4 research cases) revealed that *S. zooepidemicus* had been isolated only once (2011) in pure culture from a joint sample. In all other cases, *S. zooepidemicus* was isolated in mixed culture in combination with *Streptococcus suis*, *Staphylococcus aureus*, *Bordetella bronchiseptica*, or *Trueperella pyogenes*. In 4 research cases, *S. zooepidemicus* was isolated from tonsils of healthy pigs collected at slaughterhouses, confirming that this organism can be part of the normal mucosal flora in healthy pigs. **AHL continues to monitor all porcine cases for the presence of *S. zooepidemicus*. Recovered isolates will be stored for future testing, including WGS of pathogenic strains, if required.** *AHL*

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2. Costa MO, Lage B. *Streptococcus equi* subsp. *zooepidemicus* associated with sudden death of swine in North America. bioRxiv. 2019; doi: http://dx.doi.org/10.1101/812636.

First confirmed case of porcine sapelovirus polioencephalomyelitis in Ontario

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AHL Newsletter 2020;24(1):10-11.

In December 2019, neurologic signs (predominately ataxia) were initially observed in a low number of pigs (~0.5%) upon entry to the nursery or within 1-4 weeks of placement. Disease was unresponsive to antibiotic therapy and progressed to death in all affected pigs within 24 to 48 hours of first signs. At its peak, the overall morbidity rate reached approximately 12% in this nursery, and while the majority of deaths occurred 2-4 weeks following placement, some deaths occurred as late as 5 to 7 weeks. Similar clinical signs were also identified in pigs in a second nursery barn sourced from the same closed sow herd. Two field postmortems were performed, and tissues were submitted to the AHL for histologic examination. The following day, 3 live pigs were brought to the AHL for euthanasia and immediate postmortem. Non-suppurative encephalomyelitis was identified histologically in both sets of pigs (**Fig. 1**). Inflammation and neuronal necrosis were concentrated in gray matter of midbrain, brainstem, and cervical spinal cord. Porcine hemagglutinating encephalomyelitis virus (PHEV), rabies virus, and bacteria were not detected in brain by PCR (viruses) or culture (bacteria).

Representative brain samples from 2 pigs were forwarded to the Iowa State University Veterinary Diagnostic Laboratory for porcine sapelovirus, porcine teschovirus, and porcine astrovirus 3 testing. Porcine sapelovirus DNA was detected by PCR in both brain samples, and porcine teschovirus nucleic acid was detected concurrently in the brain of 1 pig. Although the clinical syndrome seen in this case (low mortality) was not typical of the immediately notifiable "Teschen disease" form of teschovirus infection, detection of teschovirus necessitated reporting to the Canadian Food Inspection Agency (CFIA). Further investigation was required, and these sapelovirus and teschovirus isolates were sequenced by the CFIA.

Historically, porcine enteroviruses (serotypes 1 to 11) have been members of the genus Enterovirus in the *Picornaviridae* family. Recent reclassification has resulted in the generation of two new genuses, the Teschovirus genus (serotypes 1-7 and 11-13, now porcine teschovirus) and the Sapelovirus genus (serotype 8, now porcine sapelovirus A). Porcine enterovirus serotypes 9 and 10 (PEV-9 and PEV-10) remain in the Enterovirus genus. Enteric infection with these viruses is often acquired post-weaning, coinciding with waning maternal antibodies and mixing of animals; however, most infections remain asymptomatic. Infection with virulent strains may produce clinical neurologic disease in 2 typical forms: a severe and highly fatal form (Teschen disease), and a milder form (Talfan disease).

In recent years, similar cases of neuroinvasive sapelovirus infection have been reported in pigs in the United States and Europe. While entero-like viruses including sapeloviruses are commonly carried in the intestinal tract of healthy animals, factors precipitating spread to the central nervous system have yet to be elucidated. To date, sapelovirus-associated neurologic disease has not been reported in other species. In an acute outbreak of neuroinvasive sapelovirus infection reported elsewhere, all affected animals were from a single nursery source and were placed in two different finisher barns at 9 weeks of age. Over the following 3-week period, the onset and progression of clinical signs was variable in affected pigs, and included decreased feed/water consumption, dullness, ataxia, incoordination, paresis, and paralysis. Deep pain perception and withdrawal reflexes were maintained in hind limbs, and cranial nerve deficits were not observed. Histologic lesions were similar to those described in our case, and PCR testing for porcine enterovirus, porcine teschovirus and porcine sapelovirus consistently detected porcine sapelovirus. Overall, the reported morbidity and case fatality rates were 20% and 30%, respectively. These rates differ

from the Ontario cases, which had a lower morbidity rate (~12%) and higher case fatality (~100%). **In light of these recent findings, porcine sapelovirus and teschovirus encephalomyelitis should be included as differential diagnoses in cases of neurologic disease, particularly in nursery pigs.** *AHL*

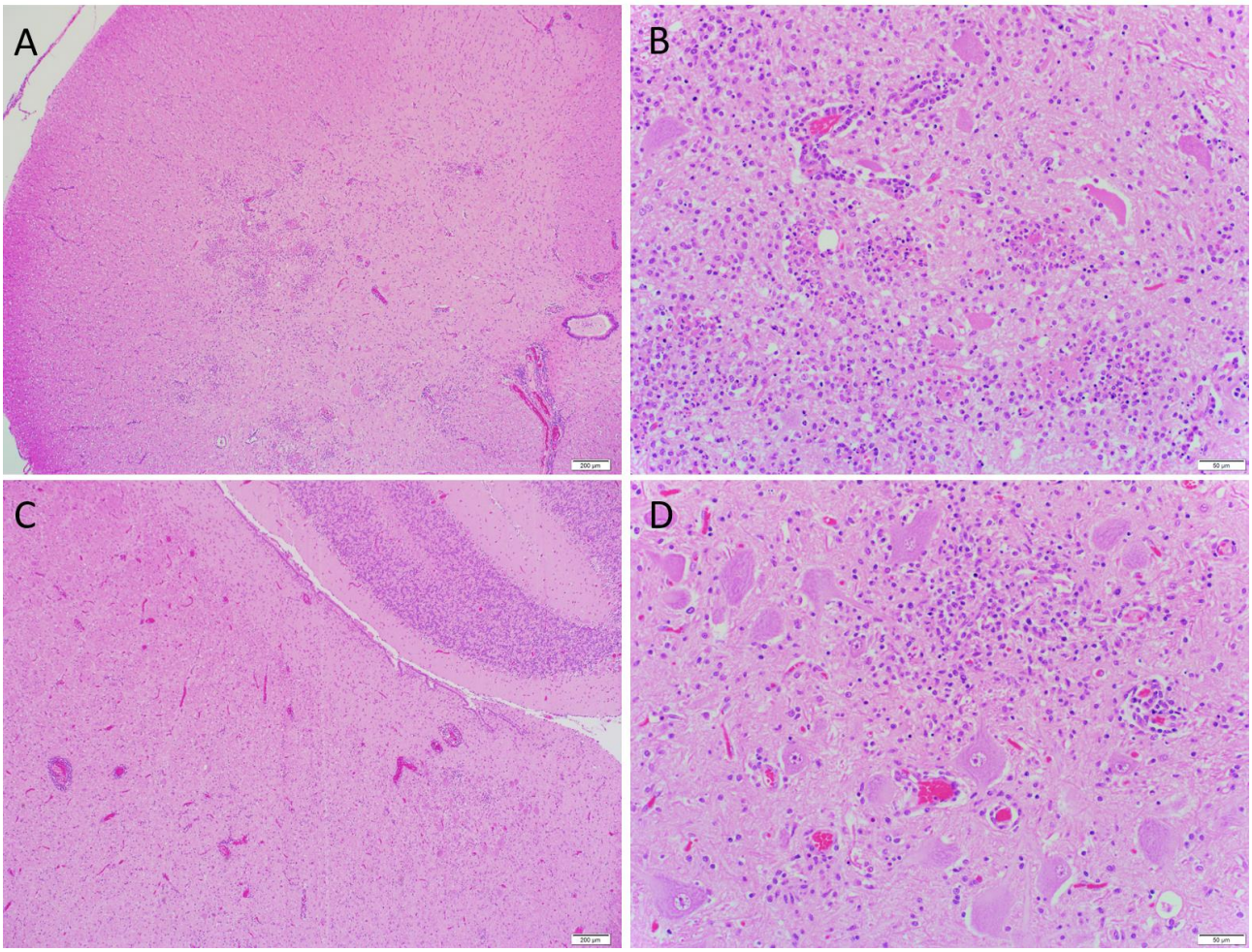

Figure 1. Porcine sapelovirus polioencephalomyelitis in nursery pigs. Spinal cord: Rarefaction of the grey matter accompanied by neuronal necrosis and inflammation (**Figures A & B**). Brain: Perivascular cuffing (**Figure C**) and a glial nodule (**Figure D**) in the grey matter of the brainstem.

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3. Arruda PHE et al. Identification of a divergent strain of sapelovirus associated with a severe polioencephalomyelitis outbreak in the US. Swine Health Information Center. https://www.swinehealth.org/sapelovirus/
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Avian reovirus variants and lameness in broilers

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AHL Newsletter 2020;24(1):12-13.

Avian reoviruses (ARV) are ubiquitous and often cause asymptomatic infections. Diseases associated with ARV infection are influenced by the virus pathotype, route of exposure, age and immune status. Certain ARV strains cause arthritis/tenosynovitis, respiratory disease, enteric disease, immunosuppression and malabsorption syndrome. Involvement of other infectious agents may also impact the nature and severity of reovirus-associated disease.

Prior to 2012, reovirus-associated leg issues occurred sporadically in Ontario broilers. However, in 2012-2013 and 2017-2018, outbreaks of reovirus-associated lameness resulted in significant morbidity and economic losses. Of 2,502 samples that were tested between 2012 and 2019 by real-time PCR, the majority (55%) were ARV-positive (**Table 1**).

Table 1. Results of avian reovirus PCR tests conducted at AHL, 2012-2019.

ARV PCR / Year	2012	2013	2014	2015	2016	2017	2018	2019
# Tested	255	243	154	145	297	554	480	374
Inconclusive			1	12	20	28	40	9
Negative	104	89	63	98	172	267	165	52
Positive	151	154	90	35	105	259	275	313
% Positive	*59.22%*	*63.37%*	*58.44%*	*24.14%*	*35.35%*	*46.75%*	*57.29%*	*83.69%*

Clinical presentation and intensity of histologic lesions varied and often appeared to be strain dependent, with cull rates ranging from 2% to 50%. In extreme cases, entire flocks had to be depopulated. Genotyping results determined that at least ten "variant" ARV strains were involved in cases with lameness issues in broilers. During the first outbreak in 2012, most variants were *Variant A* and *Variant B.* Since 2016 however, *Variant D* has been most frequently detected in affected birds, and it appears that introduction of genetically different viruses is still occurring (**Table 2**). Unfortunately, the source of introduction is unknown. *AHL*

Table 2. Avian reovirus genotypes identified at AHL, 2012-2019.

ARV Genotype / Year	**2012**	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**
Genotype 1/AR 95742 2012						1	1	
Genotype 4	1							2
Genotype 6 (SK-R12)	1				1	1		5
KR K738 2014					2	1	4	2
Not typed	1				1	1	4	2
ON classic 10-076656					10	1	2	6
ON classic 10-077184					4	2	4	4

ON classic 10-078957					3			1
S1133								1
SK-R38		2						
US AVS-B RSS	1				2	3	4	1
Variant A	16				17	2	8	21
Variant B	18	1						1
Variant C					19	5	2	4
Variant D					5	91	53	31
Variant E	4				5	2	12	2
Variant F	1				1	1	2	4
Variant G						1	3	2
Variant H							13	13
Variant I							3	4
Variant J						1	3	1

Spontaneous squamous cell carcinoma in the cloacal scent gland from a Mexican Black Kingsnake

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AHL Newsletter, 2020;24(1):13-14.

A male Mexican Black Kingsnake (*Lampropeltis getula nigrita*) presented with re-occurring swelling and inflammation around the cloacal and scent gland region. One month previously, the snake was treated with ceftazadime for a suspected scent gland infection. The infection appeared to respond to antibiotic therapy, but the swelling never completely resolved. On representation, the scent gland region was severely inflamed with thick purulent discharge (**Fig. 1A**). Additionally, two new small firm subcutaneous masses had formed on the snake's left lateral region, cranial to the cloaca. Upon surgical exploration of the scent gland abscess, multiple abnormal, distinct circular soft tissue masses were identified underlying the purulent discharge. These masses seemed to coalesce caudally, appeared to invade the surrounding musculature around the spine and effaced the left hemipene cranially. Samples for both histopathology and aerobic bacterial culture were obtained.

The histopathology results revealed squamous cell carcinoma (SCC) with local tissue invasion and associated scirrhous response (**Fig. 1B-1D**). Bacterial culture grew *Proteus mirabilis*. It is believed that the SCC originated from the left scent gland with a concomitant secondary bacterial infection. Additionally, it is suspected that masses located cranial to the initial site were secondary abscesses rather than regionalized tumor metastasis. Seven days post-operatively, the primary scent gland site had dehisced while the secondary sites remained intact.

Commonly affected anatomic sites for SCC in snakes include the oral cavity, cloacal skin, cloacal scent glands, and hemipenes. Similar to other species, SCC is usually locally aggressive and destructive. Clinical deterioration due to local disease usually occurs before metastasis to internal organ which is uncommon. Recent reports described the development of oropharyngeal SCC in snakes associated with infection by a novel alphaherpesvirus, Opheodrys herpesvirus 1. Additionally, oral SCC often occurs with concurrent stomatitis, and chronic inflammation may play a role in neoplastic transformation. This highlights the importance of further work up and histopathology for masses noted around a snake's oral and cloacal regions, especially if an initial abscess is suspected. *AHL*

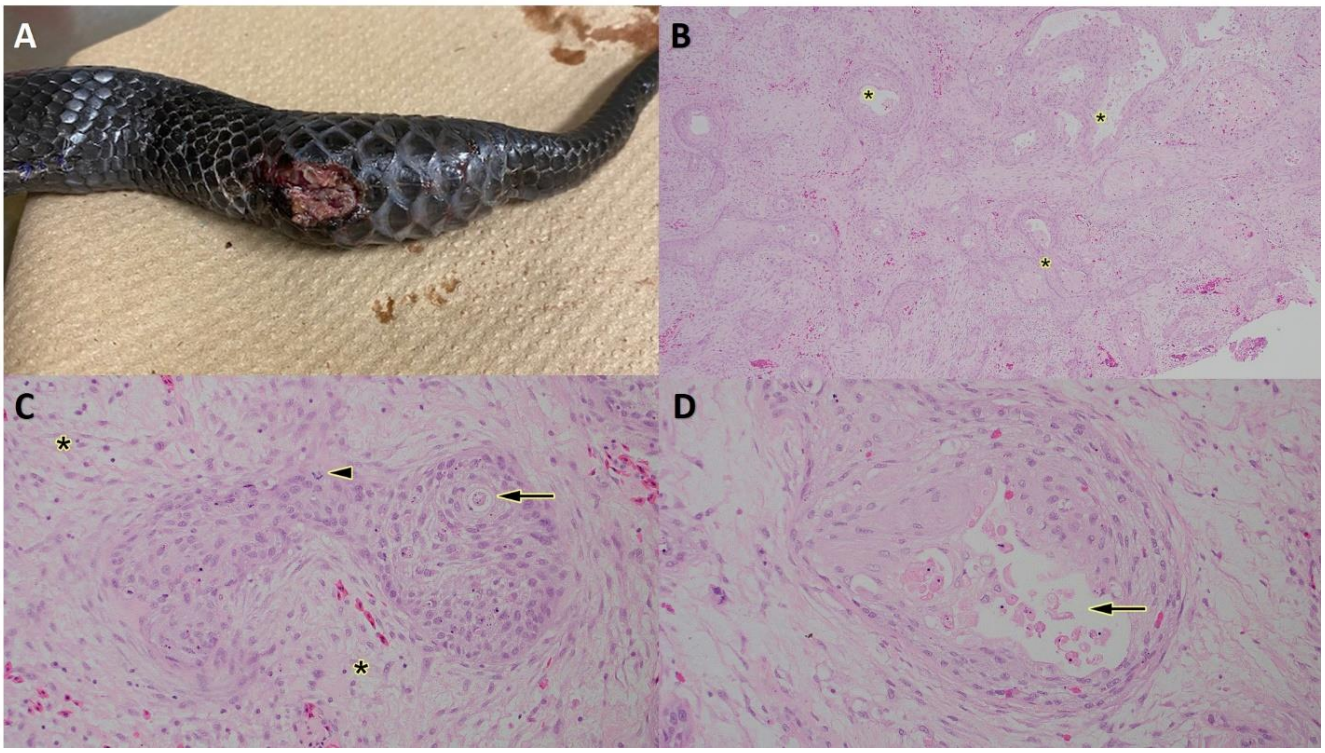

Figure 1. Scent gland squamous cell carcinoma (SCC). **A.** Peri-cloacal swelling with purulent debris extending from the left scent gland. **B.** Infiltrating anastomosing cords and islands of polygonal squamous epithelial cells (asterisks). **C & D**. Islands are separated by interspersed desmoplastic stroma (asterisks), often keratinize centrally (arrow), and contain scattered mitotic figures (arrow head).

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Diagnosing neurologic disease in backyard chickens

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AHL Newsletter, 2020;24(1):15-17.

Veterinarians who treat backyard flocks can encounter cases of neurologic disease due to a number of different etiologies. Neurologic signs can include tremors, circling, ataxia, twisting of the head and neck, falling over backwards, and paralysis.

Morbidity and mortality will be variable. How do you approach diagnosing neurologic disease in these flocks?

On **physical examination,** you can observe how the bird is moving. Determine whether it can stand and if so, whether it can walk. If the bird can only sit or lie down, observe how it is positioned. Is it hock sitting with both legs forward and the feet slightly off the ground (kinky back/spondylolisthesis)? Does it have one leg forward and one leg back (Marek's disease, **Fig. 1**)? Is it lying on its side? Different positions can give you clues to the etiology. Also note if the bird is bright, alert and responsive. Determine if the neck muscles have good or flaccid tone (botulism). Look at the eyes and note if they are cloudy or if the bird is squinting and the corneas are irregular (ammonia burn). If there are multiple birds affected, determine if they are showing similar neurologic signs.

If there is **no mortality,** a thorough history will be important. Determine if the bird is outside or indoors, what the bird is being fed (commercially available or home-made feed, scraps; access to toxic plants) and if the bird has had any vaccinations.
If there is **mortality,** this is an opportunity for postmortem examination and more intensive sampling. Postmortem examination could be done at a clinic or birds could be shipped to the AHL. If birds are shipped, please refer to the AHL website for a PM submission form:
https://www.uoguelph.ca/ahl/submissions/submission-forms
and packaging instructions:
https://www.uoguelph.ca/ahl/submissions/ahl-labnote-27-submission-instructions

Clinic Postmortem: On postmortem examination, examine the bird for any signs of trauma or puncture wounds (predation). Examine the skin for any raised nodules surrounding feather follicles (**Fig. 2**). Cut the skin between the breast muscle and leg and retract the leg laterally. Identify the triangular muscle along the medial thigh. Cut this muscle caudal to cranial next to the body wall and reflect it cranially. This will expose the femoral artery and sciatic nerve that can be collected for histology (**Fig. 3**). When the body cavity is opened and the breast is reflected cranially, the brachial plexus can be identified and collected for histology. **Collecting peripheral nerve for histology is critical for diagnosing Marek's disease** (**Fig. 4**). Remove the skull cap and carefully remove the brain, including the brain stem and cerebellum. Examine the brain for any discolouration or nodules that could indicate bacterial or fungal infection. If the cerebellum is bright red in young birds, there is possible vitamin E deficiency. One half of the brain can be collected for histology. The cerebellum is needed to detect *Baylisacaris procyonis* migration (**Fig. 5**) in cases where birds have exposure to raccoon feces. The cerebellum and brain stem are needed to diagnose avian encephalomyelitis (**Fig. 6**) in young birds. The remaining half of the brain can be collected for culture or PCR testing. (**Table 1**).

Examine the internal organs and determine if there are any nodular lesions (Marek's, other tumors; **Fig. 7**) that can be collected for histology. Examine the crop and gizzard contents to determine the type of feed or identify any foreign bodies that could be toxic. Once your examination of organs is complete, you can remove the soft tissues and cut longitudinally down the middle of the spinal column. Examine the area around the junction of the lungs and kidney to determine if there is developmental spinal compression

(kinky back/spondylolisthesis) or if there is a vertebral abscess causing impingement of the spinal column (osteomyelitis).
For more information on neurologic diseases in chickens, please refer to the OAHN website: https://oahn.ca/resources/neurologic-diseases-of-small-flock-poultry/ and https://oahn.ca/resources/small-flock-poultry-veterinary-resources/

Table 1: List of chicken neurologic disease tests available at AHL

DISEASE (by lab section)	PCR	ELISA	CULTURE	OTHER
Parasitology				
Baylisascaris procyonis				Histology (brain including cerebellum)
Virology				
Newcastle/APMV-1 (Avian Paramyxovirus 1)	X	X		
Influenza A/avian influenza	X	X		
Avian encephalomyelitis (young birds)	X			Histology (brain including cerebellum and brain stem)
Marek's disease				Histology, PCR (spleen, send out test)
Bacteriology				
Bacterial culture			X	
Mycology/Fungal culture			X	
Botulism				Mouse inoculation test
Toxicology				
Mycotoxin (feed analysis)		X		

Sampling summary:
NOTE: If screening a flock for disease, up to 5 swabs or tissues can be pooled for PCR testing. PCR tests are generally preferred as they target specific diseases and can be done quickly.
Live bird diagnostics:

- Choanal slit/tracheal swab (use viral transport medium) for PCR. PCR is a fast test and screens for multiple diseases.
  You can watch a video on how to collect samples with these swabs at: https://oahn.ca/resources/small-flock-poultry-veterinary-resources/
- Photos of affected birds can be submitted to AHL along with the case history.

Deceased bird diagnostics:
NOTE: If a postmortem is refused by the owner, swabs from the choanal slit could still be collected for PCR or bacterial culture.

- Postmortem: key test for lesion identification and sample collection.
- Histology: an option to screen tissues for lesions or determine the etiology of specific lesions such as nodular structures (bacterial, fungal, neoplastic). Collect a wide variety of tissues to place in formalin including brain, peripheral nerve, and all major organs: lung, liver, spleen, and kidney.
- PCR tests can be run on swabs or tissues. If you are sending tissues, package each tissue type separately and label the bag. Do not mix intestinal tissue with organ tissue.
- Bacterial/fungal culture can be run on gel swabs or tissues. Culture will screen for multiple bacterial organisms and the MALDI-TOF system is used for precise identification.

Reportable diseases in Canada include *Salmonella* Pullorum, *Salmonella* Gallinarum, influenza A and Newcastle disease. If high mortality and/or clinical signs lead you to suspect (avian) influenza A or Newcastle disease – quarantine the flock and phone CFIA! *AHL*

Figure 1. Abnormal leg position (one forward, one back) suggestive of Marek's disease (AAAP Slide Set #26)

Figure 2. Swelling of individual feather follicles due to lymphoid infiltration (Marek's)

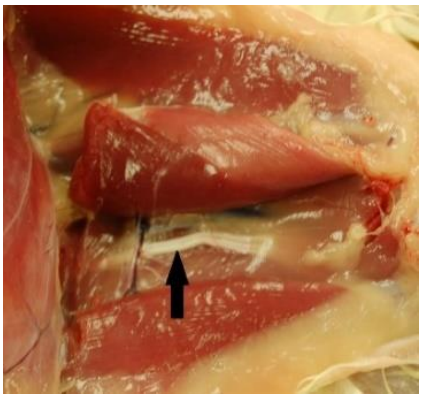

Figure 3. Sciatic nerve location (black arrow)

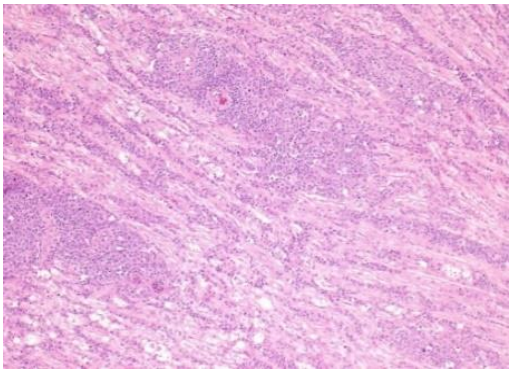

Figure 4. Lymphoid infiltration of a peripheral nerve is key to histologic diagnosis of Marek's (H&E)

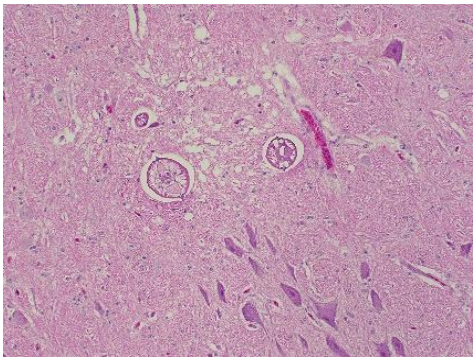

Figure 5. *Baylisascaris procyonis* larval migration in brain

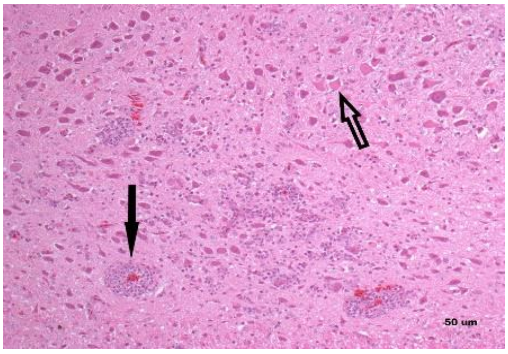

Figure 6. Avian encephalomyelitis brain lesions: perivascular inflammation (solid black arrow); neuronal degeneration (open black arrow)

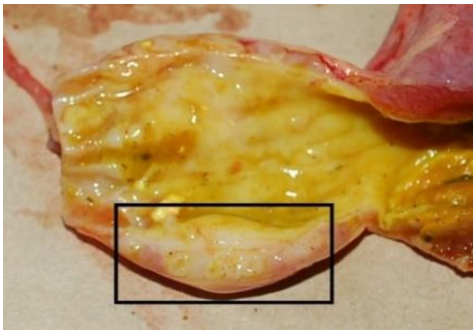

Figure 7. Marek's disease: neoplastic lymphocytic infiltrates within the wall of proventriculus (black box)

Photos courtesy of Dr. Marina Brash and Dr. Emily Martin

Stranger things: those weird bacterial names you may see in your laboratory report

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AHL Newsletter 2020;24(1):18.

Having trouble with some of those strange-sounding bacterial species on your laboratory report? Can't remember if *Terrisporobacter glycolicus* is a bacteria or fungus? You are not alone. With more precise bacterial identification available through automated devices such as the MALDI-TOF used at the AHL, new taxonomy classification schemes by microbiologists, better bacterial transport and isolation techniques, as well as PCR techniques such as 16S ribosomal RNA, a whole brave new world of bacteria (or at least bacterial names!) is being unleashed upon the unsuspecting clinician and pathologist. Between April 1st and June 30th 2019, approximately 211 different bacterial species were reported on equine cases submitted for culture at the AHL. The most common isolates are well known; however, many others are not. Interpreting the significance of bacterial isolates depends on the clinical picture and the site associated with the sample, as always.

Bacteria that are listed in a bacteriology report will either have at least one report in the literature of involvement in disease (not necessarily in horses) and/or may be reported, depending on the clinical history, if they are one of the dominant isolates taken from a sterile site. We thought it may be useful to highlight a few of these bacteria. To qualify, they had to be reported in more than 4 equine cases in the last quarter and be something that a pathologist would have to ask about (or Google!).

Nicoletella semolina: 6 isolates were reported from 5 cases. All were from guttural pouch, trachea, bronchoalveolar lavage fluid (BAL) or from a submandibular swelling. This Gram-negative bacillus is a member of the Pasteurellaceae family and is usually isolated from horses with respiratory disease. However, this does not necessarily mean it is the cause of the disease, as this bacterium can also be isolated from respiratory tract of clinically healthy horses.

Streptococcus thoraltensis: 4 isolates from 3 cases. All were from guttural pouch or pharyngeal mucosa. It has been described in humans with fever of unknown origin and isolated from the oral cavity; also from porcine vaginal and intestinal samples.

Staphylococcus vitulinus: 5 isolates from 5 cases. This bacteria is a coagulase negative *Staphylococcus* species that is isolated from human clinical samples (urine), as well as from calves, horses, fish and meat products. *AHL*

Dermatophilus congolensis infection in a horse: Don't forget the crust!!

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AHL Newsletter 2020;24(1):19-20.

A 20-year-old horse presented to the referring veterinarian with multiple, coalescing, raised, crusty, lesions along the topline, extending from the withers to the tail. Removal of scabs revealed areas of depigmentation and serous discharge. *Dermatophilus congolensis* infection was clinically suspected and multiple punch biopsies were submitted for histopathology, along with impression smears, and intact crusts for cytological evaluation. A crust with hair was also submitted for culture.
Histology revealed evidence of chronic eosinophilic dermatitis, and secondary pyoderma with epidermal hyperplasia and limited serocellular crusting (**Fig. 1A**). Thick laminated crusts typical of *D. congolensis* (**Fig. 1B**) were not captured in histologic sections of this case, and organisms were not identified. Cytology revealed evidence of marked suppurative inflammation along with limited numbers of eosinophils. Intracellular small coccoid bacteria (suspected to represent secondary bacterial infection) were identified, along with several filamentous strands comprised of parallel rows of cocci ("railroad tracks"), consistent with *D. congolensis* (**Fig. C**). Dermatophilosis was also confirmed by bacterial culture.

D. congolensis is a Gram-positive, non-acid-fast, facultative anaerobic actinomycete with a wide host range and worldwide distribution. The natural habitat for *Dermatophilus* is unknown, although organic matter is thought to have a protective effect on the organisms, and soil may act as a temporary reservoir. *Dermatophilus* can also survive in the skin of clinically normal animals, possibly acting as a source of infection once favourable conditions are present and they become activated to become motile zoospores. *D. congolensis* has a distinctive life cycle in which coccoid bodies germinate to produce branching filaments. Establishment of infection depends upon a number of factors, including the virulence of the strain, the general health of the animal, skin trauma and moisture. Moisture may act to dissolve the surface lipid film on skin and to soften the stratum corneum. Trauma from ectoparasites, skin scratches, or other injuries may serve as portals of entry. External parasites may also act as mechanical vectors. Resistance in some animals may have a genetic component.

Once the skin surface has been disrupted, and activated zoospores gain access to the epidermis, infection can develop. The zoospores germinate to form mycelia which invade the viable epidermis and outer root sheaths of hair follicles. Bacterial invasion may be related to the production of exoenzymes. As the filaments invade the epidermis, keratinocytes at these sites begin to cornify and large numbers of neutrophils migrate into the area, causing separation of the epidermis from the underlying dermis. Repeated sequences of bacterial invasion, inflammation, and epidermal regeneration produce the thick crusts characteristic of dermatophilosis. It is unknown why some animals develop mild, localized disease which resolves rapidly and spontaneously whereas others develop chronic, widespread, debilitating disease. Commensal skin bacteria, primarily *Bacillus* spp., have been shown to produce substances which inhibit the growth *of D. congolensis* in culture. A similar effect by bacteria normally present on the skin may occur in vivo.

The clinical presentation of *D. congolensis* is fairly characteristic. The earliest lesions consist of patches of slight erythema which are most visible in unpigmented areas. Papules form next and mature into pustules. Lesions become exudative and matted with hair, forming thick crusts. Removal of crusts reveals areas of eroded, painful and exudative skin when active lesions are present and dry crusts with diffuse scaling and alopecia when chronic lesions are present. Lesion distribution varies by species. In horses, the

dorsal thorax, rump and face are usually affected. Occasionally, lesions may be noted primarily on the distal limbs.

Differential diagnoses for the lesions induced by *D.congolensis* include dermatophytosis, staphylococcal dermatitis, mite infestation, various viral infections, zinc-responsive dermatosis and pemphigus foliaceus. Diagnosis of *D. congolensis* is based on clinical history, physical examination findings, with demonstration of the organism by cytology, histology, or culture. *D. congolensis* grows readily on blood agar. **In this particular case, the diagnosis would have been missed if crusts had not been submitted along with biopsy samples.** Thus, it is important to remember that crusted lesions should be selected for biopsies and care must be taken to ensure that crusts are included for histologic evaluation, as this increased the chances of capturing the organism. In addition, impression smears of a crust for cytological evaluation can be helpful in demonstrating the organisms. As crusts may be dry in more chronic infections, mixing a crust with a few drops of sterile saline and spreading the material onto a glass slide may be necessary. Heat fixation is not necessary and is contraindicated. All that is required for submission to the laboratory are air dried, well-spread unstained slides for cytological evaluation. *AHL*

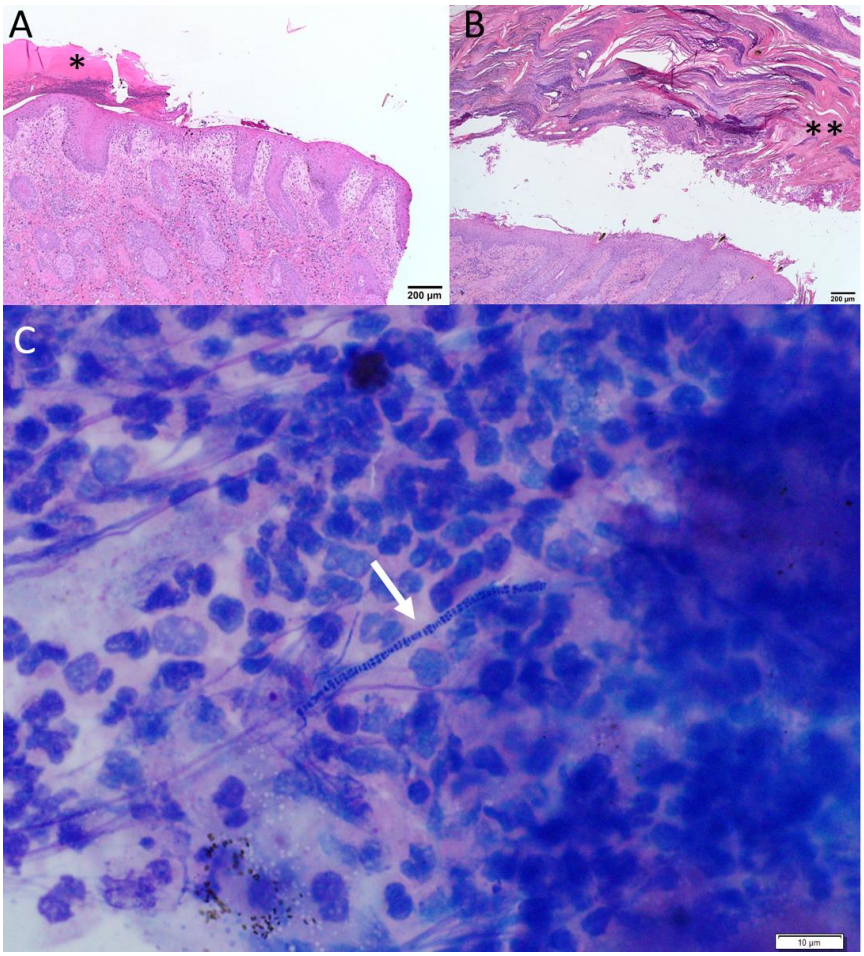

Figure 1. *Dermatophilus congolensis* infection in a horse. **A.** Skin biopsy from this case capturing epidermal hyperplasia with limited serocellular crusting lining the surface (*). **B.** Biopsy from a different case, capturing similar epidermal hyperplasia overlain by a thick crust comprised of laminated keratin (**) enmeshed with suppurative exudate. **C.** Cytologic smear containing filamentous strands of paired cocci consistent with *D. congolensis* (arrow) in a background of inflammatory cells.

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Alcohol and Gaming Commission of Ontario (AGCO) Death Registry / Equine Incidences in Ontario Racing program: 2003-2019 postmortem summary

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AHL Newsletter 2020:24(1):21-22.

The Alcohol and Gaming Commission of Ontario (AGCO; previously 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 ORC Death Registry (DR, 2003-2016) and the AGCO 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. To date, PM has been carried out on 1172 horses through these programs (**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 significant PM findings is provided in **Table 2**. A comprehensive review of AGCO PM cases was recently conducted as part of a separate retrospective study[1]. As a result of this study, some cases have been reclassified from data presented in previous editions of the AHL Newsletter. Since 2015, **computed tomography (CT) of fractured and contralateral limbs** has been carried out on select DR and EIOR 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. The protocol was continued in 2019, with CT imaging of 26/ 27 limb fracture cases submitted for PM exam. **Pre-existent lesions in bone were identified by CT and considered potentially predisposing to fracture in 11 of 26 (42%) cases.**

Exercise-associated sudden death continues to be of special concern in the racing industry. At the AHL, a modified in-depth PM protocol is used in the evaluation of these cases, with special emphasis on cardiovascular and respiratory systems. In 2019, the cause of death was investigated in 10 horses that died while exercising. Significant pulmonary hemorrhage was evident in 5 horses; aortic rupture was identified in 2 horses; cervical vertebral fracture was present in 1 horse; and no cause of death was identified in 2 horses. Among all sudden death cases from 2003-2019, significant pulmonary hemorrhage was identified in 93/ 187 (50%) horses. The term 'equine exercise-associated fatal pulmonary hemorrhage' (EAFPH) is currently used to categorize these cases, in which extensive pulmonary hemorrhage and edema is identified postmortem. In 43/ 187 (23%) exercise-associated sudden death cases from 2003-2019, no potentially fatal lesions were identified and the cause of death remained undetermined. 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 in these animals.[2] Typically, no morphologic lesions are detected in heart as a cause or result of fatal ventricular dysrhythmia, and the diagnosis cannot be confirmed based on PM findings.

Summaries of postmortem submissions to the Animal Health Laboratory under this program and diagnoses by body system for these cases are provided in the following tables. *AHL*

Table 1. Breed distribution of ORC Death Registry / AGCO EIOR submissions to the AHL, 2003-2019.

Breed / year	Standardbred	Thoroughbred	Quarter Horse	Total
2003	63	59	0	**122**
2004	81	60	0	**141**
2005	59	51	0	**110**
2006	58	46	2	**106**
2007	66	53	3	**122**
2008	27	24	0	**51**
2009	28	16	1	**45**
2010	22	8	2	**32**
2011	24	18	4	**46**
2012	20	14	0	**34**
2013	19	26	2	**47**
2014	21	22	8	**51**
2015	29	24	3	**56**
2016	15	32	3	**50**
2017	26	34	2	**62**
2018	16	33	1	**50**
2019	12	35	0	**47**
Total	**586**	**555**	**31**	**1172**

Table 2. Significant postmortem lesions identified in ORC Death Registry / AGCO EIOR submissions by body system, 2003-2019.

Diagnoses by body system:	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019
Fracture / limbs	51	69	48	43	58	16	3	7	5	2	22	23	25	27	34	29	27
Fracture / other	10	4	6	11	8	5	0	3	6	2	2	8	4	4	1	1	4
Non-fracture musculoskeletal	8	7	8	7	5	4	4	2	0	0	2	3	4	1	6	4	1
Gastrointestinal	16	22	17	16	18	4	4	6	5	6	4	6	5	5	5	2	2
Respiratory	17	12	5	4	11	6	15	7	9	7	4	6	4	3	6	5	5
(including pulm hemorrhage)																	
Cardiovascular	5	6	3	6	1	6	2	2	2	1	5	2	0	2	2	0	2
CNS	3	7	8	4	0	1	2	0	6	2	3	0	2	2	0	4	1
Renal	0	1	0	0	2	0	0	0	0	0	0	0	0	0	0	0	0
Other / whole body conditions (eg. septicemia)	2	0	6	3	5	2	4	2	5	4	3	2	6	3	4	1	2
Injection-associated	2	6	3	5	3	2	5	1	5	5	1	0	3	0	1	1	0
Cause of death undetermined	8	7	6	7	11	5	6	2	3	5	1	1	3	3	3	3	3
Total	**122**	**141**	**110**	**106**	**122**	**51**	**45**	**32**	**46**	**34**	**47**	**51**	**56**	**50**	**62**	**50**	**47**

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Salmonella Dublin septicemia in a puppy

Murray Hazlett, Patricia Lechten, Đurđa Slavić

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AHL Newsletter 2020;24(1):23.

A 12-week-old French Bulldog was presented to the veterinary clinic November 6th for vomiting and poor appetite. Hematologic (complete blood count) and biochemical results were unremarkable. The puppy was treated symptomatically with some improvement. Two weeks later, he returned vomiting and emaciated. Blood was present in the feces, but there was no diarrhea. CBC and serum biochemistry were again unremarkable. Radiographs were normal. Ultrasound showed a thickened ileum and viral enteritis was suspected. The puppy would eat if force fed. Blood cleared from stool. The puppy died a week later and was submitted for postmortem examination.

Postmortem results were mild diffuse pulmonary edema and congestion. No lesions were seen involving heart, liver, thyroids, thymus, spleen, or adrenals. Kidneys were pale with occasional non-raised red foci on the surface. The stomach was empty with small amounts of mucoid liquid tan content in lower small intestine and colon, and acute enteritis was suspected.

Histologically, there was evidence of septicemia with intravascular bacteria present in most organs. Foci of necrosis were present in the spleen, there was acute neutrophilic interstitial nephritis **(Fig. 1A and 1B)** and focal meningitis. Occasional dilated crypts were present in the intestine. Colonic culture revealed 4+ *Salmonella* Dublin with no other enteric pathogens present (testing was done for *Yersinia* spp., *Campylobacter* spp., parvovirus and coronavirus).

Salmonella Dublin is a cattle-adapted serotype of *Salmonella enterica*. It is frequently identified at the AHL where it is seen as an emerging problem in Ontario cattle, causing acute septicemia.
Like most *Salmonella* spp, *Salmonella* Dublin is zoonotic. The Centers for Disease Control and Prevention reported 3,903 cases of human *S.* Dublin infections between 1968 and 2013. It is much more commonly recovered from blood (61%) than other *Salmonella* spp. (5%). Humans with the disease required hospitalization more frequently (75%) than other Salmonella infections (27%). Some outbreaks in humans have occurred from foodborne sources, such as raw beef, raw milk, and cheese.

This puppy was from a kennel where raw meat is occasionally fed. The source of the meat is unknown, but we suspect it may have been the source of the *S.* Dublin in this case. We are not aware of *Salmonella* Dublin being reported in dogs before. No other dogs or people in the household appeared to be affected.
AHL

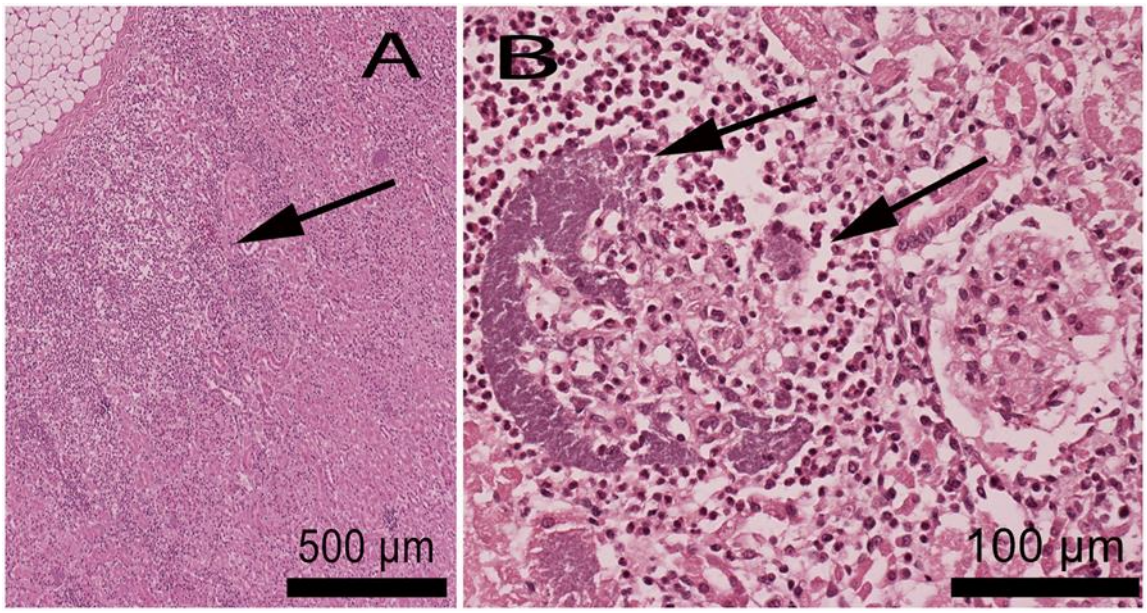

Figure 1A. Neutrophilic inflammation (arrow) in the renal cortex associated with *Salmonella* Dublin.
Figure 1B. Close-up of a glomerulus with bacterial colonies (arrow).

Reference

1. Harvey et al. Epidemiology of *Salmonella enterica* serotype Dublin infections among humans, United States, 1968–2013. Emerg Infect Dis 2017:23(9):1493-1501.