Laboratory Services Division
Animal Health Laboratory
University of Guelph
Changing Lives Improving Life

AHL Newsletter

Canada Post Publications number - 40064673

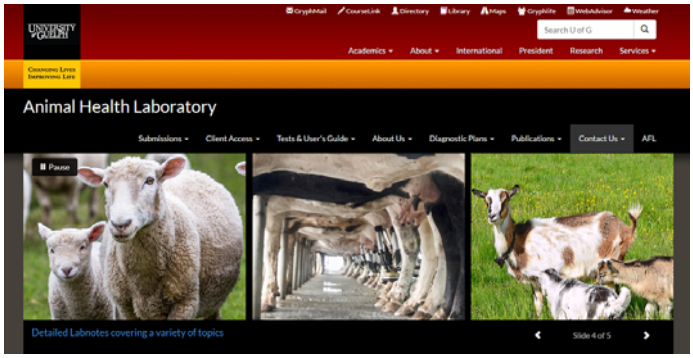
Volume 21, Number 1, page 1 **March, 2017** ISSN 1481-7179

In this issue:

AHL has an improved website!

Jim Fairles, Josie Given

⇒ Same **url**: www.ahl.uoguelph.ca
⇒ Most documents are **html**.
⇒ **User's Guide and Fee Schdule** are readily accessible from mobile devices.
⇒ Please email ahlinfo@uoguelph.ca if you do not have the **username and password** for the Fee Schedule – this is only available to veterinarians.
⇒ **Diagnostic Plans** coming soon!



Bacterial and viral transport media *Jim Fairles*

One of the most important aspects of laboratory work is the actual sampling and handling of the samples prior to being received at the laboratory.

Bacteriology and Mycoplasmology - AHL prefers a regular gel-based bacteriology swab.

Virology - AHL prefers virus transport medium (VTM) swabs for virology PCR tests. For some testing (PEDV), a fecal swab in VTM is preferred rather than feces. **Regular gel-based bacteriology swabs are unsuitable for PCR testing**. Dry swabs moistened with sterile saline in a sterile container can be used, but **VTM is preferred**.

More details on p. 11 ⟶

Carl Block Award to Dr. Grant Maxie

The Canadian Animal Health Coalition (CAHC) has named Dr. Grant Maxie, Director of the Animal Health Laboratory, University of Guelph, as the 2016 recipient of the Carl Block Award. Through his hard work and dedication, Dr. Maxie has made many significant contributions to the Canadian animal health industry.

Each year the CAHC presents the Carl Block Award to an individual nominated by his or her peers for outstanding contributions in the field of livestock animal health. This award is in memory of Carl Block, who was chair of the CAHC when he passed away as the result of a small plane crash in May 2002. "I accept the award on behalf of the great team of people that I work with in the Animal Health Laboratory and Laboratory Services Division. It's easy to look good when you're surrounded by excellent colleagues," says Dr. Maxie.

From the OVC Bulletin: http://bulletin.ovc.uoguelph.ca/post/154092010625/dr-grant-maxie-receives-2016-carl-block-award

Selected AHL Ontario outreach activities, 2016

AHL newsletter - 39 scientific articles in 2016.

Barham M. Ontario Animal Health Network: A collaborative approach to poultry surveillance. Poultry Health Research Network, Seminar Series. PAHL. U of Guelph, Guelph, ON. May 17, 2016.

Brash ML. Small flock poultry project, sample collection demonstration Small flock poultry workshop for veterinarians. OAHN/ OMAFRA/AHL event. PAHL, U of Guelph, Guelph, ON. May 7, 2016.

Brash ML. Platinum Brooding Ontario Workshops. Cargill Poultry Producers. Kitchener, ON. Feb 9/10,June 8/ 9, 2016.

Brooks AS, Brash ML, Ouckama R, Varga C, **Barham ML.** Poultry health and OAHN update. Poultry Industry Council, St. Isidore, ON. Dec 1, 2016.

Brooks AS. Diseases of sheep. Ontario Sheep Marketing Agency, District 10, Embrun, ON. Mar 2016.

Fairles J. AHL Update – overview of AHL updates and disease trends (OAHN) for 2016. Ontario Association of Swine Veterinarians, Fall Conference, October 29, 2016.

Hazlett MJ. Food animal diagnostic pathology – diseases of swine. 4th year OVC DVM students. Dec, 2016.

Maxie G. AHL laboratory overview. OMAFRA Ministry Emergency Management Planning Committee. Guelph, ON. Jun 20, 2016.

McEwen B. Animal Health Laboratory - Our role in medicolegal cases. Private Security & Investigative Services Branch Meeting. Ministry of Community Safety & Correctional Services. Toronto ON. Dec 2016.

Oesterle P, **Ojkic D,** Buchanan T, Purvis M, Shirose L, Campbell D, Jardine C. Avian influenza in wild birds - 2015 surveillance results from Ontario. Interntnl Conf Diseases in Nature Communicable to Man. Guelph, ON, August 7, 2016.

Ruotsalo K, DeLay JP. 'Opening the Black Box,' maximizing the benefits of diagnostic testing. OAVT Conference. March 2016 .

Ruotsalo K, DeLay JP. Round table discussion Lunch with a pathologist. OVMA Conference. Toronto, ON. Jan 2016.

Spinato, MT, Maxie, MG. The Animal Health Laboratory's role in emergency response. Farm-to-Fork Emergency Management Exercise for Swine Disease. Guelph, ON. Nov 25, 2016.

Samman A, Susta L, Guerin M, **Brash M**, Varga C, **Martin E**. Detection and surveillance of significant pathogens in Ontario small poultry flocks. PHRN Research Day, U of Guelph, Guelph, ON. Mar 29, 2016.

Spinato MT. KTT project: Adult small ruminant mortality project launch. Small Ruminant Veterinarians of Ontario. Mar 31, 2016.

Turner PV, Compo NR, Davidson S, **McDowell M, Cai H**, Gottstein B, Peregrine AS. Diagnoses of alveolar echinococcosis in lemurs at an exotic animal sanctuary: Implications for public health. DIN Meeting 2016.

Vaillancourt J-P, Boerlin P, **Slavic D,** Guerin M. Assessment and mitigation of contamination risks: critical knowledge to reduce diseases and increase biosecurity compliance. OMAFRA Food Safety Research Forum, Guelph, ON, May 2016.

AHL Newsletter
March, 2017 - Volume 21, Number 1

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Editorial Assistants: **Helen Oliver, April Nejedly**

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.

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ISSN 1481-7179

Canada Post Publications number - 40064673

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Our continued thanks to all of the non-author AHL clerical, technical, and professional staff who contribute to the generation of results reported in the AHL Newsletter.

Selected zoonotic pathogens and diseases from Ontario identified at the AHL, 2016

Beverly McEwen, Durda Slavic, Davor Ojkic, Hugh Cai, Josepha DeLay, Margaret Stalker, Murray Hazlett, Kristiina Ruotsalo, Jan Shapiro, Andrew Brooks, Felipe Reggeti

Many new, emerging, and re-emerging diseases of people are caused by pathogens originating from animals, or are shared between people and animals. *Streptococcus iniae*, considered an emerging zoonotic pathogen, was isolated from aquarium cichlids; human infections generally occur through injuries associated with preparing whole fresh fish for cooking. (https://wwwnc.cdc.gov/eid/article/15/12/pdfs/09-0232.pdf) and have been reported in Ontario (https://www.cdc.gov/mmwr/preview/mmwrhtml/00043200.htm).

The AHL plays an important role in public health by identifying zoonotic pathogens in >1,000 cases annually (Tables 1 and 2). These are numerator data reliant upon submission biases to the diagnostic laboratory and cannot be regarded as population prevalence estimates. Monitoring programs are not included. *AHL*

Table 1. Number of cases with selected zoonotic pathogens isolated and/or identified at the AHL, 2016.

Agent	Bovine	Swine	Equine	Ovine	Caprine	Chicken	Turkey	Canine	Feline	Other	2016	2015	2014	2013	2012	2011
Ascarids (*T. canis, T. cati, T. leonina, Baylisascaris* sp.)	0	2	0	0	0	7	0	19	8	5	**41**	17	40	36	35	ND
Blastomyces dermatitidis	0	0	0	0	0	0	0	6	0	0	**6**	21	22	17	10	10
Bordetella bronchiseptica	0	19	5	1	0	0	1	2	1	3	**32**	37	28	24	33	43
***Borrelia burgdorferi* (Lyme disease), serology**	0	0	18	0	0	0	0	4	0	0	**22**	8	12	11	3	1
Brucella* sp. *(non-abortus)	0	0	0	2	0	0	0	1	0	0	**3**	0	1	0	0	0
Campylobacter coli/ jejuni/ fetus* subsp. *fetus	8	0	0	5	0	19	3	7	1	4	**47**	16	17	6	17	12
***Chlamydia* sp.**	0	0	0	8	5	0	0	0	0	0	**13**	24	15	25	33	39
Clostridium difficile	0	4	0	0	0	0	0	0	1	0	**5**	10	11	11	19	40
***Coxiella burnetii* (Q fever)**	2	0	0	28	7	0	0	0	0	0	**37**	44	55	28	36	99
***Cryptococcus* sp.**	0	0	0	0	0	0	0	1	0	0	**1**	1	3	2	1	
***Cryptosporidium* sp.**	210	3	0	2	16	0	0	0	1	11	**243**	247	186	206	141	147
Eastern equine encephalitis virus	0	0	0	0	0	0	0	0	0	0	**0**	6	25	1	0	5
***Giardia* sp.**	5	0	0	0	0	0	0	20	1	0	**26**	30	50	48	26	31
Listeria monocytogenes	9	0	0	3	10	1	0	1	0	0	**24**	12	23	15	18	18
Methicillin-resistant *Staphylococcus aureus* (MRSA)	0	0	8	0	0	0	0	2	1	1	**12**	28	17	8	24	49
Methicillin-resistant *S. pseudintermedius* (MRSP)	0	0	1	0	0	0	0	60	1	0	**62**	88	45	141	114	192
Rabies virus	0	0	0	0	0	0	0	0	0	2	**2**	0	0	0	0	0
Salmonella enterica	89	92	5	2	0	29	25	6	2	40	**290**	332	221	308	281	256
Streptococcus suis	47	126	0	0	3	1	0	1	0	3	**181**	167	105	126	144	106
Streptococcus equisimilis	3	32	15	0	1	0	0	4	0	2	**57**	48	4	34	45	59
Streptococcus zooepidemicus	2	0	146	1	1	0	0	2	2	0	**154**	138	93	112	4	149
***Toxoplasma* sp.**	0	0	0	4	2	0	0	0	0	1	**7**	11	18	11	8	24
Verotoxigenic *E.coli* (VTEC)	11	0	0	0	0	0	0	0	1	0	**12**	8	7	18		
West Nile virus	2	0	0	0	0	0	0	0	0	16	**18**	19	6	44	36	34
Yersinia enterocolitica	2	1	0	0	0	0	0	1	0	0	**4**	2	6	4	2	1
Total	**390**	**279**	**198**	**56**	**45**	**57**	**29**	**137**	**20**	**88**	**1,299**	**1,314**	**1,010**	**1,236**	**1,030**	**1,315**

Table 2. Leptospira spp. seropositive cases identified at the AHL, 2016.

***Leptospira* spp. serovar**	**Bovine**	**Swine**	**Equine**	**Canine**
L. autumnalis	12	1	15	67
L. bratislava	7	2	13	18
L .canicola	30	2	8	46
L. grippotyphosa	8	1	3	45
L. hardjo	21	1	1	5
L. icterohaemorrhagiae	39	2	9	61
L. pomona	22	1	7	27

Ontario Animal Health Network

"Your comprehensive source for animal health information."

OAHN Update March 2017

We have wrapped up the first year of our **OAHN projects**, with some networks planning to release results and reports in the coming months. Stay tuned for updates via your species group listservs and AGMs, as well as OAHN reports.

There have been several **new disease outbreaks** of interest in Canada, and we are following them closely. Follow us on Facebook and Twitter for news items every day as we collate the news for you in one place. Don't use social media? Check out animal health links of the week, posted weekly on our website.

New podcasts!

- OAHN's **Equine Lyme Project Update** with Memo Arroyo LMV, DVSc, PhD, DACVIM, Associate Professor, at the Ontario Veterinary College.
- 3-part series on **equine proliferative enteropathy (*Lawsonia*)** with Nathan Slovis, DVM, DACVIM, CHT (Haygard Medical Centre) and Dr. Arroyo.

Do you ask your clients for their Premises IDs? Check out how fast and easy it is to do by registering your vet clinic: https://www.ontarioppr.com/home_en.html

BEES

The Bee network has followed up its first report (released in the fall 2016) with a **2-part podcast** series outlining the network's OAHN-funded project and bee testing at the AHL.

SMALL RUMINANTS

The small ruminant network met in January, with reports to be released soon. The OMAFRA network co-lead, Dr. Jocelyn Jansen, has also spearheaded **captive bolt training** sessions for sheep and goat producers, held at the AHL.

BOVINE

The OAHN Bovine Network released its latest reports in February, discussing **mycotoxins**, diagnostic rates of **abortion** cases, and more.

FISH

The OAHN Fish Network has been discussing **whirling disease**, diagnosed in Western Canada, and the possible threat to Ontario. The network is currently working on its first infographic on the topic. Wondering what whirling disease is? Find out here.

SWINE

The swine network met in January, and its report was released in February. Network reports can be found here. Last quarter, nearly 300 clicked through the **producer report** and >40 read the **veterinary report**.

ALTERNATIVE SPECIES

On alternative species calls, we discuss interesting cases with experts, and we have a **listserv** to trade case and treatment ideas. Email oahn@uoguelph.ca to join.

POULTRY

The poultry network will hold its next call in March, and its latest survey was released on Feb 1st. The network has also held calls in response to **new strains of IBV**. You can check out the IBV Fact Sheet here.

Small flock corner:
OAHN held its first small flock conference call on September 14th. Interesting cases in backyard flocks were discussed, with veterinarians providing expert advice. You can still sign up for the Small Flock Listserv by emailing oahn@uoguelph.ca.

EQUINE

The equine's latest reports can be found here. The network lead and network member will be presenting at the OAEP AGM in February on the equine Lyme project results. **NEWS:** 1 EIA case was diagnosed in Quebec in early Jan– article here.

WILDLIFE

The CWHC Q4 report was published on OAHN in January. Find the report here. Through its OAHN project, the Wildlife network has launched a **citizen surveillance website** to report wildlife disease. Find it here: http://wildlifehealthtracker.com/

COMPANION ANIMALS

The OAHN companion animal network released its quarterly report in February, covering acute infectious respiratory disease, canine lungworm, rabies, and more. The Companion Animal Network also published a new infographic on "***Echinococcus multilocularis* in Ontario**," which you can access here. As well, OAHN published a new podcast on E. *multilocularis*, featuring an interview with Dr. Andrew Peregrine and master's student Jonathon Kotwa.

AHL Lab Reports

RUMINANTS

Streptococcus gallolyticus subsp. *pasteurianus* meningoencephalitis and septicemia in goats

Murray Hazlett, Emily Brouwer, Josepha DeLay, Amanda Mansz, Durda Slavic

Formerly belonging to *Streptococcus bovis* biotype II/2 complex, *Streptococcus gallolyticus* subsp. *pasteurianus* is associated with septicemia, meningitis, and endocarditis in humans. It is commonly found in the alimentary tract of ruminants and has been isolated from cases of septicemia in goslings, ducklings, and turkey poults. **We have recently seen 4 cases of acute suppurative meningoencephalitis and septicemia associated with this organism in young goats and would like to make practitioners aware of this.**

Case 1. A 2-mo-old goat from a farm with several kids with neurologic signs (opisthotonos, ataxia). Postmortem revealed severe neutrophilic meningitis and ventriculitis. *S. gallolyticus* subsp. *pasteurianus* was isolated in large numbers and almost pure culture from brain swabs. Moderate numbers were also isolated from lung along with *E. coli* and *Pseudomonas aeruginosa*. *Listeria monocytogenes* was isolated using enrichment procedures from 1 brain swab, however lesions seen were not typical for this organism. A diagnosis of suppurative meningoencephalitis and septicemia associated with *S. gallolyticus* was made.

Case 2. Two 2-mo-old goats had stiff joints and weak hind or front limbs. About 10% of kids were affected. The major findings in the first kid were severe suppurative arthritis and severe suppurative meningoencephalitis with occasional microabscesses in the perivascular neuropil. The second kid had only arthritis. *S. gallolyticus* subsp. *pasteurianus* (3+) was isolated from meninges of kid A as well as in pure culture (1+) in 2 of the affected joints and was considered the significant pathogen here. Tests for CAEV and *Listeria* were negative.

Case 3. Two 2-4-wk-old meat goats. The kids would go off feed, with some seeming neurologic and star-gazing. An autopsy was performed on the farm on 2 of the goats, and fixed tissues submitted for histology. Both goats had severe neutrophilic meningitis and evidence of septicemia. Although the animals had been treated, because of the similarity to the other cases, brain was submitted from both goats to look for *S. gallolyticus* subsp. *Pasteurianus*, which was isolated from the submitted brain sample in one goat (2+), along with *Staphylococcus aureus* (2+) and *Streptococcus pluranimalium* (3+). No significant pathogens were isolated from the second brain. This was interpreted as a possible case of *S. gallolyticus* because of its similarity to cases 1 and 2 (Fig 1).

Case 4 - 32-day-old goat kid displaying neurologic signs and reported as a "herd health problem". At autopsy there was severe suppurative meningoencephalitis – *S. gallolyticus* subsp. *pasteurianus* was isolated (3+) as well as occasional *P. aeruginosa*.

The clinical presentation and histology seen in these cases is not typical of listeriosis, which is the most common bacterial meningoencephalitis we see in ruminants (Table 1). Although cases of septicemic listeriosis can look similar, these are rare. We strongly suspect involvement of *S. gallolyticus* subsp. *pasteurianus* as the cause of septicemia and meningoencephalitis, sometimes with arthritis, in all 4 of these herds. Contaminants and treatment do cause some doubt about interpretation of bacteriology and possibly missed cases due to failure to recover the bacteria.

For an accurate diagnosis when sampling animals with evidence of meningitis, it is important to obtain a clean sample of the meninges or brain. This can be difficult, but if the brain is partially removed so that it is "hanging" from the skull, a sterile swab can usually be worked between the leptomeninges and dura without touching contaminated surfaces. The swab can be submitted for bacterial culture pending histology results. *AHL*

Table 1. Confirmed and suggested causes of meningitis/encephalitis in 87 goat pathology submissions at the AHL May 2007 to December 2016.

Etiology	Cases	Ages	Age mean
Listeria monocytogenes	37	1 wk – 5 y	20 mo
Caprine-arthritis encephalitis virus	6	2-48 mo	28 mo
S. gallolyticus subsp. *pasteurianus*	4	1-2 mo	1.5 mo
Corynebacterium pseudotuberculosis	2	12-36 mo	24 mo
Miscellaneous or unknown bacterial	12	1-36 mo	9.9 mo
Viral or non-specific	14	1-36 mo	12.8 mo

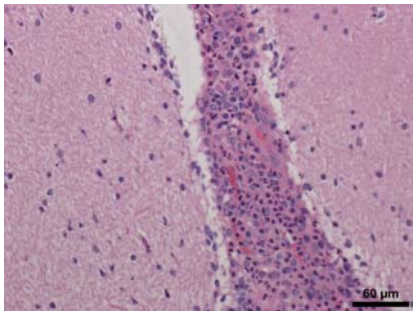

Figure 1. Severe neutrophilic meningitis associated with *S. gallolyticus* subsp. *pasteurianus*

Clostridium perfringens type D outbreaks in dairy goats

Maria Spinato, Durda Slavic, Paula Menzies

Outbreaks of enteritis in adult does caused by *Clostridium perfringens* type D have increased over the past several years, in parallel with the rapid expansion of the dairy goat industry in Ontario. Clinical presentation, postmortem and histologic lesions, and prophylactic strategies often differ in goats, compared to the classical enterotoxemia ("pulpy kidney disease") syndrome in sheep caused by the same bacterium. The classic presentation of sudden death and nonspecific gross findings of pulmonary edema, glucosuria, and hydropericardium are observed infrequently in goats. **More commonly, adult does develop diarrhea of 2-4 days duration while continuing to eat and milk well prior to unexpected death**. Severely affected animals are dehydrated, may develop dysentery with fibrinous casts, and exhibit abdominal pain and depression. Case fatality rates can be high.

At postmortem examination, does are typically in good body condition, dehydrated, and may or may not have external evidence of diarrhea. Lesions in the intestinal tract range from subtle mucosal congestion and edema, to more florid fibrinohemorrhagic enterocolitis that resembles other bacterial enteritides such as salmonellosis (Fig. 1). **Sections of mid to distal jejunum and spiral colon are the best samples to submit for histologic examination and bacterial culture to confirm *C. perfringens* type D.**

A recent feed change is commonly reported in goat dairy farms experiencing an outbreak of clostridial enterocolitis. Insufficient adaptation of rumen microflora to the altered ration results in an increased amount of undigested carbohydrate passing through to the intestines, providing a substrate for bacterial proliferation. Recently, it has been identified that a low glucose level, also due to undigested starch, actually stimulates production of the epsilon toxin by *C. perfringens* type D. It has been suggested that goats develop more severe intestinal lesions compared to sheep because intestinal absorption of epsilon toxin into circulation is slower in goats.

Goats also differ from sheep in their response to clostridial bacterins. Antibody levels tend to drop rapidly in this species, with non-protective titers reported as early as 90 days post-vaccination. This means that a vaccination program should be developed that ensures kids are properly vaccinated (12 and 16 weeks of age, perhaps as young as 8 weeks if a herd is experiencing issues with the other clostridial pathogens), and that **does are vaccinated every 4 months as a minimal frequency**. **However, vaccination alone is likely insufficient to control outbreaks of clostridial enteritis - dietary management is also important. Feeding frequency, access to fiber and avoidance of ruminal acidosis are important management practices to employ for reducing the incidence of *C. perfringens* type D outbreaks in goat dairies**. *AHL*

Reference

Uzal FA, et al. Diseases produced by *Clostridium perfringens* type D. In: Uzal FA, et al., eds. Clostridial Diseases of Animals. New Jersey: Wiley Blackwell, 2016:157-176.

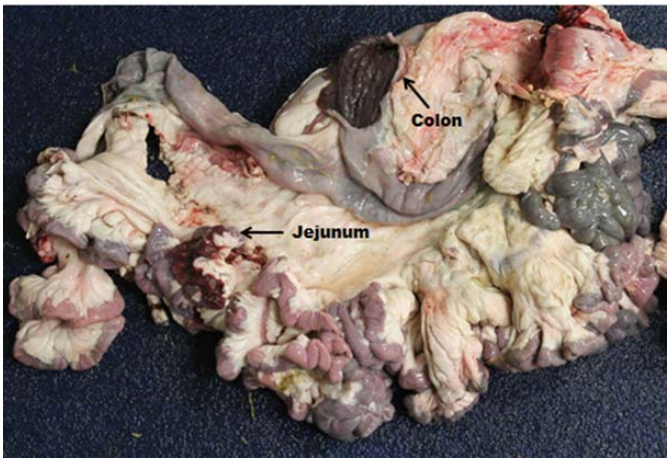

Figure 1. Intestinal tract from a doe diagnosed with *C. perfringens* type D enterocolitis. Note the opened segments of jejunum and colon with exposed hemorrhagic mucosa.

Update on Cache Valley virus abortions in small ruminants 2016-17

Maria Spinato, Janet Shapiro, Rebecca Egan, Jocelyn Jansen, Paula Menzies

Small ruminant producers and veterinarians have been closely monitoring aborted fetuses and stillborns for deformities that could presage a recurrence of the Cache Valley virus (CVV) outbreak experienced during the 2015-16 lambing period. One caprine fetus with scoliosis, lordosis, domed forehead and prognathism, and one lamb fetus with palatoschisis and a widened asymmetrical face were examined by AHL and OVC pathologists, respectively. No brain deformities were identified grossly in either fetus. Thoracic fluid or heart blood was submitted for CVV antibody determination; both cases tested negative.

Based upon anecdotal reports and the absence of deformed fetuses fitting the case definition of CVV submitted to the AHL between December 2016 - February 2017, this appears to be a quiet season for this arboviral cause of small ruminant abortions. This may be the result of the development of strong maternal herd immunity following the 2015-16 outbreak and/or decreased circulating virus in the fall of 2016. *AHL*

Anaplasma marginale in a cow in Ontario

Felipe Reggeti, Kristiina Ruotsalo

In December of 2016, the AHL clinical pathology laboratory received EDTA blood and serum from a 3-y-old Holstein cow from Wellington County, with a recent history of abdominal discomfort, scant feces, and nonspecific malaise. The cow was a recent herd addition purchased from the United States. CBC results documented marked anemia (hemoglobin 29 g/L, reference interval 84-120 g/L)., with a marked regenerative response characterized by RBC anisokaryosis, polychromasia, and basophilic stippling. **Peripheral blood smear examination revealed erythrocytes containing 1-2, small, round, basophilic, inclusions at the cell periphery, consistent with *Anaplasma marginale* (Fig. 1)**. **Anaplasmosis was confirmed by *Anaplasma* antibody cELISA in the AHL virology laboratory, and by PCR (Kansas State VDL).** *A. marginale* had been identified in July, 2013, in a cow in eastern Ontario, by the AHL.

On April 1, 2014, anaplasmosis was removed from the list of federally reportable diseases and placed on the list of provincially immediately notifiable diseases, meaning that only laboratories are required to report suspected or confirmed cases. Cows purchased from infected areas of North America are no longer tested before entering Canada; therefore, the risk of introduction may be increasing. *AHL*

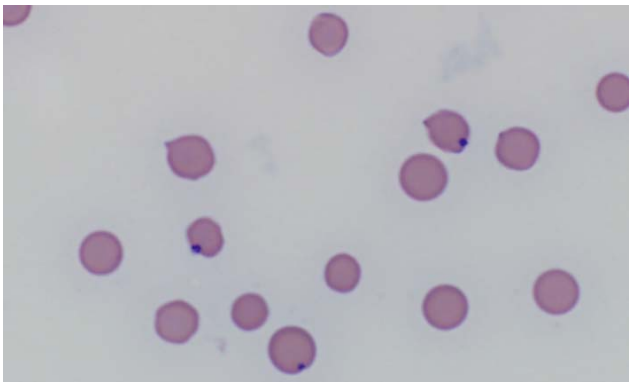

Figure 1. Anaplasma marginale in bovine red cells.

SWINE

Porcine circovirus update

Josepha DeLay, Jim Fairles, Davor Ojkic

The significance of porcine circovirus 2 (PCV-2) to the swine industry is recognized worldwide. Vaccination strategies have successfully controlled PCV-2-associated disease (PCVAD) in many herds. However the emergence and evolution of new genotypes of the virus raise concern about the level of protection offered by current PCV-2 vaccines as genotype prevalence shifts within the North American swine population. Currently, 5 PCV-2 genotypes have been recognized (PCV-2a, PCV-2b, PCV-2c, PCV-2d, and the newly proposed PCV-2e). PCV-2a, PCV-2b, and PCV-2d are associated with clinical disease, and PCV-2d is further classified as PCV-2d-1 and PCV-2d-2. PCV-2c is rarely identified and may be of limited or no clinical significance. PCV-2e was recently identified, and the clinical significance of this genotype is currently undetermined.

To evaluate the prevalence of PCV-2 subtypes identified in Ontario herds, gene sequence analysis was completed for all PCV-2 PCR-positive samples received at the AHL between June 2015 and October 2016 (Table 1). **PCV-2a and PCV-2b were the predominant genotypes among these samples**, and were present in approximately equal frequency. PCV-2d was detected in a single sample. This is in contrast to a recent report in which a change from PCV-2b to PCV-2d-2 predominance was identified among US swine. Most commercial vaccines use PCV-2a, however cross-protection against PCV-2b and PCV-2d has been demonstrated in experimental infections. Despite this, monitoring of PCV-2 genotype dynamics within swine populations is important for anticipating genotype shifts that may occur.

A novel circovirus, designated PCV-3, was recently identified by metagenomic sequencing from 2 separate case series in the US. Clinical syndromes among these cases included sow death with lesions compatible with porcine dermatopathy-nephropathy syndrome (PDNS) and concurrent abortion; and myocarditis, myocardial arteriolitis, and systemic inflammation in nursing and weaned pigs. PCV-3 antigen or nucleic acid was detected in association with lesions in these pigs. The exact role of PCV-3 regarding these disease syndromes is unknown, and Koch's postulates remain to be fulfilled. Routine testing is not currently available for PCV-3. *AHL*

Table 1. PCV genotypes detected among PCV2 PCR-positive cases at the AHL, June 2015-October 2016.

Virus / genotype	Number of cases
PCV-2a	**9 (35%)**
PCV-2b	**8 (31%)**
PCV-2c	0
PCV-2d	1
PCV-2e	4
PCV-1	3
Unable to type	1
Total	26

AVIAN/FUR/EXOTIC SPECIES

Snake fungal disease: update and first described cases in Ontario

Nicole Nemeth, Lenny Shirose, Doug Campbell, Hugh Cai, Claire Jardine

Snake fungal disease (SFD) is a recently described condition of wild snakes in North America, and is caused by infection with ***Ophidiomyces ophiodiicola***. The disease was first documented in wild snakes in New Hampshire in 2006, and has since been reported in 16 U.S. states in a variety of snake species. Grossly, scabs and crusts are on scales or skin, sometimes with underlying pustules or ulcers, subcutaneous nodules (often facial), ocular cloudiness, and dysecdysis. Histopathology includes hyperkeratosis, granulomatous dermatitis, and ulceration. Lesions may extend to underlying muscle and bone. **Ontario represents the northern extent of the geographic ranges of numerous endangered snake species that are likely susceptible to SFD.** Therefore, a better understanding of the prevalence and potential effects of SFD in Ontario is needed.

The first documentation of SFD in Canada occurred in a free-ranging eastern foxsnake (*Pantherophis vulpinus*) from southwestern Ontario in 2015; 3 additional foxsnakes have since been diagnosed. Diagnostic evaluation revealed characteristic lesions that tested PCR-positive for *O. ophiodiicola*. Based on these findings, additional studies to assess the prevalence and distribution of this fungus in Ontario have been initiated. These include testing of opportunistically collected skin swabs from live snakes, carcasses, and the environment, along with continued diagnostic evaluations.

AHL

Acknowledgments

We thank the following for sample submissions and logistical support: Tony Braithwaite (Kingsville Animal Clinic), Steve Marks (AMEC-Windsor, ON), Tarra Degazio and Tammy Dobbie (Point Pelee National Park), Emily Slavik and Pilar Manorome (Rondeau Provincial Park), Jacqueline Litzgus (Laurentian University), Christina Davy (Ontario Ministry of Natural Resources and Forestry), consultants at Blazing Star Environmental and Natural Resources Solutions, Inc., and Erin Harkness and David Cristo of the CWHC.

Figure 1. Gross lesions of SFD may include scabs and crusts on scales or skin (Figs. 1A-E), underlying pustules or ulcers, subcutaneous nodules on the head, neck, and body, ocular opacity (Fig. 1F), and dysecdysis. The above are examples of more chronic lesions in an eastern foxsnake over a 3-year period (2011-1A; 2014-1B,C), queensnake (1D, F) and eastern foxsnake (1E).

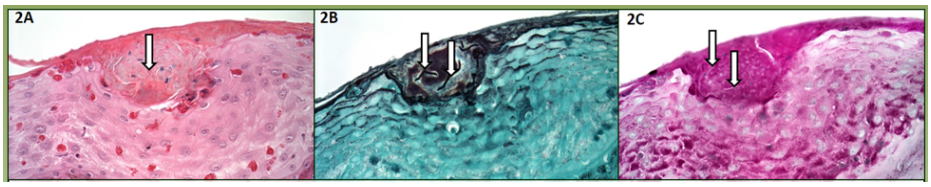

Figure 2. Fungi (arrows) within a focus of necrosis within a hyperplastic and disorganized epidermis with scattered heterophils: H&E (2A), GMS (2B), PAS (2C).

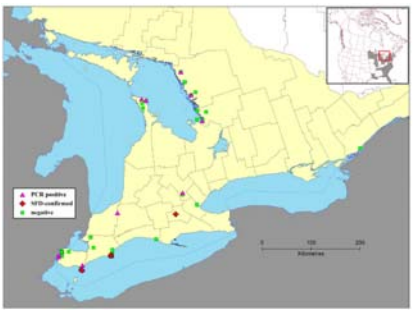

Figure 3. Locations of *O. ophiodiicola* PCR- and SFD-positive snakes in Ontario in 2012-16.

This article is based on a poster presented at the annual meeting of the American College of Veterinary Pathologists, 2016.

HORSES

OAHN equine surveillance study on Lyme disease and anaplasmosis – preliminary results

Luis Arroyo, Scott Weese, Alison Moore, Murray Hazlett

During the summer of 2016, Ontario equine practitioners were asked to participate in the collection of serum from horses in the province. In particular, we requested samples from horses that had never traveled outside of Ontario, were outdoors for more than 6 hours a day, and were more than 2 years of age. Equine veterinarians who agreed to participate submitted a total of 567 samples.

Antibodies against the **C6 antigen of *Borrelia burgdorferi*** (IDEXX Snap 4DX Plus test) were detected in 27 (5%) horses; 5 additional horses were positive for *Anaplasma* spp. antibodies.

Antibodies against **OspA, OspC, and/or OspF of *B burgdorferi* (**Cornell University equine multiplex ELISA), were identified in 39 (7%) samples.

This information was presented at the annual meeting of the Ontario Association of Equine Practitioners in February 2017. Further analysis is ongoing. *AHL*

Alcohol and Gaming Commission of Ontario (AGCO) Death Registry: 2003 - 2016 postmortem summary

The Alcohol and Gaming Commission of Ontario (AGCO; formerly the Ontario Racing Commission, ORC) continues in its proactive approach to advance racehorse welfare and safety of human and animal participants. In 2003, Ontario became one of the first North American racing jurisdictions to require mandatory reporting of racehorse deaths, in order to monitor, research and improve knowledge of why these events occur. Postmortem (PM) exams conducted at the Animal Health Laboratory through the AGCO Death Registry continue to provide comprehensive data regarding the causes of morbidity and mortality in racehorses in this province. To date, PMs have been carried out on 1,013 horses through the Death Registry program (Table 1). Annual variation in the number of PM cases reflects discretionary requirement for PM 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 conducted in 2015 as part of a separate retrospective study and as a result, some cases have been reclassified from results presented in previous editions of the AHL Newsletter. Results of the study will be published in the near future in the Journal of Veterinary Diagnostic Investigation.

Since 2015, **computed tomography (CT) of fractured and contralateral limbs has been carried out on select Death Registry 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-existing lesions, primarily in bone, that contribute to catastrophic fractures. The procedure was continued in 2016, with CT imaging of 24 of 27 (89%) fracture cases submitted for PM exam. Pre-existing lesions in bone or occasionally soft tissue were identified and considered predisposing to fracture in 10 of 24 (42%) cases.

Exercise-associated sudden death is of special concern among those cases reported through the Death Registry (Table 3). Significant pulmonary hemorrhage was identified in 78 of 163 (48%) sudden death cases. The cause of death in such cases is often attributed to exercise-induced pulmonary hemorrhage (EIPH), although the pathogenesis of pulmonary hemorrhage in these horses is not well understood. Severe acute hemorrhage involving pericardium or body cavities was identified in 30 of 163 (18%) sudden death cases. In a significant proportion of exercise-associated sudden death cases, no significant lesions were identified and the cause of death remained undetermined (37 of 163, 23%). It has been speculated that exercise-associated cardiac arrhythmia, 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.[1]

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*

Reference

1. Physick-Sheard PW, McGurrin MKJ. Ventricular arrhythmias during race recovery in Standardbred racehorses and associations with autonomic activity. J Vet Intern Med 2010; 24: 1158-1166.

Continued —>

AGCO Death Registry—continued.

Table 1. Breed distribution of AGCO Death Registry submissions to the AHL, 2003-2016.

Breed / Year	Standardbred	Thoroughbred	Quarter Horse	Total
2003	63 (52%)	59 (48%)	0	**122**
2004	81 (57%)	60 (43%)	0	**141**
2005	59 (54%)	51 (46%)	0	**110**
2006	58 (55%)	46 (43%)	2 (2%)	**106**
2007	66 (54%)	53(43%)	3(3%)	**122**
2008	27 (53%)	24(47%)	0	**51**
2009	28 (62%)	16 (36%)	1 (2%)	**45**
2010	22 (69%)	8 (25%)	2 (6%)	**32**
2011	24 (52%)	18 (39%)	4 (9%)	**46**
2012	20 (59%)	14 (41%)	0	**34**
2013	19 (41%)	26 (55%)	2 (4%)	**47**
2014	21 (41%)	22 (43%)	8 (16%)	**51**
2015	29 (52%)	24 (43%)	3 (5%)	**56**
2016	15 (30%)	32 (64%)	3 (6%)	**50**
Total	**532**	**453**	**28**	**1,013**

Table 2. Significant postmortem lesions identified in AGCO Death Registry submissions by body system, 2003-2016.

Diagnoses by body system:	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016
Fracture, limbs	51 (42%)	69 (49%)	48 (44%)	43 (41%)	58 (48%)	16 (31%)	3 (7%)	7 (22%)	5 (11%)	2 (6%)	22 (47%)	23 (45%)	25 (45%)	**27 (54%)**
Fracture, other	10	4	6	11	8	5	0	3	6	2	2	8	4	**4**
Non-fracture musculoskeletal	8	7	8	7	5	4	4	2	0	0	2	3	4	**1**
Gastrointestinal	16	22	17	16	18	4	4	6	5	6	4	6	5	**5**
Respiratory (including EIPH)	17	12	5	4	11	6	15	7	9	7	4	6	4	**3**
Cardiovascular	5	6	3	6	1	6	2	2	2	1	5	2	0	**2**
CNS	3	7	8	4	0	1	2	0	6	2	3	0	2	**2**
Renal	0	1	0	0	2	0	0	0	0	0	0	0	0	**0**
Other, whole body conditions (e.g., septicemia)	2	0	6	3	5	2	4	2	5	4	3	2	6	**3**
Injection-associated	2	6	3	5	3	2	5	1	5	5	1	0	3	**0**
Cause of death undetermined	8 (7%)	7 (5%)	6 (5%)	7 (7%)	11 (9%)	5 (10%)	6 (13%)	2 (6%)	3 (7%)	5 (15%)	1 (2%)	1 (2%)	3 (5%)	**3 (6%)**
Total	**122**	**141**	**110**	**106**	**122**	**51**	**45**	**32**	**46**	**34**	**47**	**51**	**56**	**50**

Table 3. Significant postmortem lesions contributing to or causing death in exercise-associated sudden death cases reported to the AGCO Death Registry, 2003-2016.

Body system affected and significant lesions / cause of death	Number of cases
Respiratory	**81**
EIPH	62
Pulmonary hemorrhage (not classified by pathologist as EIPH)	16
Miscellaneous	3
Cardiovascular	**35**
Body cavity or pericardial hemorrhage	17 (source confirmed in 2 cases)
Aortic rupture and cardiac tamponade	13
Miscellaneous cardiac lesions	5
Cause of death undetermined	**37**
Skull fracture (potentially secondary to collapse)	**5**
Sepsis / Disseminated intravascular coagulation	**5**
Total	**163**

Updated AHL supplies list

Josie Given, Rina Pigozzo, Jim Fairles

⇒ The AHL provides various sampling supplies at cost to our clients. We have updated the list to include 3 new products.
⇒ AHL now has **40-tube blood mailers** available for purchase at $3/box. Ensure the survival of your specimens by shipping them in a sturdy container!
⇒ AHL now has **larger histology containers** to accommodate various tissue sizes:
  ⇒ 125 mL—$2.00, 250 mL—$3.00, and 500 mL—-$4.00.
⇒ The complete list is available at:
  ⇒https://www.uoguelph.ca/ahl/sites/uoguelph.ca.ahl/files/AHLSupplyOrderForm%202017.pdf
  ⇒email ahl.supplies@uoguelph.ca to order!

Bacterial and viral transport media *Jim Fairles* - from AHL LabNote 36

One of the most important aspects of diagnostic work is the actual sampling and handling of the samples prior to being received at the diagnostic laboratory.

The best sample for microbiology testing is usually the substrate itself – feces, tissue, or fluid in leak-proof containers (liquids, feces) or leak-proof Whirl-Pak bags (tissue, feces) (Ziplock bags are discouraged!).

Bacteriology and Mycoplasmology - AHL prefers a regular gel-based bacteriology swab.

- Starswab Amies Clear - VP # 1350500, VWR # CA66410-103L; or
- BD single swab - Fisher # B4320115

Virology - If swabs are needed, AHL prefers virus transport medium swabs for virology PCR tests. For some testing (PEDV), a fecal swab in VTM is preferred rather than feces. **Regular gel-based bacteriology swabs are unsuitable for PCR testing**. Dry swabs moistened with sterile saline in a sterile container can be used, but **VTM is preferred**.

- BD™ Universal Viral Transport Standard Kit – Fisher Scientific: Catalog # 22 031 15 (Also can be used for *Mycoplasma*, *Ureaplasma*, and *Chlamydia*); or
- Multitrans™ Collection and Transportation System, Starplex® VWR # CA73270-008

The VTM swabs pictured above are also available from the AHL for $2.50 – please email ahlinfo@uoguelph.ca to order.

COMPANION ANIMALS

Disseminated idiopathic myofasciitis in a ferret

Jan Shapiro, Penny Waite

A 6-mo-old pet ferret was examined at a veterinary clinic after a 1-mo history of pyrexia, weakness, and lethargy. A respiratory tract infection was suspected, and a course of antibiotics was undertaken. One month later, the clinical signs had not abated with antibiotic treatment and the ferret was losing weight, so it was surrendered to the clinic. On re-examination, the veterinarian found the animal to be pyrexic, weak, lethargic, and dehydrated, with excessively warm and mildly swollen limbs, and a heart murmur. The ferret was euthanized. Postmortem findings were nonspecific, and included thin body condition, mottled and congested lungs, and a mildly enlarged mesenteric lymph node. The veterinarian collected a variety of tissues including multiple muscle samples, which were fixed and submitted to AHL-Kemptville for histopathology.

Significant lesions were restricted to muscle tissue and were dramatic. Intense multifocal-to-coalescing infiltrates of neutrophils admixed with small numbers of mononuclear cells were in myocardium, skeletal muscle and the associated fascia (Fig. 1), and smooth muscle of the esophagus, trachea, and colon, often extending into the submucosa. Suppurative-to-pyogranulomatous myositis was multifocally accompanied by necrosis of myofibers, and occasional skeletal myofiber regeneration, and there were more chronic areas of stromal collapse. Special stains for bacteria, mycobacteria, and mycotic agents detected no etiologic agents.

The clinical history and histologic lesions in this case are consistent with disseminated idiopathic myofasciitis (DIM) of ferrets. This is an uncommon, progressive disease with acute or subacute onset, causing inflammation of smooth, striated, and cardiac muscle and the associated fascia. DIM usually affects ferrets younger than 18 mo, with a reported range of 5-24 mo and an average age of 10 mo. The most common clinical signs are a high fever, lethargy, weakness, inappetance, pain, and dehydration that is often refractory to treatment. Signs reported less commonly are labored fast breathing, tachycardia, heart murmur, subcutaneous swelling, and enlarged lymph nodes. Most ferrets develop moderate-to-marked mature neutrophilia, but clinical chemistry is usually not diagnostic, including lack of elevation of creatine kinase.

The cause of DIM is not known. First recognized in 2003, the early cases had an association with a specific, now discontinued, canine distemper vaccine, leading to the supposition that DIM is an immune-mediated disease. However, the epidemiology of cases since then has not confirmed a causative role of vaccination. A genetic predisposition has been speculated but not proven. An infectious cause has not been ruled out, but to date, no consistent pathogen has been detected in confirmed cases. *AHL*

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Pfent CM. Pathology in practice. J Am Vet Med Assoc 2013;242:43-45.

Ramsell KD, Garner MM. Disseminated idiopathic myofasciitis in ferrets. Vet Clin Exot Anim 2010;13:561-575.

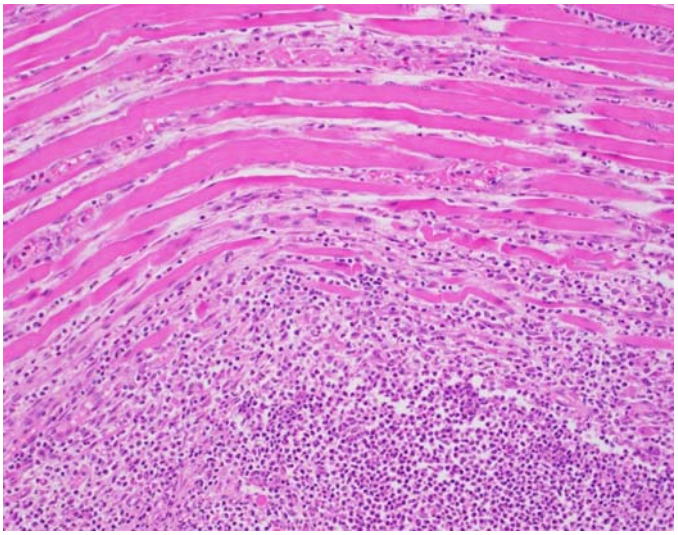

Figure 1. Skeletal muscle showing severe pyogranulomatous myositis (H&E stain)