



# AHL Newsletter

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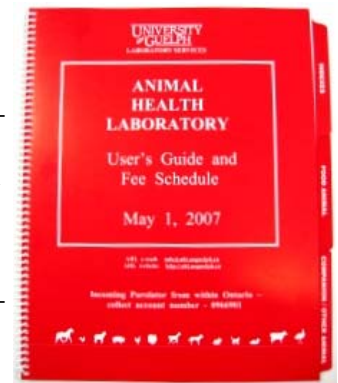
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## May 1, 2007, AHL User's Guide and Fee Schedule

We hope that this comprehensive source of information will save you time and help you deal with animal health challenges. The Guide/Schedule is also available, **on the clients-only side of our website**, at <http://www.labservices.uoguelph.ca/customer-login/index.cfm>. Please see below for client log-in sign-up information.

As noted in our User' Guide:

- **The AHL does not 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 ask for the name of a third-party veterinarian.
- **Our routine practice is to bill veterinarians only.** When animal owners appear at one of our labs in person with their lab submission, we require payment in advance (cash, cheque, credit card). We bill the veterinarian at the owner's request only if approved by the referring veterinarian.
- We report to owners only on the request of their veterinarian.



Please note that the **Companion/other animal** tab this year includes all equine tests. For clients in specialty practice, we would be happy to provide the Fee Schedule for you sorted by test name, method, etc., within your species of primary interest - available shortly. Please contact Helen Oliver at (519) 824-4120, ext 54538.

We will keep you posted on changes in test availability and fees through this quarterly AHL Newsletter - also available on the Web at [http://www.labservices.uoguelph.ca/units/ahl/news\\_notes.cfm#News](http://www.labservices.uoguelph.ca/units/ahl/news_notes.cfm#News) AHL

## Our new LIMS - Sapphire - went live May 7, 2007

VADDS, our previous laboratory information management system (LIMS) , has been replaced with a new product - "Sapphire", from LabVantage - and we went live on May 7.

Our new LIMS is designed to provide better tracking of specimens through our labs, will support improved formatting of reports, and will enhance client service. Simultaneously, we've changed many of our within-lab processes, and will continue to fine-tune these needed advances. Some highlights of the new system:

- Improved client friendliness of the report format.
- "Tests pending" are clearly identified on interim reports.
- Invoices follow completed cases on the next day.
- Web access to reports and attachments.
- Submission of cases via the Web will be available shortly.

To sign up for access to the client-only section of the AHL website and Web access to your results, please contact:

Dr. Jim Fairles, AHL Client Services Veterinarian (519) 824-4120, ext. 54611 [jfairles@lsd.uoguelph.ca](mailto:jfairles@lsd.uoguelph.ca)

Ms. Josie Dobing, Client Services (519) 824-4120, ext. 56071 [jdobing@lsd.uoguelph.ca](mailto:jdobing@lsd.uoguelph.ca)

Client feedback on these recent changes is most welcome! AHL

## Lyme disease surveillance - tick submissions to public health laboratories

Bruce McNab

Wildlife and entomologic studies have identified established populations of **black-legged ticks** and small rodents infected with *B. burgdorferi* along Lake Erie at Point Pelee National Park, Rondeau Provincial Park, Long Point peninsula and Turkey Point; as well as along Lake Ontario in the Prince Edward Point National Wildlife Area; and most recently, in the Thousand Islands of the Saint Lawrence River in the Saint Lawrence Islands National Park, near Gananoque. The extent of areas in Ontario with established Lyme disease is not known. Infected ticks can also be spread at lower densities throughout the province, adventitiously on birds migrating from endemic areas.

Public Health Officials report 5 to 10 Ontario-acquired cases of Lyme disease in people each year, plus 15 to 25 annual travel-related cases, that are believed to have been acquired outside Ontario. Models suggest that Lyme infected areas will increase in Ontario with anticipated climate change. Veterinarians may increasingly wish to consider Lyme disease as a potential rule-out in arthritic dogs, and caution clients and staff to avoid exposure to ticks.

Public Health Officials would like to obtain a better

Veterinarians may increasingly wish to consider Lyme disease as a potential rule-out in arthritic dogs

understanding of the frequency and distribution of ticks in Ontario carrying organisms of concern to public health, such as *B. burgdorferi*. Therefore, over the coming year (between June 2007 and May 2008), **veterinarians are invited to submit ticks** (in sealed plastic sample submission containers) **directly to their Regional Public Health Laboratory** of the Ontario Ministry of Health and Long Term Care, with a history of where the tick was found.

A list of Regional Public Health Laboratories may be found at:

[www.health.gov.on.ca/english/public/contact/phl/phlloc\\_dt.html](http://www.health.gov.on.ca/english/public/contact/phl/phlloc_dt.html). It would be greatly appreciated if submitters would fill out BOTH a **Public Health Laboratory Test Requisition form** (available from Regional and Central Public Health Laboratories – contact Mr. Billy Yu at Central Laboratories 416 235-6315) AND a **Parasitology Patient's History form**, available online at: [www.health.gov.on.ca/english/providers/pub/labs/specimen\\_guide/form\\_F-C-PA-027-001.pdf](http://www.health.gov.on.ca/english/providers/pub/labs/specimen_guide/form_F-C-PA-027-001.pdf).

This testing will be done for surveillance purposes. **Veterinary clinics will not be charged a laboratory fee**, but testing will be given a lower priority such that reporting of positive results will usually take several weeks. AHL

### AHL Newsletter

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Editor: **Grant Maxie**, DVM, PhD, Diplomate ACVP  
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*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 2007. Ideas and opinions expressed herein do not necessarily reflect the opinions of the University or the editor.*

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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.*

## Collection and handling of milk samples for somatic cell counts (SCCs)

*Durda Slavic*

Somatic cell counts (SCCs) are widely accepted as an indicator of udder health status. Cows with high SCCs (above 200,000/mL) are more likely to suffer from subclinical mastitis than cows with lower SCCs. When collecting samples for SCCs, please keep the following points in mind:

1. **Minimum of 20 mL of milk is required** (Fig. 1).
2. Collect milk in sterile screw-capped plastic vials available from the AHL.
3. Label each vial with the animal ID.
4. **Refrigerate (DO NOT FREEZE).**
5. Complete the submission form.
6. Ship the samples to the lab.

Samples with less than 20 mL of milk and with visible changes in milk consistency will be deemed as unacceptable for SCC. Culture and susceptibility, however, can still be done on these samples. *AHL*

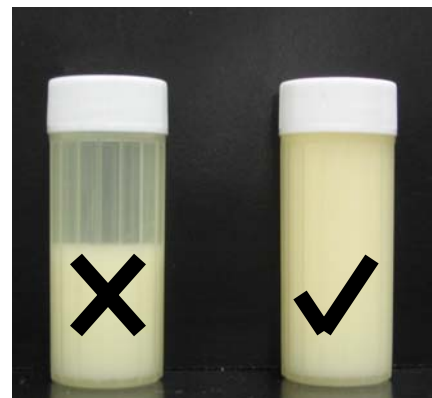


Figure 1. Incompletely filled milk vial (left), vs. completely filled vial (right) that is acceptable for somatic cell counting.

## Interpretation of laboratory tests – what makes a diagnosis?

*Murray Hazlett, Susy Carman*

Why did your in-house canine parvovirus test result negative, while the histopathology report insists on parvovirus being the problem? Why was your strong clinical impression of swine influenza virus infection not confirmed by PCR testing? Why isn't there a PCR test for BSE?

Each diagnostic test in veterinary medicine must be interpreted in conjunction with clinical assessment, an understanding of the pathogenesis of the disease of interest, and an understanding of the advantages and pitfalls of the methodology used in the diagnostic tests selected.

Diagnosticians at the AHL frequently receive phone calls from practitioners about test interpretation, most often when there are conflicting results between 2 tests.

In the next few issues of this Newsletter, we will examine some situations based on real events that come across our phone lines, and grouping these case scenarios by species. We welcome your questions or suggestions. We pride ourselves that our veterinary staff has spent time in private practice and has a good idea of the challenges faced by clinicians in selecting and interpreting diagnostic tests. With the wide battery of tests and methodologies available, confusion can result - we hope to help.

**Case 1: Bently the scouring dog.** Bently, a 12-week-old puppy, had an acute onset of diarrhea. He had received 2 vaccinations for *Canine parvovirus 2* (CPV). A CPV anti-

gen ELISA performed at the clinic was negative. Dr. Smith was wondering about salmonellosis, and was told by another lab that *Salmonella* spp was difficult to isolate and advised that he should request PCR testing. A positive PCR test was obtained for *Salmonella* spp in feces, leading to a working diagnosis of salmonellosis. Bently died, and histopathology of formalin-fixed sections from intestine identified cryptal necrosis typical of CPV-2 infection. What went wrong here?

**Answer: A CPV antigen ELISA frequently gives a false negative result.** With parvovirus in puppies, there is frequently a window of opportunity for disease when maternal antibody is overwhelmed by viral contamination of the environment. Damage to the intestinal mucosa by the virus can result in hemorrhage and exudation of serum proteins, including maternally derived anti-CPV antibody, into the intestinal lumen, where it binds to and neutralizes viral antigen. As a result, CPV antigen present in feces is unavailable for reaction in the ELISA, producing a false negative ELISA result. *Salmonella* spp should not be difficult to isolate in a clinical case of salmonellosis, unless the animal was treated prior to culture. **A positive PCR test result for any infectious agent only indicates the agent is present**, perhaps in a carrier state, but not necessarily causing disease (assuming the test has been validated and has reasonable specificity and sensitivity). *AHL*

# AHL Lab Reports

## RUMINANTS

### Hindsight is 20/20 - Sample submission and test interpretation in a calf pneumonia / diarrhea case

Josepha DeLay

Three heifer calves in a group of 8 developed acute-onset, rapidly progressive anorexia, coughing, and diarrhea. One affected calf was euthanized by the practitioner for on-farm necropsy and additional testing. Formalin-fixed samples from multiple tissues, including lung and small intestine, were submitted to the AHL for histopathology. Significant lesions were confined to intestine and included necrosis of mucosal crypts and submucosal lymphoid aggregates. Based on these lesions, BVD and bovine coronavirus enteritis were the main differential diagnoses.

#### 1. Helpful features of this submission:

- Euthanasia of a recently affected, untreated animal. In enteric disease cases, there is a greater chance of capturing and preserving useful histologic lesions if intestine is sampled from a just-euthanized animal and placed immediately in formalin.
- The practitioner described the gross lesions identified at necropsy - this is very helpful to the pathologist interpreting histologic lesions.
- In addition to lung and intestine, a wide range of other tissues were included for histopathology (esophagus, rumen, abomasum, liver, lymph node, kidney, heart).

#### 2. Room for improvement:

- Submit (fresh) or save at your clinic (frozen) samples from the same tissues sampled for histopathology, with emphasis on tissues with gross lesions or associated with your differential diagnoses. Formalin will inactivate infectious agents, making it impossible to use formalin-fixed tissues for other test methods such as bacterial culture, virus /mycoplasma isolation, PCR, antigen ELISA, and immunofluorescence. Immunohistochemistry (IHC) is the only additional, commonly available test used to examine formalin-fixed tissue, but IHC tests are not available for all infectious agents.

- For histopathology, submit multiple sections from organs such as lung and intestine. These organs have significant regional variations in exposure / susceptibility to infectious agents, making it worthwhile to include samples from sites such as cranial, middle, and caudal lobes of lung, and all levels of small intestine (duodenum, jejunum, ileum) and colon. For enteric disease cases, multiple samples from each level of intestine is even more helpful, as lesions may be present in one area but absent from others.

#### Results from this case:

**Both BVDV and Bovine coronavirus antigen were identified by IHC in areas of intestine with histologic lesions, resulting in a diagnosis of concurrent BVDV and BCV enteritis.** Although a useful diagnosis was reached here with suboptimal tissue submissions, this is not always the case, and negative results can leave a practitioner no further ahead in reaching a diagnosis. Although it's tempting here to assume that BVDV and/or BCV is also responsible for pneumonia, it is important to remember that lung has not been tested for any infectious agents, and making this assumption (however logical it seems), could be detrimental if it is wrong.

In addition to histopathology, as requested, a more complete diagnostic approach to this case would have included:

- small intestine, colon, and sections (fresh) for bacterial culture.
- colon (fresh) for *Bovine coronavirus* ELISA.
- freezing and holding (at the clinic) samples from lung, ileum, mesenteric lymph node, and spleen for possible virus isolation and typing (IHC cannot determine the type of BVDV involved). *AHL*

### SRMs and diagnostic labs, July 12, 2007

Canada's **enhanced ruminant feed ban** comes into effect on July 12, 2007. Specified risk materials (SRMs) are prohibited from being used in all livestock feed, pet food, and fertilizers. In BSE-infected cattle, SRMs contain the prion that may transmit the disease, and include skull, brain, trigeminal ganglia, eyes, tonsils, spinal cord, and dorsal root ganglia of cattle aged 30 mo or older, and the distal ileum of cattle of all ages. The CFIA controls the handling, transporting, and disposal of SRM by way of permits. **Permits are not required for transport of cattle to AHL laboratories for necropsy examination.** SRMs are disposed of from AHL post-mortem rooms by incineration or by alternative means. For more information see: <http://www.inspection.gc.ca/english/anim/feebet/rumin/enhrene.shtml>. *AHL*



# AVIAN SPECIES

## Blackhead (histomoniasis) in turkeys

Lloyd Weber, Marina Brash

Blackhead, also known as histomoniasis and as enterohepatitis, is a disease that is not infrequently diagnosed at the AHL every fall in small turkey flocks and results in significant mortality. Prevention of this disease needs to start with placement of the poults in the spring.

**Blackhead is caused by a protozoan parasite, *Histomonas meleagridis***, which is carried by the common poultry cecal worm *Heterakis gallinarum* found in the ceca of chickens and turkeys. This protozoal organism is fragile and cannot live outside the bird host for very long, but can survive for long periods of time in the environment when in the cecal worm or its eggs. Earthworms can carry these infected cecal worms and so are also important in transmitting blackhead. Flies, beetles, grasshoppers, sowbugs and crickets can also serve as mechanical vectors.

Typically, outbreaks in turkeys begin with the ingestion of infected earthworms, cecal worms, larvae or eggs from contaminated soil where previous flocks of chickens or turkeys have been raised. Pheasant, partridge and peafowl can be affected, so blackhead can be also a problem on game farms where other avian species are being raised. Bird-to-bird transmission through direct contact with infected droppings can contribute to the maintenance of the outbreak.

Once the *H. meleagridis* organisms are ingested, they travel to the cecum, penetrate the cecal mucosa, multiply, cause inflammation and thickening of the cecal wall, enter the bloodstream and travel to the liver inciting an inflammatory response. Interaction with coccidia and bacteria, including *Clostridium perfringens* and *E. coli*, increases the severity of disease with more tissue damage. Turkeys can show clinical signs of illness 7-12 days post ingestion including yellow feces (sulfur droppings), drowsiness, ruffled feathers, drooping of the wings, placement of the head down and close to the body or tucked under the wing, and anorexia. The head may or may not be cyanotic - this is actually how the disease got the name of blackhead. Death follows shortly, with mortality in the flock peaking at about 17 days postinfection and subsiding after 4 weeks.

At necropsy, the lesions are characteristic, with enlargement of the ceca with marked thickening of cecal walls with caseous or fibrinonecrotic cores (Fig. 1). Sometimes there is extension of the inflammation through the serosa with localized peritonitis. The liver is enlarged, tan, with multiple circular depressed areas of necrosis circumscribed by raised yellow rings described as bull's-eye type of lesions (Fig. 2).

**Histopathology is required to confirm the diagnosis;** histomonad organisms can be seen within the cecal and hepatic tissues.

**Prevention of this disease is the key, as there are no approved treatments.** In small turkey flocks, the approach is multipronged and includes:

1. Chickens and turkeys should be raised in separate areas. **Turkeys should not be reared where chickens have been housed previously.** Free-roaming chickens and turkeys are much more likely to pick up cecal worms and may visit areas frequented by wild turkeys, pheasants and partridge.
2. If there is a history of blackhead on the farm, free roaming domestic fowl must either be relocated to new fenced pastured areas or moved indoors to prevent contact with intermediate hosts and contaminated soil. Concrete floors are preferred to dirt floors.
3. Straw and bedding storage areas should be bird proof to prevent fecal contamination. New litter should not be stored /contaminated with old litter.
4. Control mechanical vectors including earthworms, beetles, flies and other arthropods.
5. Routine deworming to control cecal worm infestations may be helpful. A gut scraping can be used to confirm the diagnosis, and a veterinarian is then best placed to advise on management and deworming.
6. Histostat 50 from Alpharma Inc. is the only product approved in Canada as an aid in the prevention of histomoniasis in turkeys. Major feed companies in Ontario maintain stock of Histostat medicated turkey grower for small flocks. Veterinarians with questions concerning Histostat and use in turkeys can contact Dr. Maurice Smith of Alpharma at 1-800-265-7167 ext 230. AHL

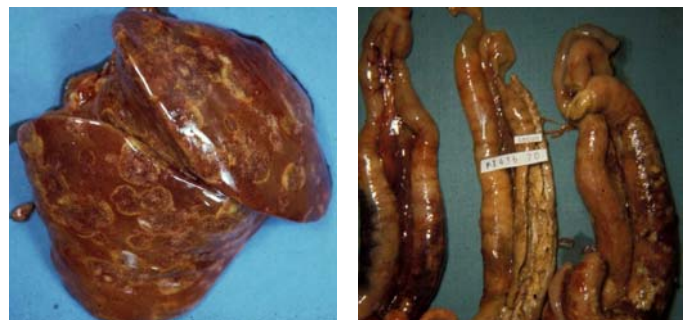


Figure 1 (left): Typical histomoniasis lesions in turkey liver.

Figure 2 (right): Ceca of turkeys with histomoniasis.

(Photos courtesy of Dr. Bruce Hunter and Dr. Richard Julian, Dept of Pathobiology, Ontario Veterinary College, University of Guelph.)

# SWINE

## Porcine circovirus 2 - associated disease diagnoses in 2006

Susy Carman, Beverly McEwen, Josepha DeLay, Hugh Cai, Jim Fairles, Tony van Dreumel

**Porcine circovirus 2 (PCV-2)-associated disease continued into 2006**, with 408 new cases presented in 2006, compared to 350 cases in 2005. The percent total of swine submissions increased from 8.9% in 2005 to 10.1% for 2006 (Fig. 1). Because these data are impacted by submission biases to the diagnostic laboratory, they cannot be regarded as population prevalence estimates.

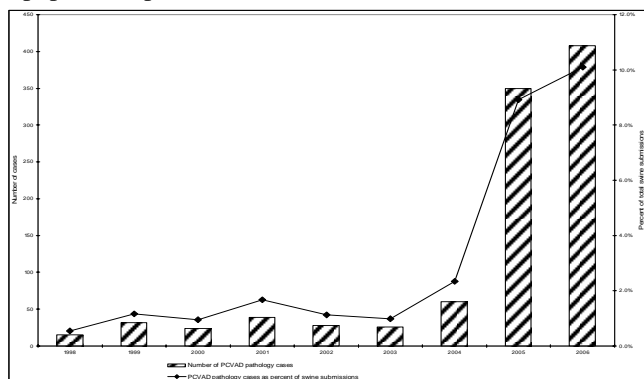


Figure 1. Number of PCV-2 pathology cases, and PCV-2 pathology cases as percent of total swine submissions, 1999-2006.

PCV-2 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) which was seen in 130/172 cases. However RFLP type 422 is still present within Ontario swine, and was seen in 16/172 cases. Both RFLP types 422 and 321 have been demonstrated in the same herd. AHL

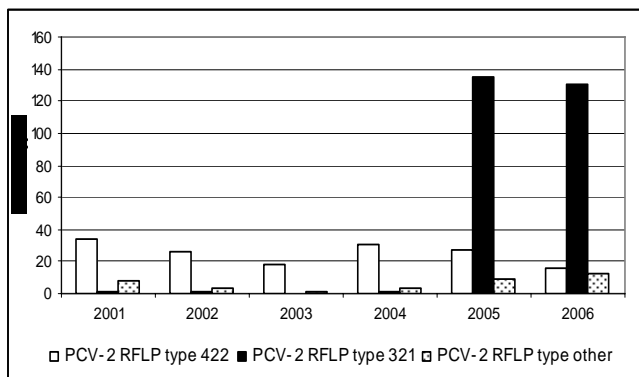


Figure 2. PCR-RFLP typing of PCV-2, 2000-2006.

## Pneumatosis intestinalis ("bubbly gut") in swine

Murray Hazlett, Tim Blackwell, Bob Friendship

About once a year, tissues are submitted to the AHL from pigs with **emphysema beneath the intestinal serosa**. Severity can be very mild to prominent (Fig 1). Histologically, emphysema can be observed through all levels of the gut wall, often with accompanying histiocytes, multinucleated giant cells and lymphocytes. Pigs appear clinically normal, and the condition is not noticed until slaughter, when costs are incurred because the intestine cannot be used for sausage casings. Over the past 10 years, various bacterial stains as well as immunohistochemistry for PCV-2 have been attempted with no significant leads to an etiology.

**A recent submission from a producer who had a problem over the last 6 months was typical.** Between 5 and 20% of the intestinal tracts from pigs shipped each week were discarded for rendering. Both male and female pigs were affected. A quick look at carcasses on the processing line showed some evidence of pulmonary pathology. Analysis of the gas from the bubbles indicated it was mostly air with some methane.

Whey feeding has been felt by some to be associated with this condition, and this farm incorporates liquid whey in the diet for finishing pigs. This case may be significant because of the increase in liquid feeding systems in some large finishing operations. It will be interesting to see if diseases that may be particular to these units such as bub-

bly gut, whey bloat, salt toxicity, and foot rot (damp conditions) increase.

Pneumatosis intestinalis is well recognized in humans, and while idiopathic, it has been associated with at least 58 causative factors. Chronic diseases associated with it fit into 2 basic categories - intestinal disease, and pulmonary disease (cystic fibrosis, COPD). Immunosuppressive therapy and diseases also seem to play a significant role.

Could the disease in pigs be associated with chronic enteritis or respiratory disease? The histology described in pulmonary-associated cases in humans seems to match well with what we are seeing in the pigs submitted to the lab. AHL

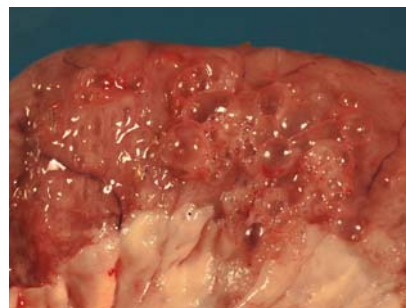


Figure 1. Subserosal emphysema of the small intestine of a pig.

# HORSES

## Equine encephalomyelitis associated with EHV-1 infection

Murray Hazlett, Dorothee Bienzle, Susy Carman, Jim Fairles

Traditionally, *Equid herpesvirus 1* (Equine abortion virus, EHV-1) has been associated with respiratory disease and abortion in horses, as well as encephalomyelitis. Neurologic disease tended to be sporadic, with rare outbreaks. However, in 2005 and 2006, 16 outbreaks were reported, mostly in the eastern USA. Affected premises were quarantined. Recently it has been shown that a single amino-acid difference in the DNA polymerase made by the virus is strongly associated with neurological versus non-neurological EHV-1 outbreaks.

**In 2006, 3 cases of encephalomyelitis associated with EHV-1 were identified at the AHL, all in Thoroughbred horses of racing age.** Clinical signs ranged from “neurologic disease”, to difficulty eating and wasting of head muscles, to proprioceptive losses and a head tilt. Microscopically, inflammation was seen in brain sections from all 3 (Fig. 1), and spinal cord changes were seen in 2 of these horses. This included perivascular lymphocytic cuffing, which is common for viral encephalitides, and is not specific for EHV-1. A diagnosis of EHV-1 infection was made using PCR amplification of a region of the viral DNA polymerase.

**PCR testing** from nasal swabs, blood or brain tissue for EHV-1 is currently only available at Guelph on selected cases. Clinically healthy, infected horses may yield positive PCR results from pharyngeal swabs, but not blood. We can send samples to California for a PCR test to identify EHV-1 specifically associated with neurological disease.

The cost is \$65 per test, plus handling and air shipment.

In addition, **we offer paired serology and virus isolation** as tools for identifying EHV-1 in outbreak situations and in single animals with neurologic disease. For serology, please submit acute and convalescent sera. Virus isolation has been successful in isolating herpesviruses from white blood cells harvested from EDTA blood. **Immunohistochemistry and fluorescent antibody testing** are also available on necropsy samples. AHL

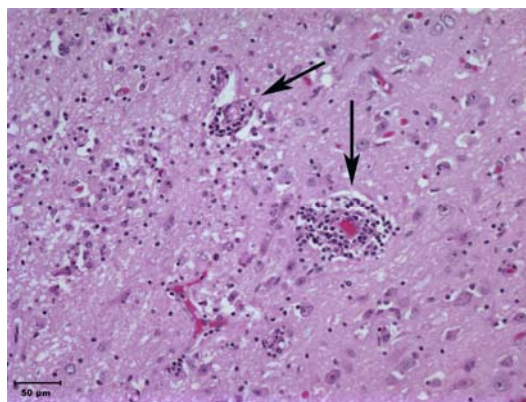


Figure 1. Encephalitis due to EHV-1 showing perivascular lymphocytic inflammation (arrows) typical of many viral encephalitides, including infections with *West Nile virus*, *Rabies virus*, and *EEE virus*. AHL

## Equine multinodular pulmonary fibrosis: an uncommon but histologically distinct disease

Josepha DeLay

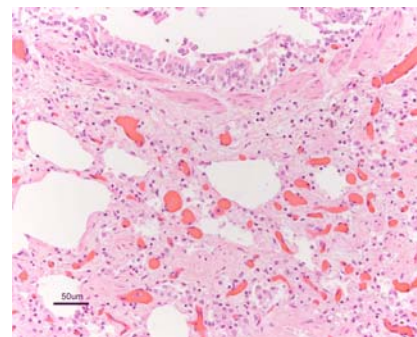
A 6-year-old Thoroughbred gelding was presented for productive cough and weight loss of 2 months duration. Radiographs identified a diffuse alveolar pattern in lung, and lung biopsy demonstrated lesions consistent with equine multinodular pulmonary fibrosis (EMPF). The horse was euthanized due to the poor prognosis for this condition. At necropsy, lungs were diffusely firm, with multifocal to coalescing, poorly defined regions of lung parenchyma that were pale tan and extremely tough.

Histologically, alveolar septa were widely expanded by fibrosis, and alveoli contained a mixture of inflammatory cells, including numerous multinucleated giant cells in some areas. **The hallmark of EMPF is fibrosis of alveolar septa with preservation of the general alveolar architecture in lung.**

Infectious and toxic etiologies have been proposed for alveolar septal fibrosis in horses, although a definitive etiology is rarely determined. Silicate pneumoconiosis in horses living in a geographically distinct area of California

can produce similar gross and histologic lesions. Recently, *Equid herpesvirus 5* (EHV-5), a gamma herpesvirus, has been associated with the distinctive pattern of multinodular pulmonary fibrosis seen in this horse, although **the exact pathogenesis of the disease remains to be determined.** AHL

Figure 1. Equine multinodular pulmonary fibrosis, demonstrating marked fibrotic expansion of alveolar septa.



### Reference

Caswell JL, Williams KJ. Respiratory system. In: Maxie MG, ed. Jubb, Kennedy and Palmer's Pathology of Domestic Animals. 5th ed. vol 2. Oxford: Elsevier. 2007:569.



# COMPANION ANIMALS

## The melamine-cyanuric acid pet food recall

*Grant Maxie, Brent Hoff, Perry Martos, Andrew Moore*

Beginning in March, 2007, practitioners in Ontario and other locales in North America witnessed a dramatic increase in cases of acute renal failure in cats and dogs. Affected animals had a common history of anorexia, vomiting and marked azotemia, after consuming one of a number of recalled pet foods. The recall began with Menu Foods, but the list of recalled products grew gradually to over 100 from a variety of manufacturers, and eventually extended to include swine feed, poultry feed, and farmed fish food.

Urinalyses of affected cats and dogs indicated low specific gravities and unusual crystalluria (Fig. 1). A toxicant was suspected. Several medications - notably, sulfonamides and methotrexate - are associated with production of crystals that are insoluble in feline and canine urine. Intoxication with ethylene glycol, mycotoxins, heavy metals, or vitamin D was ruled out early in the laboratory workup. The round, brown crystals were determined by Lab Services to contain melamine. The US Food and Drug Administration had identified melamine in contaminated wheat gluten imported from China, and subsequently identified cyanuric acid as well. Rice protein concentrate and corn gluten were also soon found to be contaminated.

**The feline urine crystals were identified as melamine-cyanuric acid in the Canadian Veterinary Urolith Centre.** In vitro, addition of a melamine solution to a cyanuric acid solution, in the ratio of 1:1, resulted in the instantaneous formation of crystals identical to those found in the urine of affected cats. FTIR (Fourier transform infra-red) spectroscopy scans of the feline urine crystals and the crystals formed in vitro revealed them to be identical (Fig. 2).

Since mid-March, 2007, 15 cats and 3 dogs have been submitted to the Animal Health Laboratory for necropsy after consuming some of the recalled pet food for a period of 3-7 days. Histologic lesions consist of nephrosis (acute tubular necrosis) and interstitial edema or fibrosis (tubulointerstitial disease), but often against a background of chronic renal lesions (glomerulosclerosis, chronic tubulointerstitial disease, etc.). Hence, our impression is that the proximate cause of death may have been melamine-cyanuric acid intoxication, but that there was significant pre-existing

disease in at least some cases. Crystals similar to those seen in urinalyses are present in affected kidneys, but often in small numbers, likely the result of solubilization in formalin.

Although contaminated product has been identified, and characteristic crystals and nephrosis have been observed, the pathogenesis of this syndrome remains obscure, given that both melamine and cyanuric acid are relatively non-toxic in their own right. *AHL*



Figure 1. Melamine-cyanuric acid crystals in feline urine.

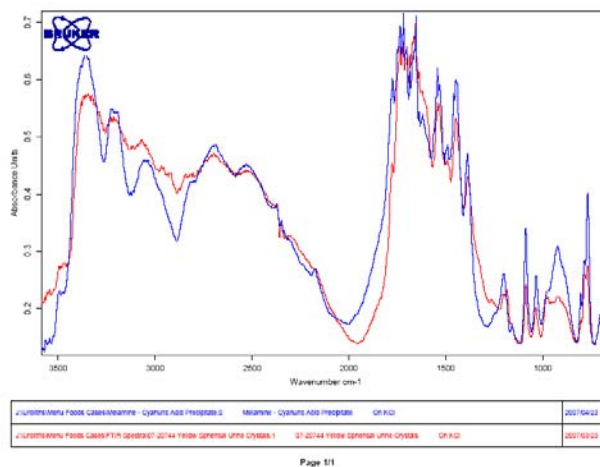


Figure 2. Concordance of FTIR spectra of feline urine crystals with in vitro produced melamine-cyanuric acid crystals.

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