



# AHL Newsletter

Volume 10, Number 1, page 1

March, 2006

ISSN 1481-7179

## In this issue:

Demographics	1
VCPR	1
Submission form completion	2
<b>Zoonosis update</b> 2001-2005	3
<b>Ruminants</b> - Bovine coronavirus in respiratory disease	4
<b>Poultry</b> - Viral infection of young pigeons	5
<b>Swine</b> - Porcine circovirus 2 update	6
Interpreting PCV2 pathology reports	6
Ongoing PRRSV infections	7
<b>Horses</b> - <i>Mycobacterium holsaticum</i> abortion	7
ORC Death Registry, 2003-2005	8
<b>Companion animals</b> - OFA canine sebaceous adenitis testing	8

## Demographics - thank you!

In return for OMAFRA financial support of AHL testing of food animal specimens, the AHL shares case data with OMAFRA for the purpose of disease surveillance in Ontario. For these data to become useful information, certain fields are mandatory on the submission form:

### Veterinary clinic/veterinarian

Owner - at least **owner name and postal code**, full address not necessary

### Demographics

- **species, breed, age, sex, weight, ID**
- **no. at risk, no. sick, no. dead**, duration of problem
- **body system(s)** affected

For those who routinely supply these data - thank you! If these data are missing, the usefulness to OMAFRA and to you is greatly reduced.

*Please see page 2 for an example of a completed form.*

*Enclosed with this newsletter is a short note and questionnaire from OMAFRA regarding farm animal demographic data.*

## AHL client communications and the veterinarian-client-patient relationship

Veterinarians in the AHL do not have a valid veterinarian-client-patient relationship (VCPR) with animal owners, and our message to owners has always been "The AHL provides specialized diagnostic services to support your veterinarian. We do not give treatment recommendations. Given their knowledge of your premises and of your local area, your own veterinarian is best placed to give you specific information about prevention, treatment, and control of disease."

Just as we as human patients deal through our own physician concerning our lab results, it is appropriate to have animal owners deal through their own veterinarian concerning veterinary lab results.

- **The AHL will no longer accept submissions directly from owners without a veterinarian named as the primary contact.**
- In the event of a dispute with the owner's current veterinarian, we will ask for the name of a third-party veterinarian.
- The AHL will report to owners only on the request of the veterinarian.
- We will bill the veterinarian at the owner's request only if approved by the referring veterinarian.
- Our general practice is to bill veterinarians only. When animal owners appear at one of our labs in person with their lab submission, we will require payment in advance (cash, cheque, credit card). We will continue to direct animal owners to contact their veterinarian for interpretation of their laboratory report.

Visit our new website at  
<http://ahl.uoguelph.ca>

The May 1, 2006 AHL  
User's Guide and Fee  
Schedule will be  
distributed in April.

Watch for it!!

# Critical surveillance information on submission forms

Below is a generic AHL submission form with information completed as noted on p. 1:

## ANIMAL HEALTH LABORATORY GENERIC SUBMISSION FORM



AHL case # \_\_\_\_\_  
Resubmission?  Yes  
Previous # \_\_\_\_\_

NOT to be used for OHSFP submissions

Date & time samples taken: 2006-02-10 Date sent: 2006-02-10  
Submitted by:  Veterinarian  Owner  Agent Bill:  Veterinarian  Owner  Agent  Insurance claim?  
 Possible litigation?

Clinic no. <u>12345</u>	Client no.
Clinic <u>Anytown Veterinary Clinic</u>	Owner/Farm name <u>G. and M. Jones</u>
Address <u>123 Main St., Anytown, Ont.</u>	Address
Veterinarian <u>DR. J. SMITH</u>	County <u>NOB</u> Postal code <u>250</u>
Phone <u>519-824-1234</u> Fax <u>519-821-1235</u>	Phone Fax
Email <u>anytownvc@telus.com</u>	Email

Demographic information must be supplied for submissions if you wish to be charged the rates supported by the Ontario Ministry of Agriculture and Food (OMAF). Information related to the testing done on samples from Ontario food animals submitted to the Animal Health Laboratory (AHL) may be used by the AHL and may be shared with OMAF for the sole purpose of statistical surveillance of animal health in Ontario. The AHL complies with the federal Health of Animals Act regarding reportable or notifiable diseases. Inquiries may be directed to: Manager, AHL (519) 824-4120 ext. 54510.

SUBMITTER SIGNATURE: J. Smith  
Species: BOVINE Breed: HOLSTEIN Age: 4 d. w. m. y. Sex:  F  M  Neuter

Barn ID: HUTCH 5 Herd/flock size: 150 Cattle:  Dairy  Veal  Beef cow-calf  Beef stocker-feeder  
Swine:  Farrow  Farrow-finisher  Weaner  Grower-finisher  SEW

Weight: 45 g kg Animal ID (List multiples on back): 98765  No ID

No. at risk: 10 No. (or %) sick: 5 No. (or %) dead: 2 Duration of problem: 6 d. w. m. y.

Body systems affected:  Digestive  Respiratory  Nervous  Integument / Senses  Cardiovascular  Hemopoietic  
 Endocrine  Reproductive  Mammary  Musculoskeletal  Urinary  Sudden death  Abortion  Multisystemic

### AHL Newsletter

March 2006 - Volume 10, Number 1  
Editor: **Grant Maxie**, DVM, PhD, Diplomate ACVP  
Editorial Assistant: **Ms. Helen Oliver**

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. All material is copyright 2006. Ideas and opinions expressed herein do not necessarily reflect the opinions of the University or the editor.*

Articles may be reprinted with the permission of the editor and with appropriate credit given to the AHL Newsletter.

#### Mailing address & contact information:

(please return all undeliverable Canadian addresses to:)

Animal Health Laboratory  
Laboratory Services Division, University of Guelph  
Box 3612, Guelph, Ontario, Canada N1H 6R8  
Phone: (519) 824-4120 ext. 54538; fax: (519) 821-8072  
Email: [holiver@lsd.uoguelph.ca](mailto:holiver@lsd.uoguelph.ca)

ISSN 1481-7179

Canada Post Publications number - 40064673

### Contributors to this issue:

#### From the Animal Health Laboratory:

- Brian Binnington, DVM, Dip Path, Diplomate ACVP
- Hugh Cai, DVM, MSc, DVSc
- Susy Carman, DVM, Dip SA Med, PhD
- Joseph DeLay, DVM, DVSc, Diplomate ACVP
- Jim Fairles, DVM, MBA
- Murray Hazlett, DVM, DVSc, Diplomate ACVP
- Brent Hoff, DVM, DVSc, DipTox
- Peter Lusic, DVM, MSc
- Emily Martin, DVM, MSc, Diplomate ACPV
- Beverly McEwen, DVM, MSc, PhD, Diplomate ACVP
- Davor Ojkic, DVM, MSc, PhD
- Jan Shapiro, DVM, DipEqSurg
- Durda Slavic, DVM, MSc, PhD

#### Other contributors:

- Larry Butler DVM, Jane Keilly DVM, Ottawa Valley Large Animal Clinic
- Ben Mighton, DVM, Walkerton-Hanover Veterinary Clinic

*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 identified at the AHL, 2001 - 2005

Beverly McEwen, Durda Slavic, Davor Ojkic, Susy Carman, Peter Lusic

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 2005, AHL staff identified **rabies, methicillin-resistant *Staphylococcus aureus* (MRSA), and West Nile virus infections**. Other selected zoonotic pathogens isolated and/or identified at the AHL are given in 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.

*Leptospira spp.*, *Salmonella sp.*, *Streptococcus sp.* infections, and *Cryptosporidium sp.* are the most frequently identified zoonotic pathogens at the AHL since 1999. Occupational exposure to pigs and horses is a risk factor for *S. suis* and *S. zooepidemicus* infections. *West Nile virus* continues to be identified by PCR in non-domestic species and occasionally in horses by IgM ELISA, histology and immunohistochemistry. AHL

Table 1. Selected zoonotic pathogens isolated and/or identified at the AHL, 2001-2005

Agent	Bovine	Swine	Equine	Ovine	Caprine	Chicken	Turkey	Canine	Feline	Other	2005	2004	2003	2002	2001
<i>Bartonella henselae</i>									1		<b>1</b>	0			
<i>Bordetella bronchiseptica</i>		46	5					2	6	11	<b>70</b>	78	84		84
<i>Borrelia burgdorferi</i>								1			<b>1</b>				
<i>Campylobacter coli/ jejuni/ fetus subsp. fetus</i>	3	3		1				4	1	2	<b>14</b>	26	31	33	36
<i>Chlamydomphila sp.</i>				3	2					1	<b>6</b>	6	19	10	12
<i>Coxiella burnetii</i> (Q fever)				5	1						<b>6</b>	4	6	6	7
<i>Cryptosporidium sp.</i>	189	6	1				2			3	<b>201</b>	61	129	199	160
<i>Eastern equine encephalitis virus</i>											<b>0</b>	2	10	0	0
<i>Francisella tularensis</i>											<b>0</b>	1	1	1	3
<i>Giardia sp.</i>	8							10	1		<b>19</b>	13	10	4	19
<i>Listeria monocytogenes</i>	5	2		3	6						<b>16</b>	34	27	37	44
Methicillin-resistant <i>Staphylococcus aureus</i>			21			1		2			<b>24</b>	133	73	13	1
<i>Mycobacterium bovis</i>											<b>0</b>	0	0	1	0
Rabies	1										<b>1</b>	0	0	0	5
<i>Salmonella sp.</i>	83	357	263	7		15	25	2		84	<b>836</b>	640	824	716	565
<i>Streptococcus suis</i>	4	604									<b>608</b>	447	392	477	380
<i>Streptococcus equisimilis</i>	1	63	14							4	<b>82</b>	140	111	144	126
<i>Streptococcus zooepidemicus</i>	8	2	174						2	2	<b>188</b>	186	211	222	175
<i>Toxoplasma sp.</i>				2	1				1	2	<b>6</b>	10	5	12	12
<i>West Nile virus</i>			4							96	<b>100</b>	158	173	91	
<i>Yersinia enterocolitica</i>		2						1			<b>3</b>	8	8	5	8

Table 2. *Leptospira spp.* seropositive samples ( $\geq 1:320$ ) identified at AHL, 2001 – 2005, microscopic agglutination test (MAT)

<i>Leptospira spp.</i> serovar	Bovine	Swine	Equine	Canine	Feline	Other & not specified	2005	2004	2003	2002	2001
<i>L. autumnalis</i>			3	161		51	<b>215</b>	226	122	46	32
<i>L. bratislava</i>		151	64	144	1	1	<b>361</b>	329	151	139	120
<i>L. canicola</i>			1	42			<b>43</b>	56	17	4	6
<i>L. grippityphosa</i>		44	8	107		1	<b>160</b>	196	76	29	44
<i>L. hardjo</i>	47			4			<b>51</b>	60	34	30	21
<i>L. icterohaemorrhagiae</i>	17	1	3	37			<b>58</b>	111	122	163	289
<i>L. pomona</i>	16	39	22	70		1	<b>148</b>	128	122	105	110
<b>Total</b>	<b>80</b>	<b>235</b>	<b>101</b>	<b>565</b>	<b>1</b>	54	<b>1036</b>	1106	644	516	622

# AHL Lab Reports

## RUMINANTS

### Bovine coronavirus in respiratory disease

*Murray Hazlett, Ben Mighton, Josepha DeLay, Susy Carman*

After purchase of several 300 lb beef replacement heifers, 2 resident cows developed dysentery, pneumonia and high fever, and 1 of these died after a 2-day course. Subsequently other cows developed a cough and fever, and diarrhea was noticed in a calf. Tissues from the dead cow and fecal samples were submitted to the AHL for histopathology and ancillary testing.

Histology of the lung of the dead cow revealed neutrophilic bronchiolitis, with necrosis of some smaller bronchioles. Immunohistochemistry (IHC) staining showed *Bovine coronavirus* (BCV) antigen present in bronchiolar epithelium in 1 of 2 sections of lung tested (Fig 1). There was a positive antigen detection ELISA on 1 of 2 fecal samples submitted. *Pasteurella multocida* was also isolated from the lung.

**BCV has been associated with calf diarrhea, winter dysentery, calf pneumonia and respiratory disease in adult cattle, as well as instances of concurrent pneumonia and diarrhea in young and older cattle.** The case above, with a history of both pneumonia and diarrhea, is a good example. Most often at the AHL, a diagnosis of BCV respiratory infection is confirmed serologically, and IHC for BCV is not commonly performed on lung for pneumonia cases. In both respiratory and diarrhea cases associated with BCV, it is rare to find BCV as the only agent involved.

A search of the diagnostic pathology records of the AHL since 1998 identified 49 cases of BCV infection. History and/or lesions often indicated only intestinal disease, but 8 of these cases had evidence of both respiratory and gut disease, and in 3 there was respiratory disease alone. Table 1 divides the animals into 3 groups depending on age (birth to 2 months, 3 months to 1 year, and 1 year and over). Many of the 36 respiratory cases in the young age group were calves with diarrhea, that also had aspiration pneumonia. Diagnoses at individual case levels were made by antigen detection ELISA on feces (33 cases), direct florescent antibody testing on intestine (8 cases), IHC on lung or intestine (8 cases), and virus isolation on lung (3 cases), with one presumptive case based on clinical history and gross lesions. AHL

**Reference**

Carman PS, Hazlett MJ. Bovine coronavirus infection in Ontario 1990-1991. *Can Vet J* 1992; 33:812-814.

Table 1: Number of cases by age and body system submitted to the AHL from 1998 to 2005 with bovine coronavirus infection.

System	Age group Birth to 2 mo	3-11 mo	≥1 yr
Intestinal disease	36	1	1
Respiratory disease	0	3	0
Both intestinal and respiratory disease	5	1	2

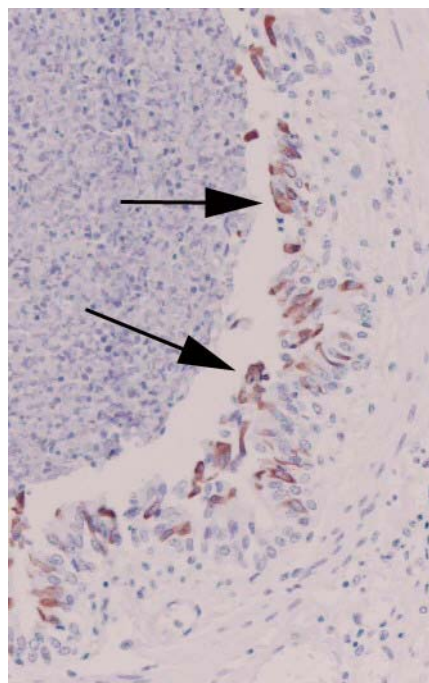


Figure 1. IHC demonstrating positive staining of bronchiolar epithelium (arrows).

# POULTRY

## Concurrent viral agents and secondary infections in young pigeons

Emily Martin, Brian Binnington

At the Animal Health Laboratory, we receive cases of mortality in young racing pigeons (< 1 year) with non-specific clinical signs including weight loss (going light), loose droppings, respiratory signs, etc. The history may indicate that there have been new introductions or mixing of birds in the loft. On gross post mortem examination, there may be no specific findings or there may be lesions suggestive of an infectious process (e.g., bacterial septicemia, fungal infection, parasitism). However, on histologic evaluation we have also found lesions consistent with concurrent viral infections. In sections of liver, hepatocytes contain intranuclear inclusion bodies with amphophilic staining consistent with adenovirus (Fig. 1, left) and/or Cowdry type A inclusions that are consistent with either an adenovirus or herpesvirus. Lymphoid organs (thymus, spleen, bursa) reveal lymphoid depletion, and the bursa of Fabricius has macrophages containing globular eosinophilic intracytoplasmic inclusion bodies consistent with circovirus (Fig. 1, right).

### Circovirus

*Columbid circovirus* is antigenically and genetically distinct from psittacine beak and feather disease (PBFD). There is no clear clinical picture that is specific for circoviral infections. Clinical signs can include loss of appetite, weight loss, diarrhea, inability to fly, nasal discharge, and respiratory difficulty. There is usually high morbidity and low mortality depending on concurrent infections, and death is usually due to secondary infections. Both horizontal and vertical transmission is suspected. The virus is found in young pigeons (i.e., <1 yr), and the first indication of infection may be the discovery of multiple other disease agents. *Columbid circovirus* is thought to be immunosuppressive, and diagnosis is primarily by histologic evaluation. The virus is environmentally stable and relatively resistant to inactivation by common disinfectants. These properties make the virus almost impossible to eliminate once in the loft. *The best approach is to prevent infection and maintain high levels of biosecurity in the loft.*

### Adenovirus

There are two types of adenovirus infections in pigeons. **Type I (classical adenovirus)** is considered to be in young pigeons and associated with stress. Clinical signs consist of watery diarrhea, vomiting and weight loss. Morbidity in young birds can be up to 100% within 2 days. If birds recover they can have impaired racing performance. Liver damage is not extensive, and *E. coli* can be a secondary infection that can cause more severe diarrhea, vomiting and death.

**Type II (necrotizing hepatitis)** was diagnosed in Belgium in 1995. It can occur in all ages and there are few clinical signs (i.e., vomiting, yellow droppings, sudden death within 24 hours of disease onset). There can be up to 100% mortality. On post mortem, the liver is swollen and pale yellow. On histologic evaluation for both Type I and Type II adenovirus, there are amphophilic intranuclear inclusion bodies associated with hepatic necrosis.

### Herpesvirus

*Columbid herpesvirus 1* (Pigeon herpesvirus) is antigenically different from other avian herpesviruses, e.g. Marek's disease herpesvirus (*Gallid herpesvirus 2* and *3*); Infectious laryngotracheitis virus (*Gallid herpesvirus 1*), and Duck enteritis virus (*Anatid herpesvirus 1*). There can be latent carriers with intermittent shedding, and transmission is by direct contact. Adults can transmit the virus to squabs shortly after hatching. Clinical signs in acute disease include conjunctivitis, sneezing, and mucus in the nostrils. A chronic condition can develop if there is secondary infection (e.g. *Trichomonas*, bacteria, mycoplasma), and clinical signs include sinusitis and severe dyspnea. On post mortem, there can be congestion of the oropharynx, development of a diphtheritic membrane over the pharynx, airsacculitis, pericarditis and hepatic necrosis.

In many cases, a loft may have a history of new introductions or mixing of birds from multiple sources. This situation provides an excellent opportunity to introduce multiple infectious agents into a loft. If circovirus is present it may be the primary infectious agent. The resulting immunosuppression could allow a bird to subsequently contract secondary or tertiary infections including viruses (adenovirus, herpesvirus, pigeon paramyxovirus), bacteria (*E. coli*, *Salmonella*), parasites (*Trichomonas*, coccidia), and fungal agents. AHL

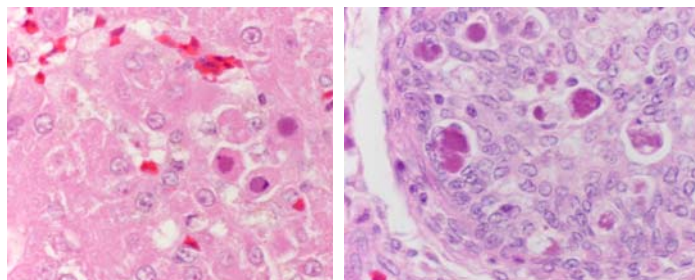


Figure 1. Left - Intranuclear inclusion bodies consistent with adenovirus in hepatocytes. Right - Intracytoplasmic inclusion bodies consistent with circovirus in bursa of Fabricius.

# SWINE

## *Porcine circovirus type 2-associated disease continued in fall of 2005*

*Susy Carman, Beverly McEwen, Josepha DeLay, Hugh Cai, Jim Fairles*

**Porcine circovirus type 2 (PCV2)-associated disease continued to be important over the fall of 2005**, with 92 new cases presented from Oct-Dec 2005, being similar to July-Sept 2005 when 90 cases were submitted. In total for 2005, there were 350 pathology cases determined to be associated with PCV2, which is 8.9% (Fig. 1) of the total of all swine cases submitted to the AHL in 2005. This is much higher than for 2004, when PCV2-associated disease was identified in 60 (2.3%) swine cases.

The PCV2 PCR-RFLP typing for all PCR testing requests continued to show a significant change from RFLP type 422 seen in previous years, to RFLP type 321 (Fig. 2), with the number of cases with RFLP type 321 increasing from 1 in 2004 to 135 in 2005. These changes in RFLP typing patterns reflect a consistent difference in the gene se-

quence recognized by two restriction enzymes. Ontario RFLP type 321 viruses continue to have greater than 99% sequence homology with each other, and to have 98% sequence homology with those reported for the UK, France and China. These Ontario viruses have only 91.6% sequence homology to the previously dominant Ontario RFLP type 422 viruses and are only 92-93% similar to those previously reported from the USA. AHL

### References

- Carman S, et al. *Porcine circovirus type 2-associated disease continues to increase.* AHL Newsletter 2005;9:30.  
 DeLay J, et al. *Porcine circovirus type 2-associated disease is increasing.* AHL Newsletter 2005;9:22.  
 van Dreumel T, et al. *Porcine circovirus type-2 associated conditions in pigs.* AHL Newsletter 2005;9:5.

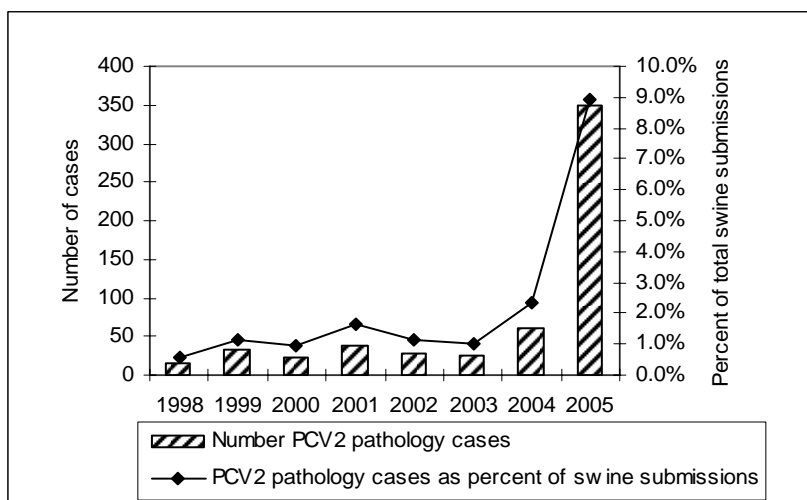


Figure 1. Number of PCV2 pathology cases, and PCV2 pathology cases as percent of total swine submissions.

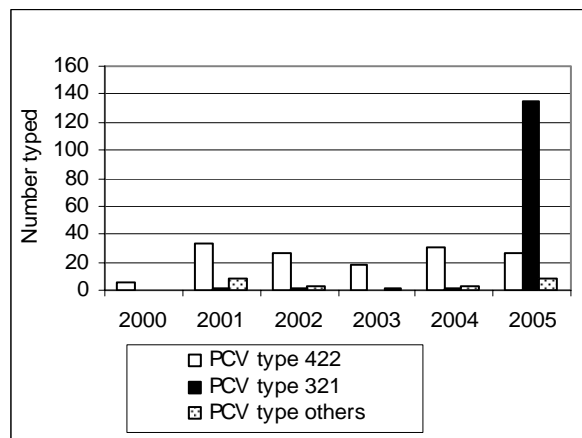


Figure 2. PCR-RFLP typing of PCV2, 2000-2005.

## Interpreting *Porcine circovirus 2* pathology reports

*Peter Lulis*

On necropsy and/or histopathology cases, **a final diagnosis of *Porcine circovirus 2* infection or PCV2-associated disease does not necessarily indicate a diagnosis of PWMS, PDNS or other clinical syndromes**, but indicates that there are lesions typical of circovirus infection and usually, positive IHC and/or PCR or other tests. **Negative IHC, PCR or other results do not necessarily rule out PCV2 infection** because distribution of the virus in tissues can be very variable - we occasionally see cases that are PCR-negative/IHC-positive or PCR-positive/IHC-negative in the same animal, cases where some lymph nodes are IHC-positive and other lymph nodes are negative in the same animal, and so on. If typical PCV2 lesions are present on histopathology, and PCR, IHC or other tests are negative or not done, a diagnosis of **PCV2 infection (PCV2-associated disease), probable PCV2 infection, or suspect PCV2 infection** may be made, depending on lesions (some lesions are considered to be pathognomonic of PCV2 infection).

Lymph node, tonsil and lung are best for PCV2 PCR tests, and these and other tissues (spleen, kidney, liver, intestine, skin) are best for PCV2 histopathology and IHC testing. AHL

## PRRSV infection is ongoing in Ontario swine herds

Susy Carman, Beverly McEwen, Jim Fairles

For Oct-Dec 2005, 38% (199/520) of all swine cases tested were identified as PRRSV-PCR positive (Fig. 1), which is less than the 55% (108/201) that tested positive in Oct-Dec 2004. Although the proportion of PRRSV-PCR positive cases has declined since 2004, **the total number of PRRSV-PCR positive cases increased** from 285/553 (52%) cases in 2004, to 682/1536 (44%) in 2005.

These data do not include semen monitoring cases. However it was not possible to exclude serum monitoring cases, such that increases in testing and PRRSV-positive cases may represent enhanced monitoring of swine herds with ongoing infections, rather than new outbreaks. AHL

### Reference

Carman S, et al. PRRSV outbreak in Ontario declines. AHL Newsletter 2005;9:31.

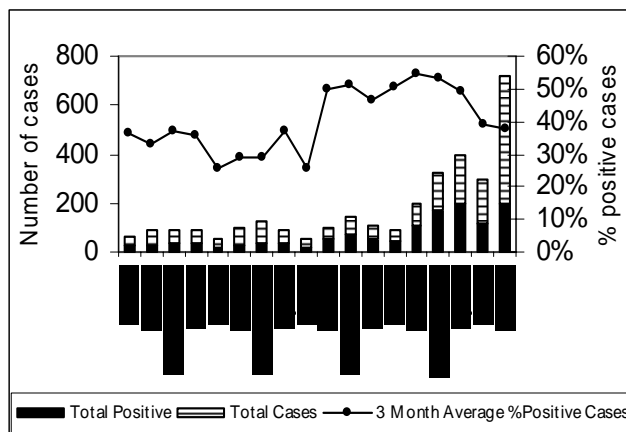


Figure 1. PRRSV PCR submissions tested by the AHL April 1, 2001- December 31, 2005, with semen monitoring removed.

# HORSES

## Equine abortion associated with *Mycobacterium holsaticum*

Jan Shapiro, Durda Slavic, Larry Butler, Jane Keilly

A female equine fetus and allantochorion, aborted at 8.5 months gestation, were submitted to the AHL in Kemptville for necropsy. The mare, a 22-year-old Thoroughbred, had a high fever for 10 hours in her third month of pregnancy, followed 1 month later by a mucoid vaginal discharge that resolved in 1 day. She had exhibited no signs of illness immediately prior to aborting or afterward.

The fetus was moderately autolysed, and had scant internal fat stores. There were a few petechiae on the skin. The liver capsule and parenchyma were mottled and petechiated. The umbilical cord showed moderate diffuse edema and ecchymotic hemorrhage. The amnion was thickened due to generalized edema, numerous 4-7 cm diameter edema-filled stromal cysts, and many 1-6 cm diameter hemorrhagic plaques. The allantochorion showed generalized thickening due to congestion, edema, and ecchymotic hemorrhage.

Fetal histopathology consisted of nonsuppurative meningoencephalitis characterized by intense multifocal gliosis and a mild diffuse mononuclear cell infiltrate in the meninges. There were intense multifocal infiltrates of lymphocytes and macrophages in skeletal and cardiac muscle. Intense multifocal infiltrates of neutrophils, macrophages and lymphocytes were in the interstitium of renal cortex and medulla. The lung had alveolar infiltrates of neutrophils, plasma cells, lymphocytes and scattered multinucleated giant cells, and there were mild perivascular infiltrates of lymphocytes.

Placental histopathology consisted of moderate multifocal to confluent hyperplasia of chorionic trophoblasts, and focal mineralization of villi; fibrin thrombi were in some

stromal blood vessels. Lesions in the amnion were dramatic, and consisted of hyperplasia of amniotic epithelium, with focal fibrin exudation and erosion, and intense multifocal to diffuse stromal infiltrates of lymphocytes, with variable numbers of neutrophils and macrophages. Hemosiderin-laden macrophages were scattered throughout the stroma, and many stromal blood vessels had fibrin thrombi.

Bacterial culture of fetal lung and stomach content yielded large numbers in pure culture of a bacterium subsequently identified by 16S rDNA sequencing and HPLC as *Mycobacterium holsaticum*. Acid-fast stains of fetal tissues showed weakly positive staining intracytoplasmic tiny coccoid organisms in macrophages in the lung and placental trophoblasts. Additional testing done, with negative results, included Warthin-Starry staining for leptospire, the fluorescent antibody test for *Equine herpesvirus*, virus isolation, and mycoplasma culture of fetal lung and stomach content.

***Mycobacterium holsaticum*, a new species of fast-growing non-tuberculous mycobacteria, was first reported in Germany in 2002, from sputum, urine and gastric fluid of human patients.** It was also isolated from a bovine lymph node in N. Ireland in 2005. **To the best of our knowledge, this organism has not been reported in horses, or as a cause of abortion in animals.** AHL

### References

- Hughes MS, et al. Molecular analysis of mycobacteria other than *M. tuberculosis* complex isolated from Northern Ireland cattle. *Vet Microbiol* 2005;108:101-112.
- Richter E, et al. *Mycobacterium holsaticum* sp. nov. *Int J Syst Evol Microbiol* 2002;52:1991-1996.

## ORC Death Registry: necropsy diagnoses, 2003 - 2005

Joseph DeLay

Since 2003, the Ontario Racing Commission (ORC) has required that all racehorse deaths be reported if the horse has died or been euthanized within 60 days of racing or qualifying in Ontario, and the necessity for a necropsy is then determined. This program continues to provide excellent data regarding the causes of morbidity and mortality in Ontario racehorses, and supports research to improve animal health and welfare in the racehorse industry.

Of the 110 horses necropsied in 2005, 59 (54%)

were Standardbreds and 51 (46%) were Thoroughbreds. Overall, the causes of death or euthanasia were similar to those in previous years. **Fractures remained the most common cause of death or euthanasia**, with 48 (44%) of animals diagnosed with limb fractures and 7 (6%) with severe fractures at other sites. Non-fracture diagnoses were varied and often single events, and the majority of cases involved the gastrointestinal and respiratory systems. *AHL*

Table 1. ORC Death Registry primary necropsy diagnoses by year and body system.

Primary diagnosis by body system	2003	2004	2005
Fracture / limbs	53 (42%)	69 (49%)	48 (44%)
Fracture / other	10	4	7
Non-fracture musculoskeletal	8	6	6
Gastrointestinal	15	19	17
Respiratory	21	17	9
Cardiovascular	5	6	5
Central nervous system	6	11	7
Integumentary	0	0	1
Renal	0	2	0
Hematopoietic	2	1	1
Whole body conditions	1	7	5
Cause of death undetermined	4	0	4
<b>Total</b>	<b>125</b>	<b>142</b>	<b>110</b>

# COMPANION ANIMALS

## OFA canine sebaceous adenitis testing at the AHL

Joseph DeLay

Sebaceous adenitis testing is now available through the AHL for registration in the Orthopedic Foundation for Animals (OFA) sebaceous adenitis (SA) database. Evaluation involves both clinical examination of the dog and skin biopsy, performed and documented by the referring veterinarian. Histopathology of biopsies at the AHL determines if lesions of SA are present, and **animals are classified according to combined histologic lesions and clinical findings.**

Submissions to the AHL for SA testing must include:

- the biopsy samples,
- completed and signed SA database application form, and
- separate OFA payment.

Regular AHL histopathology fees apply and will be billed to the referring clinic. The AHL will report results to both the clinic and to OFA. Application forms and specific biopsy instructions are available on the OFA website:

<http://www.offa.org/index.html> *AHL*

Newsletter Figures are posted in full color in the Web version of the Newsletter - <http://ahl.uoguelph.ca>